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APICAL DOMINANCE IN PEA: EFFECT OF MORPHACTIN ON TRANSPORT OF AUXIN AND KINETIN[1,2]

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Abstract

IT3456, a morphactin, at low concentration applied by spraying, released lateral bud of Alaska pea (*Pisum Sativum* L.) from correlative inhibition. Direct application of morphactin on the lateral buds, however, completely inhibited the outgrowth of lateral buds. The transport of IAA-2-^{14}C and kinetin-8-^{14}C in the presence or absence of morphactin were studied by using intact seedlings and internode segments. The morphactin could inhibit the transport of IAA-2-^{14}C but could not inhibit the acropetal movement of kinetin-8-^{14}C. The results were discussed in relation to the possible role of auxin-directed transport of root-produced cytokinin in the regulation of lateral bud growth.

Introduction

The release of lateral bud from correlative inhibition is a well known physiological function of cytokinins (Wickson and Thimann, 1958; Sachs and Thimann, 1964). It is generally accepted that the activation of lateral buds is controlled by the auxin-cytokinin balance (Sach and Thimann, 1967). The endogenous auxin synthesized in the apical tissues of the shoot is transported in a polar manner down to the lateral buds, resulting in the inhibition of the lateral bud growth (Wickson and Thimann, 1960), while cytokinin-like substances are mainly generated from root system (Buttrose and Mullins, 1968; Kende, 1964; Mullins, 1968; Smith and Wareing, 1964). Recently, enough evidence indicated that the polarizing effect of auxin on distribution of root-produced cytokinin may play an important role in the inhibition of outgrowth of lateral buds (Chang, 1970, 1974; Morris and Windfield, 1972; Wooley and Wareing, 1972). Chang (1970, 1974) noted that the removal of root system of

(1) This study was supported by the National Science Council, Republic of China.
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(3) Abbreviations: IAA: indole-3-acetic acetic acid; Kinetin: 6-furfurylaminopurine; IT3456: methyl-2-chloro-9-hydroxy-fluorene-(9)-carboxylate; PPO: 2,5-diphenylo-xazole; TIBA: 2,3,5-triiodobenzoic acid.

Alaska pea prevented the lateral bud growth which would normally occur when the seedlings were decapitated. Kinetin applied to the low part of rootless seedlings could simulate the the lateral bud growth. The presence of IAA[3] in the shoot greatly promoted the uptake of ^{14}C-kinetin supplied at the basipetal end of the rootless shoot and also promoted the acropetal transport of absorbed ^{14}C-kinetin. Morris and Windfield (1972) found that decapitation resulted in the transport of significant amount of ^{14}C to the lateral buds from root supplied with ^{14}C-8-kineitn. However, the pretreatment of IAA on the cut internode surface of decapitated plants inhibited the of transport of the labelled kinetin to the lateral buds, resulting in its accumulated in the IAA-treated region of the stem. In order to clearly understand the role of auxin effect on kinetin acropetal transport in the mechanism of apical dominance, morphactin was employed in this investigation inasmuch as it was found to favor the outgrowth of lateral bud (Schneider, 1970). Additionally, the inhibition of IAA transport and the alternation of IAA level after application of morphactin were reported (Knelle and Libbert, 1968; Mann *et al.*, 1966; Tongnoni *et al.*, 1967). The stimulatory action of morphactin on lateral bud growth, however, may not be solely due to the blocking of inhibitory effect of IAA since the synergistic effect of morphactin and cytokinins was reported on other morphogenetic system (Pieniazak and Sabiewsko, 1968). Thus, studies were undertaken to determine whether morphactins could interact with cytokinin in controlling apical dominance and promote the acropetal movement of cytokinin from root system. Additionally, a comparison study of the effect of morphactin on the transport of IAA applied to apical buds and to mature leaves was investigated.

Material and Method

Uniform 6 to 14-old seedlings of pea (*Pisum sativum* L. cv. Alaska) were used as experimental material. Kinetin-8-^{14}C with a specific activity of 16.5 mCi/mM was obtained from the Radiochemical Centre, Amersham, U.K. Indole-3-acetic acid-2-^{14}C (IAA-2-^{14}C) (sp. act. 15 mCi/mM) was purchased from New England Nuclear, U.S.A. Chlorofluronol (methyl-2-chloro-9-hydroxyfluorene-9-carboxylate, called IT3456), one of the most potent morphactin, was kindly donated by Merck AG, Darmstadt, Germany. To easy understand the experimental results, the specific methods are described in detail in each following experiments.

Results

Effect of morphactin on lateral bud growth of intact seedlings

Pea seeds were soaked in tap water for 12 hr, and sown in plastic cups containing vermiculate-sand mixture (1:1). Plants were grown under greenhouse

conditions and the temperature in day and night was 20 to 27°C. The plants were watered daily with tap water and with the half-strength Hoagland's nutrient solution twice a week. Seedlings with two fully expanded leaves and 5 visible internodes were used (about 12 to 14 days after planting) for all the experiments. Fifteen seedlings for each treatment were sprayed with different concentration of morphactin (IT3456) with a 0.01% surfactant, Tween 20. Total plant height and the combined length of lateral bud were recorded 7 days after application of the chemicals. Results (Table 1) showed that at the concentration of 0.005 ppm to 10 ppm the morphactin dwarfed the treated seedlings, but greatly promoted outgrowth of lateral buds. Generally, leaves of morphactin-treated seedlings were greener than that of control.

Table 1. *Response of Alaska pea to a morphactin, IT3456*

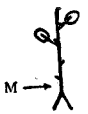
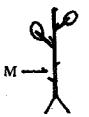
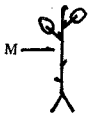
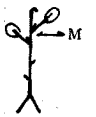
Concentration(1)	Seedling height (mm)(2)	Combined length of lateral buds (mm)(2)
Control	190±13	6±4
IT3456 0.001 ppm	174±21	11±3
0.005	156±17	17±5
0.01	144±23	22±4
0.05	138±19	24±5
0.1	123±25	37±6
0.5	119±18	43±9
1	112±16	47±7
5	116±27	41±6
10	103±21	36±7

(1) Treated solutions contained 0.01% Tween 20.
(2) Average and standard deviation of 15 seedings. Measurements were made 7 days after spraying.

Effect of local application of morphactin on outgrowth of lateral buds of intact seedlings

Seedlings at the 12th day were used for this investigation. Five hundred microliter of lanolin paste containing 50 μg IT3456 was applied as a narrow ring around the selected internode, using a 1.0 ml syringe. Plants applied with a lanolin by the same manner were served as the control. The length of lateral buds was measured 5 days after treatments. The results (Table 2) indicated that the action of morphactin on outgrowth of lateral buds seemed to be polarized in a basipetal direction. This was clearly shown by the response of lateral buds to morphactin application since morphactin applied above the bud induced the release of bud from apical dominance, while it had no effect when applied below the bud.

Table 2. *Influence of the application site of a morphactin, IT3456, on the release of lateral buds from apical dominance*

Position of bud	Length of lateral bud, mm[1]						
	Site of morphactin application[2]						
	Control	Decapitated	1st internode	2nd internode	3rd internode	4th interned	5th internode
							
4th 	0.3±0	2.0±0	0.3±0	0.3±0	0.3±0	0.4±0	1.5±0.7
3rd	0.4±0	2.5±0.4	0.3±0	0.3±0	0.4±0	2.0±0	1.2±0.5
2nd	2.5±0.4	6.2±0.7	2.6±0.7	2.3±0.4	5.8±0.5	5.5±0.7	5.2±0.5
1st	2.0±0.3	4.2±0.5	2.3±0.5	5.8±0.5	3.7±0.6	3.5±0.4	3.1±0.7

(1) Results shown were recorded 5 days after treatment.

(2) Morphactin, IT3456, was incorporated into warm hydrous lanolin. About 0.5 ml (ca 50 μg IT3456) was applied as a narrow ring around the selected internode, using a 1.0 ml disposable syringe.

Effect of kinetin and morphactin on bud outgrowth of intact and decapitated seedlings

Seven-day-old seedlings were used for this study. The effect of kinetin and morphactin or both chemicals on the release of lateral buds from apical dominance was observed. The length of axillary shoots was measured 6 days after treatments. The results indicated that the drop application of morphactin on the lateral bud of intact or decapitated seedlings greatly repressed the outgrowth of lateral bud, while application by spraying favored the bud growth (Table 3). Morphactin in drop application antagonized the stimulatory effect by kinetin. However, there was no synergistic effect of kinetin with morphactin applied by spraying on bud growth.

Table 3. *Effect of a morphactin, IT3456 and kinetin on the lateral bud growth of Alaska pea*

Treatment	Combined bud length[3]
Intact seedlings	
Control	2.3±0.7
Kinetin, 100 ppm (drop)[1]	7.4±1.7
IT3456, 100 pm (drop)	2.4±0.5
IT3456, 10 ppm (spray)[2]	6.2±0.4
Kinetin, 100 ppm (drop)+IT3456, 10 ppm (spray)	8.7±1.5
Kinetin, 100 ppm (drop)+IT3456, 100 ppm (drop)	3.5±0.8
Decapitated seedlings	
Control (decapitated)	9.8±1.7
Kinetin, 100 ppm (drop)	10.2±1.9
IT3456, 10 ppm (spray)	7.6±1.2
IT3456, 100 ppm (drop)	3.7±0.6
Kinetin, 100 ppm (drop)+IT3456 100 ppm (drop)	3.2±0.6
Kinetin, 100 ppm (drop)+IT3456 10 ppm (spray)	8.2±1.9

(1) Drop: drop application on lateral bud (about 0.05 ml).
(2) Spray: spray application on whole seedlings.
(3) Bud length as measured 6 days after treatment.

Interaction of morphactin and kinetin on bud growth ***In vitro***

Seeds of Alaska pea were germinated in vermiculate at 25°C in darkness with occassional red light illumination. Stem section of 3.5 cm long with first (lowest) bud in the middle were used as material. They were immediately laid in sterile Petri dishes, containing 1% sucrose in half-strength Hoagland solution, then, incubated at 20°C at light intensity of 2,000 foot candles. The growth of buds from its extreme tip to the site of intersection of the scale

leaf was measured. It was found that the morphactin greatly inhibited the growth of lateral bud (Table 4). The antagonism effect between morphactin and kinetin was also shown that morphactin significantly inhibited the promotive action of kinetin.

Table 4. *Effect of a morphactin (IT3456) and kinetin on the lateral bud growth of Alaska pea grown* ***in vitro***

Treatment	Bud length (mm)(1)
Water control	2.5±1.9
1% sucrose	8.6±2.1
10 ppm IAA	1.5±0.2
Kinetin 5 ppm	11.2±2.7
Morphactin, 1 ppm	2.6±0.4
Morphactin, 0.1 ppm	3.2±0.5
Morphactin, 1 ppm+Kinetin 5 ppm	3.5±1.4
Morphactin, 0.1 ppm+Kinetin 5 ppm	5.2±1.6

(1) Initial bud length is about 0.8 mm. Results were recorded 10 days after treatment.

Effect of a morphactin on IAA-2-^{14}C movement in isolated internode

The first internode segments of 7-day old seedlings were used in this experiment. A 5 mm internode segment was cut from each seedling at a distance of 5 mm below the first node, using two razor blades mounted apart in a plexiglass holder. The segments were treated with various concentrations of morphactin for 12 hr before the experiment. Agar blocks used as recciver and donner blocks were prepared as follows: Bacto-Agar (Difco) at a concentration of 1.5% was molted in a phosphate citrate buffer 10^{-3} M at pH 4.6. After the temperature of agar solution decreased to about 60°C, the IAA-2-^{14}C was added and thoroughly mixed. Then, the agar was poured in Petri dishes, cooled, and cut in discs with a cock borer. The agar disc had a diameter of 20 mm and 3 mm thickmess. The agar discs without radioactive chemicals were used as receiver blocks. Ten internode segments were then placed on a donner agar block resting on a microscrope glass slide. Two modeling compound columns a little higher than the receptor block plus segments, were attached at the two ends of the slide. The donner block, placed on another glass slide, was pressed down on the modeling compound column until all segments made good contact with the donner and receptor blocks. Each translocation set-up subsequently placed in a Petri dish containing a moistened filter paper on the bottom. After translocation periods, agar block and internode segments were transferred to the counting vials containing 1 ml methanol.

After 3-day extraction, 10 ml of liquid scintillation cocktail contained 9 g PPO and 100 g naphthalene in one liter dioxane was added and counted with a liquid scintillation spectrometer.

The results in Fig. 1 indicated that morphactin at concentration above 10^{-8} M greatly inhibited IAA-2-^{14}C translocation through the internode segments. Further experiments were conducted to demonstrate the effect of morphactin IAA transport in intact plants.

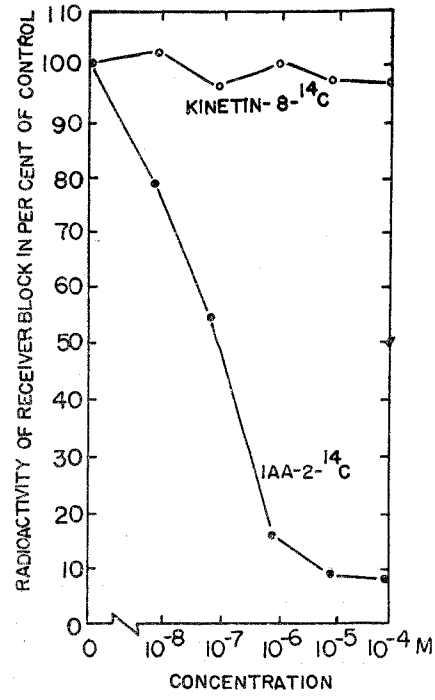


Fig. 1. Effect of a morphactin, IT3456, on the basipetal transport of IAA-2-^{14}C and Kinetin-8-^{14}C through pea internode segments. Radioactivity of receiver block after 1.5 hr translocation was expressed in percentage of water control.

Effect of morphactin on transport of IAA-2-^{14}C of the intact seedlings

Uniform 12-day old seedlings were used for this study. About 0.5 ml of hydrous lanolin containing 50 μg of a morphactin, IT3456, was applied as a narrow ring around the selected internode using a 1.0 ml disposable syringe. After 24 hr application of morphactin, a dosage of about 15,000 dpm of IAA-2-^{14}C was applied to the apical buds or first true leaf. For another 12 hr translocation, all the seedlings were divided into parts and radioassayed by using a scintillation counter as described earlier. Quenching by chlorphyll was corrected by a external standard. 'Morphactin applied on internode 5 or 3

Table 5. *Effect of a morphactin (IT3456) on the distribution of radioactivity in intact pea seedlings following the application of IAA-2-^{14}C to the shoot apex**

Plant Part	Mean radioactivity (dpm)		
	 Control	 Morphactin applied at 5th internode	 Morphactin applied at 3rd internode
Shoot apex	98403±6427	107920±7582	97545±7173
Internode 5	7412±197	2287±176	1936±231
Internode 4	1573±152	127±64	1753±215
Internode 3	749±91	25±6	927±89
Internode 2	231±74	10±6	94±21
Internode 1	217±39	—	11±6
Lateral buds, 3, 4,	15±7	—	23±5
Lateral buds, 1, 2,	28±8	—	—
Leaves	315±73	—	235±79
% Translocated	5.31	2.04	4.85
% Recovery	62.03	65.87	61.19

* The morphactin, IT3456, in hydrous lanolin (0.1 mg/ml) or plain lanolin were applied to the internode for 24 hr before the application of IAA-2-^{14}C (about 167,000 dpm/seedling) to the shoot apex, and was allowed to transport for 12 hr. Each treatment was performed by five replications.

greatly inhibited the basipetal movement of IAA-2-^{14}C in intact pea seedlings from the shoot apex (Table 5). On the contrary, morphactin did not influnce the transport of ^{14}C from IAA-2-^{14}C applied to the first true leaf (Table 6).

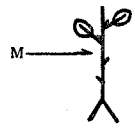
Effect of a morphactin on acropetal translocation of kinetin-8-^{14}C in internode segments

Transport experiments were conducted by mounting ten 5 mm segments of the first internodes of 6-day old seedlings between two agar discs as described before. Internode segments were treated with various concentrations of morphactin for 10 hr before mounting between agar dises. The donner blocks contained kinetin-8-^{14}C at a concentration of 2.5 ppm. Each set-up was subsequently placed in a Petri dish with a moistened filter paper on the bottom. After 1.5 hr translocation, agar block discs and internode segments were radioassayed by a LSC. The results, presented in Fig. 1, clearly showed that there was no inhibitory effect of morphactin on kinetin movement in donner block-tissue-receiver block system.

Effect of morphactin on upward movement of kinetin-8-^{14}C in intact seedlings

Seven-day-old seedlings were used in this experiment. Roots of all seedlings

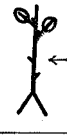
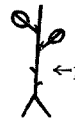
Table 6. *Effect of a morphactin (IT3456) on the distribution of radioactivity in intact seedlings following the application of IAA-2-^{14}C to the first true leaves**

Plant part	Mean radioactivity (dpm)	
	 Control	 Morpactin applied at the 3rd internode
Shoot apex	425±127	478±104
Internode 5	1732±154	1643±237
2nd true leaf	1524±241	1625±213
Internode 4	1841±217	1752±401
1st true leaf	51937±4273	61027±4158
Internode 3, 2	1873±214	1732±412
Internode 1	1127±198	972±174
Lateral bud	127±75	98±55
% Translocated	14.28	11.97
% Recovery	47.53	54.39

* The morphactin, IT3456 in hydrous lanolin (0.1 mg/ml) or plain lanolin was applied to the internode for 24 hr before application of IAA-2-^{1}'C (about 127,00 dpm/seedling) and was allowed to transport for 12 hr. Each treatment was performed by five replications.

were washed with tap water and incubated in tissue culture tube with 1 ml of half-strength Hoagland solution. About 0.5 ml of hydrous lanolin containing 50 μg of a morphactin, IT3456 was applied as a narrow ring around the selected internode using a 1.0 ml disposable syringe. After 24 hr morphactin application,

Table 7. *Effect of a morphactin (IT3456) on acropetal translocation of kinetin-8-^{14}C in 7-day old seedlings of Alaska pea*

Plant part	Radioactivity[1]	
	 ←morphactin in lanolin	 ←plain lanolin
Root system	77841±873	77124±1047
Tissue below morphactin ring	25143±417	26732±515
Tissue above morphactin ring	22731±614	23764±845

[1] An average dosage of kinetin-8-^{14}C, 167,000, was added to the root system.

a dosage of about 170,000 dpm of kinetin-8-^{14}C was added to each of the culture medium for another 12 hr translocation. All seedlings were divided into parts and were radioassayed. The results (Table 7) indicated that there was no significant inhibitory effect of morphactin on acropetal translocation of kinetin-8-^{14}C.

Discussion

The results of the experiments described above indicated that the weakening effect of morphactin on apical dominance of Alaska pea agreed with the earlier findings on other plants (Mann *et al.*, 1966; Schneides, 1967), namely, at low concentration the morphactin applied by spraying greatly weaken the apical dominance and favor the outgrowth of lateral buds. However, morphactin directly applied to the lateral bud completely inhibited the outgrowth *in vitro* and *in vivo* (Table 3 and 4). This is quite understandable since morphactin is a inhibitor for meristematic tissues (Schneider, 1970). The results obtained in Table 2 indicate that the biological effect of morphactin on apical dominance seems to be polarized in a basipetal direction. This is clearly shown by the behavior of the lateral buds since morphactin applied above the bud induces release from apical dominance when applied under the bud is no effect. Thus the action of morphactin on releasing lateral buds from apical dominance should not be due to its effectiveness in the meristematic tissue of the lateral bud. In view of auxin-directed root-produced cytokinin movement as a mechanism for apical dominance, the promotive effect of morphactin on lateral bud activation could be due to either an inhibition of IAA basipetal transport from shoot apex to the laterals, or the promotion of acropetal transport of root-produced cytokinin. Results of this investigation well supported the ideas that morphactin was able to inhibit IAA basipetal movement, and not significantly affected on kinetin acropetal movement.

The results of Table 5 and 6 supported the theory that there were two physically distinct pathways for the long distance transport of exogenous IAA in the intact plant (Morris and Kadir, 1972, Morris *et al.*, 1973). IAA appled to mature leaves may be transported acropetally and basipetally with exporting assimilates in the phloem, while the transport of exogenous IAA from apical bud may not depend on the transport of assimilates in the phloem. The failure of morphactin to influence transport of ^{14}C from IAA-2-^{14}C applied to foliage leaves confirms that the IAA transport in the pathway from apical bud to the downward organs and the transport of auxin in phloem sieve tubes take place in entire different mechanism. This fundamental difference in IAA transport mechanism has also been demonstrated by using TIBA as auxin transport inhibitor (Morris *et al.*, 1973).

The role of phloem in the movement of endogenous auxin has not fully

been understood. Especially the source and amount of the auxin in the plant and its physiological significance remain unclear. Nevertheless, it is clear that the auxin in the phloem is quite inactive in controlling lateral bud growth since the outgrowth of lateral buds below the site of morphactin application may represent either that only a small amount of endogenous auxin is transported in the phloem or the most of the endogenous auxin that retards the lateral bud growth is come from apical bud. A further experiment on the metabolic pattern of IAA in both transport systems as related to lateral bud growth will be attempted.

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豌豆的頂芽優勢: Morphactin 對 Auxin 和 Kinetin 運移的影響

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把 IT3456 之稀溶液噴施於14天大的豌豆 (*Pisum Sativum* L. var. Alaska)，則幼苗能打破頂芽優勢，促進側芽生長。但直接把 IT3456 施於側芽，却抑制側芽的生長。用整株植物做實驗結果發現：morphactin 對施於頂芽的 IAA-2-^{14}C 之向下運移有抑制的效果；但對施於根部的 kinetin-8-^{14}C 的向上運移却沒有影響。因此得知 morphactin 之所以能够打破頂芽優勢促使側芽生長之原因主要是抑制頂芽所產生的 auxin 向下運移，對來自根部供側芽生長用的 cytokinin 之運移沒有影響。用節間 5 mm 長之組織做運移試驗也證實了 morphactin 對 auxin 之運移有抑制的效果，而 cytokinin 之運移却不受其控制。施於成熟葉片之 IAA-2-^{14}C 之向下運移却不受 morphactin 的抑制。此表示：morphactin 只抑制在皮層組織內運移的 auxin 而對在篩管中運移的 auxin 少有影響。