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Ethylene, indoleacetic acid and apical dominance in peas: A reappraisal

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Decapitation of peas (*Pisum sativum* L. cv. Greenfeast) promoted sprouting of the lower buds with the most active growth in the first week occurring in the bud at the lowest fully expanded leaf node. Addition of 3-indolyl acetic acid (IAA; a 0.03 *M* solution, applied at 10 and 25 μg/plant) inhibited bud outgrowth whether added to the cut stump or injected above or below the lowest leaf node. Ethylene evolution by the nodal region decreased following decapitation, but increased greatly if IAA was added to the cut stump. Ethylene gas (3, 15 and 1 500 μl/l) or the precursor ACC (1-aminocyclopropane-1-carboxylic acid) reduced bud outgrowth while factors which scrub ethylene (mercuric perchlorate), inhibit ethylene synthesis (canaline), or prevent its action (silver nitrate), enhanced bud growth on decapitated plants. It was concluded that auxin-induced inhibition of bud growth through an increase in ethylene synthesis is a more logical hypothesis than the direct inhibition by auxin per se since a) acropetal movement of the inhibitory principle occurred whereas [^{14}C] IAA movement in stems was basipetal, b) a decline in the levels of ethylene evolution was correlated with bud outgrowth in decapitated plants and c) exogenous application of chemical agents which increase or decrease ethylene level or response lead to correlative decreases or increases in bud outgrowth, respectively.

Additional key words – *Pisum sativum*, canaline, silver nitrate, 1-aminocyclopropane-1-carboxylic acid, flushing, bud growth.

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Introduction

The involvement of auxin in apical dominance in plants was suggested by the classical experiments of Thimann and Skoog (1934) whereby removal of the apex of pea plants (*Pisum sativum* L.) apparently removed a factor inhibiting lateral bud growth. Since inhibition of lateral bud growth in decapitated pea plants could be reinstated by application of 3-indolyl acetic acid (IAA) to the cut stump it was suggested (Thimann and Skoog 1934) that it is auxin diffusing basipetally from the intact apex which inhibits lateral bud growth in intact plants.

Various objections to the classical auxin theories of apical dominance were reviewed by Phillips (1969). These objections include the observation that a) in Thimann's and other studies relatively high concentrations of auxin are used for inhibition of lateral bud growth and the need for exact substitution of exogeneous IAA in investigations of presumed inhibitory effect of auxin (Jacobs et al. 1959) and, more importantly, b) the inhibiting principle, unlike auxin, can move acropetally in pea plants (Snow 1937).

The possibility that ethylene is involved in correlative inhibition of lateral bud growth was suggested since a) applied IAA stimulated ethylene production in the nodal region of pea stems (Burg and Burg 1968a,b) and b) ethylene gas inhibited lateral bud growth by decapitated pea plants when applied at low concentrations (1 and 10 μl l^{-1}) of ethylene (Burg and Burg 1968a,b).

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Conflicting theories have arisen concerning the role of auxin and ethylene in apical dominance. Earlier studies by Burg and Burg (1968a) suggested that although ethylene synthesis by nodal regions of pea plants was reduced following decapitation, the auxin-induced ethylene production might not be the cause of apical dominance since no diminution of ethylene production was observed in nodal sections of decapitated pea plants possessing buds but lacking leaf scales, and apical dominance was not broken either by hypobaric treatment or increased carbon dioxide. These two treatments are thought to eliminate other symptoms of ethylene in some systems (Burg and Burg 1968a).

The ability of IAA and synthetic auxins to stimulate ethylene production (Crocker et al. 1935, Abeles and Rubinstein 1964, Morgan and Hall 1964, Coleman et al. 1980) by plants suggests that some auxin effects are actually caused by ethylene. Furthermore, responses to auxin in which ethylene appears to play at least some role include epinasty, inhibition of leaf and stem (Abeles 1973) but not root elongation (Andreae et al. 1968, Muir and Richter 1970), adventitious root formation (Fabijan et al. 1981) hypocotyl hypertrophy (Wample and Reid 1979), growth and shoot initiation in tobacco callus (Huxter et al. 1981), and branch angle in *Cupressus* (Blake et al. 1980). In view of these findings the rejection of a role for ethylene in apical dominance by Burg and Burg (1968a) should be reconsidered. In the present study the hypothesis was considered that auxin-induced ethylene may indeed play a role in apical dominance.

Abbreviations – ACC, 1-aminocyclopropane-1-carboxylic acid; EtOH, ethanol; IAA, 3-indolyl acetic acid; MeOH, methanol.

Materials and methods

Seed of *Pisum sativum* L. cv. Greenfeast, a semi-dwarf variety of pea were germinated and grown in a mixture of equal proportions of perlite, vermiculite and peat moss at 23±1°C day and 18±1°C night temperatures. Plants were grown in a growth room under a 16-h photoperiod, the fluorescent tubes (Sylvania, type F72T 10/cw) were supplemented with 60 W incandescent tubes (10%) to provide a total irradiance of 100 W m^{-2}. The relative humidity was maintained at 55% during the 16-h photoperiod and 90% during the 8-h dark period. Plants were watered with full-strength Hoagland's solution (Hoagland and Arnon 1950) twice daily to bring the soil to field capacity. After a period of 14 days the plants were used for experiments.

Seedlings (12 per treatment) were decapitated above the third leaf node (i.e. actually above node 5 since the 2 lowest nodes are subtended by vestigial, scale leaves) so that all leaves, leaf scales and bracts below the third leaf node were removed. The "leaf nodes", referred to are those subtended by fully developed leaves. Plants thus decapitated together with intact controls were treated as described below.

IAA and bud growth. IAA was applied to the stump immediately after decapitation and at 2-day intervals. This is the time required for uptake of applied IAA by pea tissue (Burg and Burg 1968b). The exogenous hormone was applied in two ways: a) 25 µg of IAA in 5 µl EtOH (0.03 *M* IAA) pipetted to the top of the decapitated stump, or b) 10 µg IAA in 2 µl EtOH (0.03 *M* IAA) were injected with a syringe 1.5 cm above or below the lowest fully developed leaf node (node 3). Control (decapitated) plants received similar treatments of EtOH with no IAA.

Movement of [^{14}C] *IAA.* Two µl EtOH containing 0.005 mmol 3-indolyl [1-C^{14}] acetic acid (Amersham, Oakville, Ontario) (59 mCi/mmol = 2.18 GBq/mmol) was injected 0.5 cm above or below node 1. Four replicate plants from each of the two application sites (above or below) were harvested 24, 48, 72 and 120 h after application of [^{14}C] IAA. Harvested plants were dissected into leaves, roots, seed remnant and stems which were cut into 0.5 cm segments. Samples were oven-dried at 60°C for 24 h, ground and 1 ml 80% MeOH was added to each. After 12 h shaking, 5 ml Scinti Verse I (Fisher Scientific) was added to each sample and [^{14}C] content was determined through liquid scintillation spectroscopy using external standard calibration.

Ethylene and bud growth. The following treatments were carried out to modify ethylene levels:

a) $AgNO_3$, which inhibits ethylene action (Salveit et al. 1978), was applied as an aqueous foliar spray over the whole plant at a concentration of 200 m*M* 2 h prior to decapitation.
b) ACC, a precursor of ethylene (Bradford and Yang 1980), was applied to lateral buds immediately after decapitation and every second day, at a concentration of 0.5 m*M* in 3 µl water with graduated syringe.
c) Canaline, a compound which inhibits ethylene synthesis (Murr and Yang 1975), was applied after decapitation and every second day as an aqueous solution to each lateral bud at a concentration of 1 and 10 m*M* in 5 µl of water.
d) Ethylene was applied to decapitated seedlings bagged in 5 l transparent plastic bags by injecting ethylene gas into the bags with a syringe to provide a concentration of 3, 15 and 1 500 µl l^{-1}. Bags were opened for 10 min each day for ventilation and bud measurements and then closed and fresh ethylene gas was injected.
e) Mercuric perchlorate (0.25 *M*) was used as an ethylene trap to remove ethylene (Abeles 1973) from bagged seedlings and ethylene was left to ac-

cumulate in another (control) group of bagged seedlings.

f) Ethylene measurements – these were conducted for:
   (i) decapitated stem segments containing lateral buds and
   (ii) lateral buds obtained by excising the nodal region from the lowest 3 leaf nodes of decapitated pea stems.

Ethylene evolution was measured by sequentially decapitating pea seedling 0 (control), 0.5, 1, 2 and 5 days after decapitation. Pea stem segments (segments of stems or leaf nodes with lateral buds, 6 plants per treatment, 2 estimates per plant) were incubated for 2 h in glass vials (5 cm × 2.4 cm diameter) sealed with rubber serum caps. The ethylene concentrations in the vials were then sampled by extracting 1 ml of gas using a gas-tight syringe. Samples of gas were injected into a Varian Series 3 700 gas chromatograph with a Poropak Q or N column for ethylene determinations with flame ionization detection as previously described (Blake et al. 1980).

Results

Effects of decapitation and IAA on bud outgrowth

The lowest bud in the axil of a fully developed leaf (node 3; leaf node 1) elongated most in the 6 days following decapitation with less growth occurring on the buds in leaf nodes 2 and 3. IAA (0.03 *M* solution applied as 25 µg in 5 µl ethanol) inhibited lateral bud outgrowth in all 3 lower leaf nodes. Data are shown for the bud elongation of buds in the lowest and uppermost leaf node only in Fig. 1.

Injection of IAA (0.03 *M* solution applied as 10 µg IAA in 2 µl ethanol) above or below leaf node 1 (lowest) strongly inhibited the outgrowth of the bud at this node. Injection of IAA above the leaf node was somewhat more inhibitory than injection below the leaf node (Fig. 2).

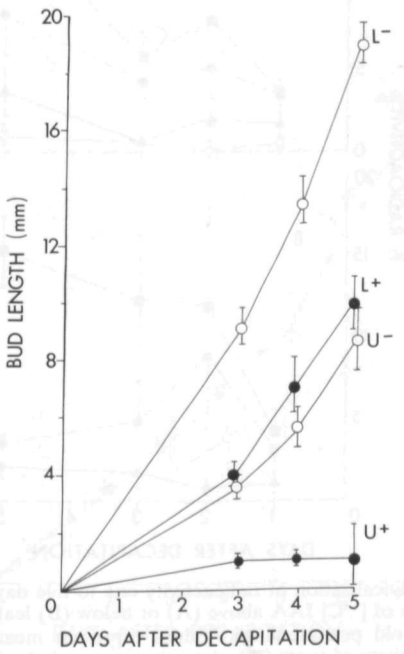

Fig. 1. Bud elongation after decapitation on the lowest (L) and third lowest i.e. uppermost (U) nodes with fully developed leaves in the presence (+) and absence (−) of auxin (0.03 *M*, 25 µg/cut stump) applied to the cut stump immediately after decapitation and at 2-day intervals. Values represent means ± SE.

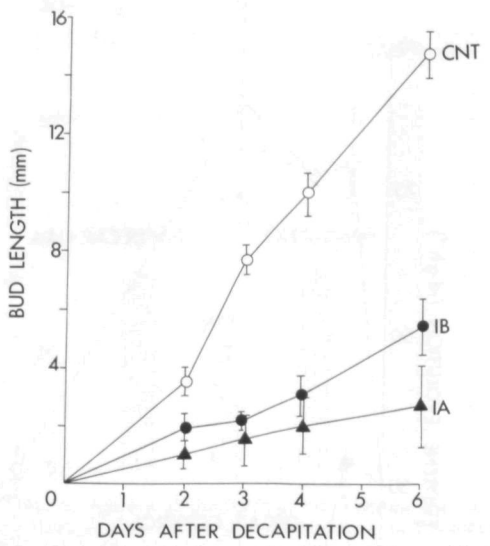

Fig. 2. Elongation of the bud in the lowest fully expanded leaf node following decapitation in the presence (IAA-treated) and absence (CNT, controls) of IAA (0.03 *M*, 10 µg/plant) which was injected above (IA) or below (IB) the lowest leaf node. Values represent means ± SE.

Effects of decapitation and IAA on ethylene evolution

Ethylene evolution by the nodal regions (3 lowest leaf nodes) was reduced by two thirds following decapitation compared with production by intact plants. Addition of IAA (0.03 *M* solution applied as 25 µg in 5 µl ethanol) to the decapitated stump resulted in a trebling of ethylene production by day 3, with a return to rates found in intact plant by day 5 (Fig. 3). Ethylene evolution by decapitated stem segments containing lateral buds was also reduced following decapitation (data not shown).

The diminution of ethylene production in decapitated plants was verified for the individual leaf nodes from leaf node 1 and 3 (data not shown) as well as the node segments 1 to 3 taken together (Fig. 3).

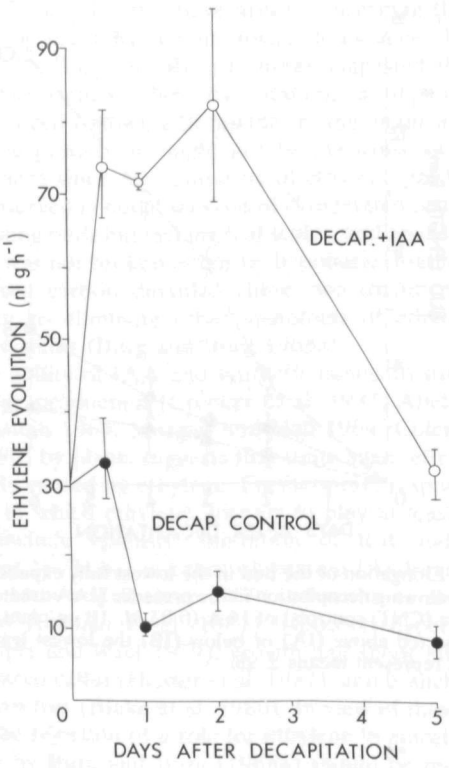

Fig. 3. Ethylene evolution by decapitated pea stems with and without the addition of IAA (0.03 *M*, 25 µg/plant) to the cut stump after decapitation at 2-day intervals thereafter. Values indicate means ± SE.

Movement of [^{14}C] IAA

Movement of [^{14}C] following the injection of [^{14}C] IAA was slight and almost entirely basipetal (Fig. 4). About 75–80% of the applied [^{14}C] remained at the site of application over the 120 h sampling period. Roots were the principal sinks for the [^{14}C] (Fig. 4).

Effects of ethylene gas and mercuric perchlorate on bud outgrowth

Ethylene (3, 15 and 1 500 µl l^{-1}) gassing of decapitated seedlings inhibited bud outgrowth (Fig. 5). Conversely, scrubbing of ethylene gas with mercuric perchlorate promoted bud outgrowth on decapitated plants (Fig. 5).

Effects of canaline, $AgNO_3$, and ACC on bud outgrowth

Compounds with decrease ethylene synthesis (canaline) or inhibit its action ($AgNO_3$) doubled the rate of bud outgrowth compared with decapitated (control) seedlings (Fig. 6). The slower rate of bud outgrowth in this experiment compared with that shown in Fig. 1 may possibly reflect a difference in seed batches.

Injection of the ethylene precursor, ACC (0.5 m*M*), into the cut stump reduced bud outgrowth (considered as the total length of the shoots from buds in the 3 lowest leaf nodes) and canaline (10 m*M*) doubled the total shoot length 11 days after decapitation compared with the decapitated controls (Fig. 7). In another experiment there was a similar degree of inhibition with ACC regardless of whether the ACC was injected above or below the lowest leaf node (data not shown).

Discussion

The correlated integrated nature of plant growth in intact pea plants was recognized by the nineteenth century botanists in their study of correlative inhibition. Some early botanists suggested that the active growth of the plant apex channeled off nutrients from the lateral buds thereby preventing their growth (the "nutrient diversion" theory is reviewed by Phillips 1969). When

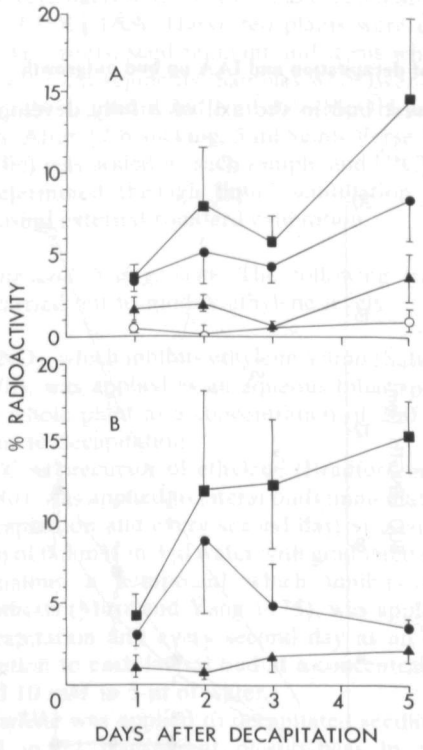

Fig. 4. Localization of radioactivity one to five days after the addition of [^{14}C] IAA above (A) or below (B) leaf node 1 of 15-day-old pea seedlings. Values represent mean ± SE % radioactivity of roots (■), the primary stem below the site of application (●), leaves and lateral shoots below application (○), and the primary shoot above the application (▲). Percentage radioactivity expressed as:

$$\frac{B_q \text{ recovered from the specific organ}}{\text{total } B_q \text{ from the whole plant}} \times 100$$

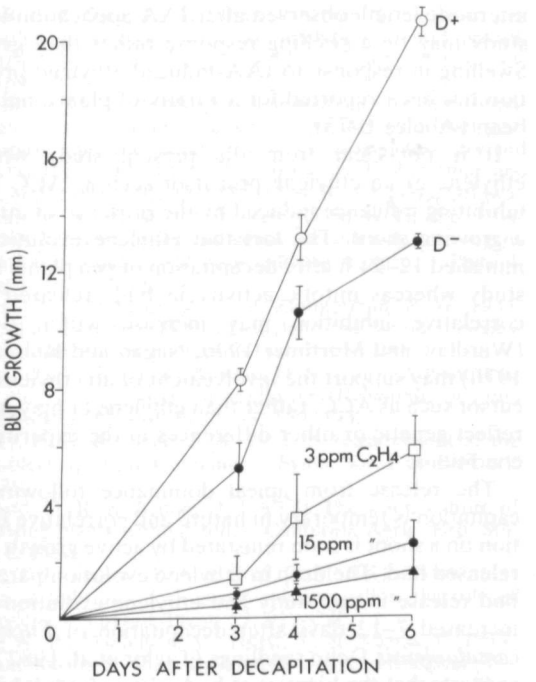

Fig. 5. Elongation of the bud in the lowest leaf node following decapitation in the presence of ethylene gas (3, 15 and 1 500 µl l^{-1}) and the presence (D+) or absence (D−) of the ethylene scrubbing agent mercuric perchlorate.

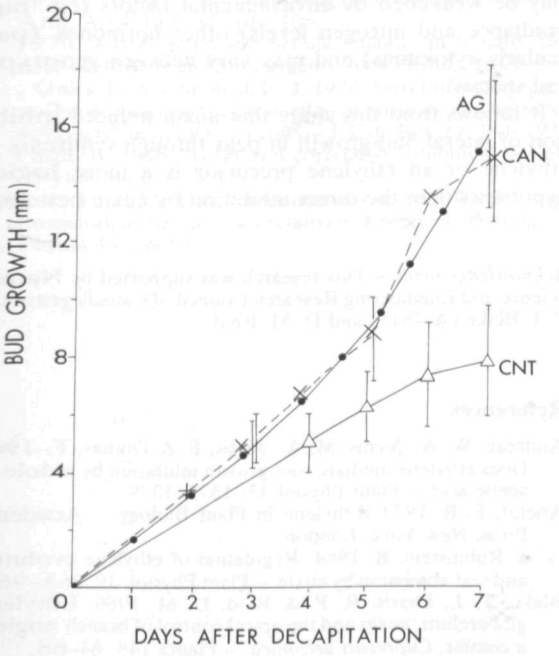

Fig. 6. Elongation of the bud in the lowest leaf node of decapitated plants in the presence or absence of the ethylene affecting compounds, silver nitrate (Ag, applied as a 200 m*M* foliar spray prior to decapitation) and 10 m*M* L-canaline (CAN) applied every 2 days. Control seedings (CNT) were supplied with a water spray only.

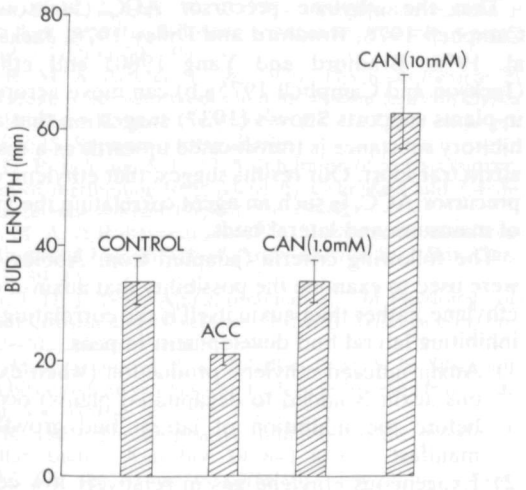

Fig. 7. Total elongation of the buds in the 3 lowest leaf nodes following decapitation in control (untreated) and seedlings with ACC (10 m*M*) injected into the cut stump, or canaline (1.0 and 10.0 m*M*) applied as a droplet onto these buds every 2 days.

plant hormones were discovered the nutritional theories were largely neglected until the work of Gregory and Veale (1957) who found that apical dominance in flax (*Linum usitatissimum* L.) was manifest only under conditions of nutrient deficiency and apical dominance was manifestly reduced with adequate nutrition. They suggested apical dominance was caused by a competition by shoot meristems for available nutrients, particularly nitrogen and carbohydrates.

A diffusable substance was shown to cause bud inhibition in pea plants by Snow (1925) and controversy followed as to whether auxin per se inhibited bud development (the 'direct' theories of Thimann and Skoog 1934 and others) or caused the production of an inhibitory substance or specific deficiency ('indirect' theories of Snow 1937, 1939 and others).

Much evidence has accumulated in the literature suggesting that auxin is not the direct cause of correlative bud inhibition (cf. review by Phillips 1969). It is possible that the passage of auxin down the stem causes production or release of another substance which inhibits bud growth (Snow 1937, Libbert 1954). Experiments with split pea plants showed that while the inhibitory principle travels both up and down the plant (Snow 1937), auxin transport is mainly basipetal (our results, also Le Fanu 1936). Although Wickson and Thimann (1960) demonstrated some acropetal transport of [^{14}C] when [^{14}C] IAA was added to nodal segments of decapitated pea plants we observed little acropetal transport in the plants themselves. Thus, it is possible that the previously observed acropetal transport (Wickson and Thimann 1960) may have been an artifact in excised stem segments.

That the ethylene precursor ACC (Jackson and Campbell 1976, Bradford and Dilley 1978, Jackson et al. 1978, Bradford and Yang 1980) and ethylene (Jackson and Campbell 1975a,b) can move acropetally in plants supports Snow's (1937) suggestion that an inhibitory substance is translocated upwards as a result of auxin transport. Our results suggest that ethylene or the precursor ACC, is such an agent correlating the growth of mainstem and lateral buds.

The following criteria (adapted from Abeles 1973) were used to examine the possibility that auxin-induced ethylene, rather than auxin itself is the correlating agent inhibiting lateral bud development in peas.

1) Auxin-induced ethylene production (when exogenous auxin is added to decapitated plants) occurred before the inhibition of lateral bud growth was manifest.
2) Exogeneous ethylene gas in relatively low concentrations mimicked the effect of auxin in inhibiting lateral bud growth on decapitated plants.
3) Chemical treatments which remove ethylene (mercuric perchlorate; Abeles 1973), inhibit ethylene synthesis (L-canaline; Murr and Yang 1975) or inhibit its action (Ag^{2+} ions; Salveit et al. 1978) promoted bud outgrowth.
4) Application of the ethylene precursor ACC inhibited bud growth on decapitated plants.

The fact that these criteria were satisfied supports the hypothesis that auxin-induced ethylene is the correlative inhibitor. Several other lines of evidence also support this contention:

1) Acropetal movement of the inhibitory principle was observed whereas auxin movement was principally basipetal.
2) Ethylene evolution decreased following decapitation and remained low during the onset of lateral bud outgrowth.
3) Kinetin reverses the inhibitory action of 1 $\mu l\ l^{-1}$ ethylene on pea buds (Burg and Burg 1968b) just as it reverses the inhibition caused by auxin (Wickson and Thimann 1958, 1960).

It is also noteworthy that the effects of ethylene and ethylene-related compounds were observed in decapitated plants. Since auxin levels are reduced following decapitation of pea stems (reviewed by Phillips 1969), this again suggests that it is ethylene rather than auxin, which inhibits lateral bud growth.

A relationship between auxin-induced ethylene and bud inhibition was first investigated for excised nodal pea sections by Burg and Burg (1968a,b). The elongation of buds of bean (*Phaseolus vulgaris* L.) plants in response to exogenous IAA added directly to buds led Yeang and Hillman (1982) to reject the hypothesis that auxin-induced ethylene was the cause of correlative inhibition in that species. It is likely that the very small (0.18 mm) and temporary nature of the increase in bud internode length observed after IAA application in this study may be a swelling response rather than growth. Swelling in response to IAA-induced ethylene production has been reported for a variety of plants including bean (Abeles 1973).

It is not clear from the present study whether ethylene or an ethylene precursor such as ACC is the inhibiting influence induced by the presence of auxin in a growing shoot. The fact that ethylene evolution diminished 12–24 h after decapitation of pea plants in our study whereas mitotic activity in buds released from correlative inhibition may increase within 6–12 h (Wardlaw and Mortimer 1970, Nagao and Rubenstein 1976) may support the involvement of an ethylene precursor such as ACC, rather than ethylene or may merely reflect genetic or other differences in the experimental conditions.

The release from apical dominance following decapitation is temporary in nature and correlative inhibition on a shoot is soon reinstated by active growth of the released bud. The drop in ethylene evolution paralleled bud release in our study and ethylene evolution again increased 7–12 days after decapitation of *Eucalyptus camaldulensis* Dehn seedlings (Taylor et al. 1982). This suggests that the kinetics of bud release from inhibition and the reinstatement of correlative inhibition agree with levels of ethylene.

In conclusion, the mechanism controlling the expression of apical domination in intact plants is complex and may be weakened by environmental factors (eg. high irradiance and nitrogen levels) other hormones (particularly cytokinins) and may vary between genotypes and species.

It follows from this study that auxin-induced inhibition of lateral bud growth in peas through synthesis of ethylene or an ethylene precursor is a more logical hypothesis than the direct inhibition by auxin per se.

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