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Abscission: Potentiating Action of Auxin Transport Inhibitors[1]

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ABSTRACT

Reduction in petiolar auxin transport has been proposed as one of the functional actions of endogenous or exogenous ethylene as it regulates intact leaf abscission. If this hypothesis is correct, auxin-transport inhibitors should hasten the rate or amount of abscission achieved with a given level of ethylene. Evidence presented here indicates that the hypothesis is correct. Three auxin transport inhibitors promoted ethylene-induced intact leaf abscission when applied to specific petioles or the entire cotton plant (*Gossypium hirsutum* L., cv. Stoneville 213). In addition, the transport inhibitors caused rapid abscission of leaves which usually do not abscise under the conditions employed. No stimulation of abscission occurred during the initial 3 to 5 days after plants were treated with transport inhibitors unless such treatments were coupled with exogenous ethylene or that derived from 2-chloroethylphosphonic acid. However, vegetative cotton plants did abscise some of their youngest true leaves during the 2nd and 3rd weeks of exposure to transport inhibitor alone. Taken as a whole, the results indicate that reducing the auxin supply to the abscission zone materially increases sensitivity to ethylene, a condition which favors a role of endogenous ethylene in abscission regulation. Such a role of ethylene indicates the importance of auxin-ethylene interactions in the over-all hormone balance of plants and specific tissues.

Recently a model has been proposed for the role of endogenous ethylene in the regulation of natural abscission of intact leaves (7). Jackson and Osborne (14) have also proposed that ethylene is a natural regulator of abscission. Briefly, the proposed regulatory system (7) first involves a modification of the hormone balance in the abscission zone achieved as rising ethylene levels reduce the auxin transport capacity of petioles. With auxin levels declining and ethylene levels rising, a point or balance is reached where ethylene exerts direct actions in the abscission zone such as stimulating synthesis of hydrolytic enzymes (2, 3, 13, 21) and secretion of these enzymes into the cell wall (4). When abscission is accelerated by exposing plants to ethylene, there is some evidence that the gas may alter auxin levels in the abscission zone of some plants by stimulating destruction (12, 20) and binding (9) and by inhibiting synthesis (23), as well as reducing transport capacity (7). Beyer and Morgan (7) suggested that the initial, ethylene-mediated events are less apparent in experiments with explants because excision removes the natural auxin source and wounding results in a temoprary sharp increase in ethylene synthesis.

One extension of the ethylene-auxin transport natural abscission model is that synthetic auxin transport inhibitors should perform the proposed initial function of ethylene and thereby hasten or increase the amount of leaf abscission that will result from a given level of ethylene. Beyer (6) has come to similar conclusions independently, and experiments similar to some of the ones reported here have been performed (5).

We report here that several auxin transport inhibitors increase the rate and extent of leaf abscission induced by ethylene or ethylene-producing ethephon. For periods up to a week after application, the transport inhibitors do not cause abscission themselves, but they potentiate the intact leaf to abscise when exposed to ethylene. Additional experimental approaches are possible and may be employed in future studies; however, the basic message of this paper is a straightforward result which justifies the approach employed.

MATERIALS AND METHODS

Plant Culture. Cotton plants (*Gossypium hirsutum* L., cv. Stoneville 213) were grown in a greenhouse in vermiculite in 11- × 15-cm plastic pots or 10- × 47- × 72-cm redwood trays. Plants were thinned to four per pot or five rows of 10 plants each in the trays and were watered with modified Hoagland's solution every 4th day. Unless otherwise specified, all plants were used 24 days after planting.

Auxin Transport Inhibitors. Plants were treated by spraying the entire plant with transport inhibitor without run off or by localized application of the inhibitor to specific petioles in lanolin from a micrometer activated syringe or in water solution with a small brush. Growing medium and pots were covered with Saran wrap during spray operations which employed compressd air operated glass atomizers.

Ethylene Treatments. At 24 hr after application of auxin transport inhibitors (unless time is specified otherwise), these plants and nontreated plants were placed in 268 or 50 liter Plexiglas chambers, and appropriate amounts of reagent grade ethylene were introduced to the sealed chambers with a gastight syringe. One-liter beakers half filled with 10% KOH and with large paper towel wicks were placed in each chamber to keep CO_2 from accumulating. Similar chambers including auxin transport inhibitor-treated and control plants, CO_2 traps, but no ethylene served as experimental controls. Thus, there were generally four treatments in each experiment: auxin transport inhibitor plants with and without ethylene and plants without auxin transport inhibitor but with or without ethylene. Ethylene levels were verified at the end of each 12-hr fumigation period by gas chromotography (19) and fell within the target concentration ± 10% error from period to period. Averages of ethylene levels actually measured are reported, and control chambers never contained measurable ethylene at the end of any 12-hr period. For convenience, ethylene con-

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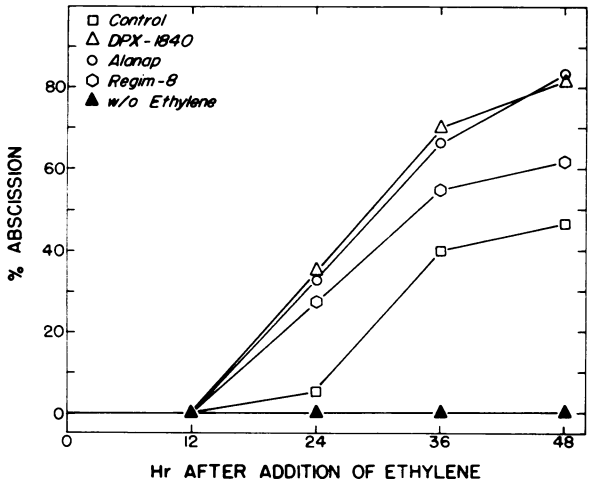

FIG. 1. Effect of auxin transport inhibitors at 1 mM on leaf abscission of 26-day-old cotton seedlings exposed to 5.34 μl (open symbols) and 0 μl (closed symbols) ethylene/liter of air. Control treatments included plants exposed to ethylene but no auxin transport inhibitor (open squares) and those not exposed to ethylene but either with or without transport inhibitors (solid triangles). With the no ethylene plants, no abscission occurred with or without transport inhibitors.

centrations in μl of the gas/liter of air are also reported as parts per million.

In some later experiments, ethephon (Amchem Products, Inc., Ethrel, formulation 68-240 of 2-chloroethylphosphonic acid) was applied as a spray in the same manner as the auxin transport inhibitors. These plants were not enclosed in chambers, and occassionally abscission was recorded each 24 rather than each 12 hr.

Plants were sprayed in a fume hood, allowed to dry before being moved, and were then placed in a room with a 15-hr photoperiod, cool white fluorescent lighting of 1000 ft-c at plant level, and temperatures of 31 $\pm$ 3 C during the day and 29 $\pm$ 2 C at night.

Abscission. Each 12 or 24 hr after plants were treated with ethylene or ethephon, the number of leaves which abscised when a 5-g weight was applied to the leaf blade-end of each petiole was recorded by each plant for each leaf position. In ethylene experiments, the plants were then resealed in chambers and ethylene added to the chambers, a procedure not followed with ethephon-treated plants. There was a minimum of two pots or two rows of plants per treatment, and experiments were repeated for verification of results. Where variability was observed, all of the data are presented.

Auxin Transport Inhibitors. The auxin transport inhibitors used were 2,3,5-triiodobenzoic acid (22) as the acid (Eastman Organic Chemicals, TIBA) and the dimethylamine salt, 14.2% active ingredient (Chemagro Corporation, REGIM-8); N-1-naphthylphthalamate as the sodium salt, 93.7% active ingredient (Uniroyal Chemical, ALANAP), (15, 18); and 3,3a-dihydro-2-(*p*-methoxyphenyl)-8*H*-pyrazolo-(5,1-a) isoindol-8-one (6) as the 80% active wettable powder (E. I. du Pont de Nemours and Company, experimental DPX-1840). All materials applied as sprays included 0.05% Tween 20 wetting agent in the spray solution. For convenience, the names in all capital letters given above will be used in the following sections.

In addition to the auxin transport inhibitors, a group of anti-auxins and related auxin antilogs with little or no growth-promoting activity (8, 16, 17) were tested. These included phenoxyacetic acid, 2-chlorophenoxyacetic acid, 4-chlorophenoxyacetic acid, 2,5-dichlorophenoxyacetic acid (2,5-D), 2,6-dichlorophenoxyacetic acid, 2,4-dichloroanisole, and 2,4,6-trichlorophenoxyacetic acid. These materials were supplied by Amchen Products, Incorporated.

RESULTS

The effect of three auxin transport inhibitors (6, 15, 18, 22) on leaf abscission is illustrated in Figure 1. All three materials caused a marked increase in leaf abscission over control plants also exposed to ethylene but not to an auxin transport inhibitor. During the time interval indicated, none of the transport inhibitors induced any abscission by themselves, *i.e.*, without ethylene. Several additional experiments not reported in detail confirmed these results.

We conducted additional experiments in which the auxin transport inhibitors were applied directly and exclusively to the petiole. We employed younger plants, 17 days from planting, so that the cotyledons would not have begun to visibly senesce and abscise and thus could be included in the test. Previous experience indicated that with such young, vegetative plants the cotyledons and some of the youngest, incompletely expanded leaves would be the first to abscise when exposed to ethylene and the oldest, nearly fully expanded leaf would be the last to fall (12 and unpublished data). The differences in age and stage of development of leaves of these plants are much less than occur several weeks later when the lower leaves will begin to visibly senesce. As indicated in Figures 2A and 3A, the localized application of auxin transport inhibitors to petioles greatly increased abscission of first true leaves and cotyledons of plants exposed to ethylene. Abscission of true leaf number one before younger leaves is a striking reversal of the usual pattern of abscission of vegetative cotton plants. In general, TIBA was least effective, and its acid form was more effective than the dimethylamine salt. Both forms tended to damage the treated petioles as indicated by discoloration and tissue shrinkage. TIBA at 1%, and to some extent the salt of TIBA and Alanap at both concentrations, increased the abscission of younger, untreated leaves located above those which were treated in comparison to the abscis-

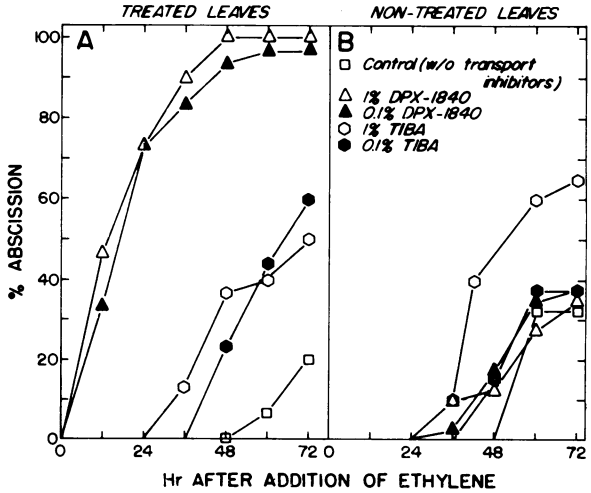

FIG. 2. A: Effect of auxin transport inhibitors on the abscission response to 5.19 μl ethylene/liter air. Inhibitors were applied in a lanolin ring midway along the petiole of the cotyledonary and first true leaves. B: Abscission response of leaves not treated with transport inhibitors (those above or younger than transport inhibitor treated leaves in A). In a simultaneous control experiment with 0 μl ethylene there was no abscission of any leaves or cotyledons either with or without transport inhibitors.

sion of the same leaves on plants receiving ethylene but no transport inhibitors (Figs. 2B, 3B). This result may indicate that DPX-1840 is not as readily transported as the other materials.

At the conclusion of the experiment detailed in Figure 2, we determined whether the localized application of auxin transport inhibitor would still increase ethylene-induced abscission. The control tray (no ethylene) of plants was sprayed with Ethrel (10 mM) on day 7 and again on day 8 (days numbered from the application of auxin transport inhibitors). At 60 hr after the initial Ethrel application, 1% TIBA and DPX-1840 increased abscission of treated leaves to 66 and 61% respectively *versus* 7% for the same leaves on plants without transport inhibitors.

The relationship between the level of one transport inhibitor, DPX-1840, and the level of ethylene on leaf abscission at 48 hr after exposure to ethylene is summarized in Figure 4. At ethylene concentrations from about 2 to 7 μl/l, the amount of abscission was relatively constant and was independent of DPX-1840 concentration in the range of 10^{-2} to 10^{-5} M. If the auxin transport inhibitor was not present, ethylene produced a completely different dose-response curve. Below 1 μl/l ethylene, the amount of abscission was influenced by both the level of ethylene and the level of transport inhibitor (Table I). At low levels of ethylene there was a minimum level of DPX-1840 required and abscission increased as inhibitor increased. The threshold level of ethylene for any abscission in 72 hr was between 0.03 and 0.10 μl/l. The optimum concentration of DPX-1840 was usually 1 mM for ethylene levels below 0.5 μl/l.

We determined that the auxin transport inhibitors increased the abscission activity of ethephon (Ethrel) as well as ethylene. We used that material to determine the optimum time between application of the transport inhibitor and ethylene (as Ethrel, Fig. 5, A and B). There was very little difference in abscission whether the inhibitors were applied 24, 12, or 0 hr before Ethrel.

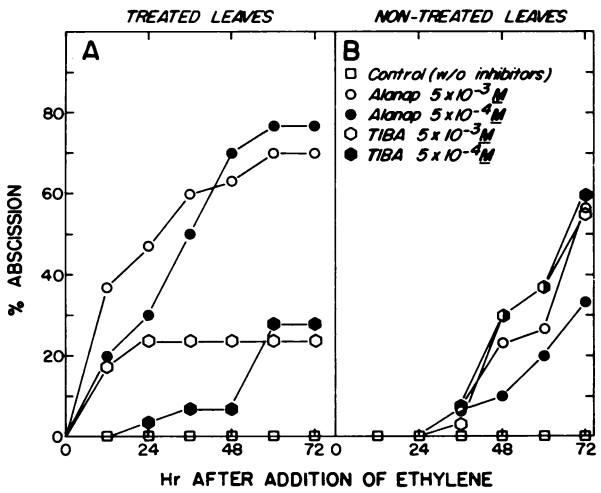

FIG. 3. A: Effect of auxin transport inhibitors on the abscission response to 5.16 μl ethylene/liter air. Inhibitors were applied in aqueous solution by brush to cotyledonary and first true leaf petioles. B: Abscission response of leaves not treated with transport inhibitors (those above or younger than treated leaves in A). In a simultaneous control experiment with 0 μl ethylene there was no abscission of any leaves or cotyledons either with or without transport inhibitors. The base of all petioles was treated with warm, liquid lanolin prior to application of transport inhibitors. The resultant ring of lanolin restricted inhibitor solution to the petiole above the abscission cells.

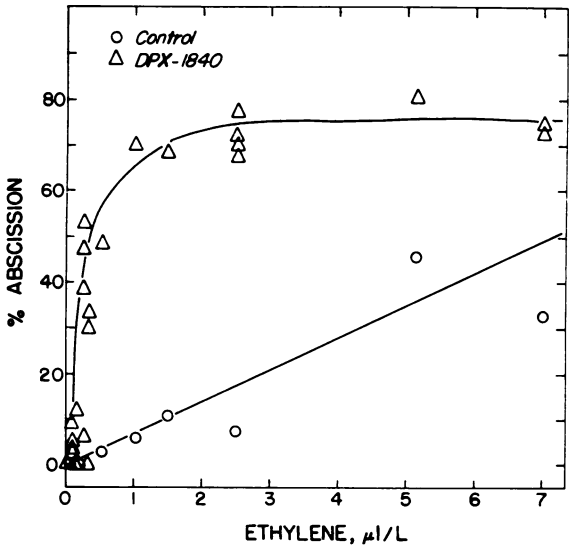

FIG. 4. Relationship between ethylene concentration (μl/liter air or ppm) and presence or absence of DPX-1840 on percentage of leaf abscission at 48-hr exposure to ethylene. Control indicates no ethylene. Data are from six experiments on 24- to 31-day-old cotton plants treated with DPX-1840 ranging from 10^{-2} to 10^{-6} M, and each point represents a minimum of eight plants in two pots. Data at 0 μl/l ethylene represents 12 observations (eight plants each) at DPX-1840 levels ranging from 0 to 3.6 mM. At ethylene levels from 0.03 to 0.17 μl/l and DPX-1840 ranging from 0 to 1 mM, no abscission occurred.

Table I. *Relationship between Auxin Transport Inhibitor (DPX-1840) Concentration and Ethylene Concentration with Respect to Leaf Abscission at 72 Hr after Exposure to Ethylene*

Data are from four separate experiments; each value is the average abscission of eight plants in two pots. Experiment D was done with plants from a growth room at previously described conditions (7).

DPX-1840 Concn	Experiment and Ethylene Concn (μl/liter air)							
	A		B		C		D	
	0.03	0.33	0.10	0.26	0.12	0.29	0.08	0.29
M	% Abscission							
0	0	0			0	0	0	0
10^{-6}	0	0	0	3	0	0	0	0
10^{-5}	0	18	6	25	0	27	0	0
10^{-4}	0	45	25	69	4	53	0	13
10^{-3}	0	55	13	88	35	63	0	31
10^{-2}			31	75	8	66	0	37

We tested the ability of auxin transport inhibitors to potentiate leaf abscission at abscission-saturating levels of ethylene and found that the degree of enhancement of abscission by DPX-1840 over that with ethylene alone decreases as ethylene levels increase (Fig. 6). At 1 to 3 μl/l ethylene, DPX hastened initiation of abscission as well as increased the magnitude of the response (Figs. 4 and 6), whereas at 52 μl/l there was only a relatively small difference in magnitude (Fig. 6).

TIBA is both an anti-auxin (10, 16), a very weak auxin (10, 16), and an auxin transport inhibitor (22). Therefore, we tested a series of 2,4-D analogs, many with significant anti-auxin activity, for their ability to potentiate Ethrel-induced abscission (Table II). At 1 mM, only phenoxyacetic acid and

2,4-dichloroanisole produced a consistent, clear-cut stimulation of abscission over that achieved with Ethrel alone. None of these anti-auxins were comparable to DPX-1840 and Alanap in their ability to promote abscission. Increasing the concentration of the anti-auxins 5-fold reduced their promotion of abscission.

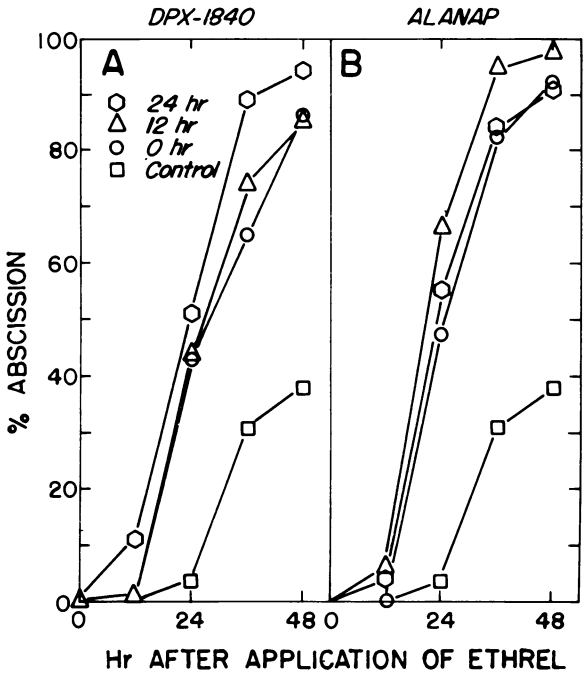

FIG. 5. Effect of auxin transport inhibitors DPX (A) and Alanap (B) on abscission induced by 0.1 M Ethrel. Inhibitors applied 24, 12, or 0 hr before Ethrel. Control received Ethrel but no transport inhibitors. Plants receiving neither Ethrel nor transport inhibitors exhibited no abscission.

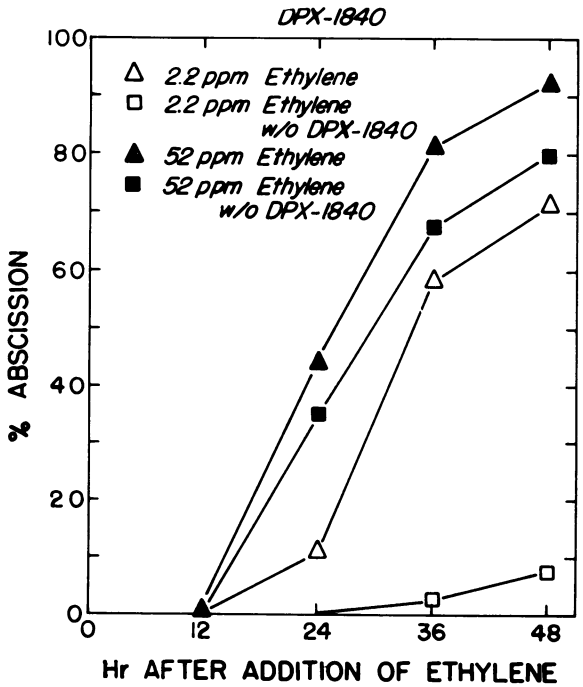

FIG. 6. Effect of auxin transport inhibitor DPX-1840 at 1 mM on abscission response to saturating and subsaturating levels of ethylene.

Table II. *Effect of Various Anti-Auxins and DPX-1840 on Leaf Abscission Induced by 0.1 M Ethrel 48 Hr after application of Anti-Auxins*

Treatment	Experiment and Anti-Auxin Concn			
	A	B	C	D
	1 mM	1 mM	1 mM	5 mM
	% Abscission[1]			
Control		21	32	10
Phenoxyacetate	63	42	44	8
2-Chlorophenoxyacetate	25	23	27	
4-Chlorophenoxyacetate	3	3	0	
2,5-Dichlorophenoxyacetate	18	17	16	
2,4,6-Trichlorophenoxyacetate	33	11	22	
2,6-Dichlorophenoxyacetate		26	38	8
2,4-Dichloroanisole		40	37	25
3,3a Dihydro-2-(p-methoxyphenyl)-8H-pyrazolo-(5,1-a)isoindol-8-one[2]		68	59	90

[1] Plants receiving neither anti-auxin nor Ethrel exhibited no leaf abscission during these experiments. Each anti-auxin treatment was also applied to plants receiving no Ethrel and all such exhibited no leaf abscission during these experiments.

[2] 1 mM.

As previously stated, plants treated with auxin transport inhibitors alone abscised no true leaves during the usual 3 or 4 day observation period after application of inhibitor. However, plants treated with DPX-1840 alone abscised leaves during the interval from 7 to 21 days after treatment while maintained in a greenhouse. All leaves which abscised were among the youngest, partially expanded leaves at the time of treatment, and there was more abscission with 10 mM than 1 mM DPX-1840. Lower levels of DPX-1840 caused neither abscission nor abnormal growth, but the two highest levels produced extensive growth of lateral buds to form distorted branches. New primary leaves exhibited growth symptoms similar to those produced by 2,4-D.

DISCUSSION AND CONCLUSIONS

The data here support the hypothesis that ethylene acts in natural abscission regulation by its well established capacity to inhibit auxin transport in the petiole (7). Once the auxin-ethylene balance has been changed in the abscission cells, then ethylene can proceed with its more direct actions of inducing synthesis and secretion of hydrolytic enzymes (2–4, 13, 21). The fact that a variety of auxin transport inhibitors will promote abscission over a range of concentrations, both with ethylene and ethylene derived from 2-chloroethanephosphonic acid (Figs. 1–3, 5), supports the model (7). The ability of the compounds to act when applied locally to petioles (Figs. 2, 3) and in particular their ability to potentiate the abscission of the oldest true leaf, which usually will not abscise in 48 hr at the ethylene levels employed in these tests, is further evidence that the critical action of these materials is to limit the auxin supply. This is further supported by the fact that the transport inhibitors did not cause any abscission by themselves during a 2- or 3-day test period but did so only when coupled with ethylene. Apparently, DPX-1840 at 10 mM and 1 mM reduced auxin transport sufficiently to cause abscission of incompletely expanded leaves beginning a week after treatment (text).

The auxin-transport inhibitors were more effective than anti-auxins in their ability to promote ethylene-induced abscission

(Table II). However, anti-auxins should also reduce the effective level of auxin by competing for auxin sites, and there was some promotion of Ethrel-induced abscission by these compounds. The anti-auxin which was most active in abscission promotion (Table II, phenoxyacetic acid) was among those with the least growth-promoting activity, highest minimum concentration to completely inhibit abscission, and no capacity to promote abscission in stage II explants (8). On the other hand, those compounds which were least active as abscission promoters in our tests (4-chlorophenoxyacetic acid, 2,5-D) were higher in growth promoting activity, lower in minimum concentration to completely inhibit abscission and, for 2,5-dichlorophenoxyacetic in particular, could produce a significant acceleration of abscission when applied to stage II petiole explants (8). All three of these latter properties were more strongly expressed by the more active auxins, 2,4-D and 2,4,5-trichlorophenoxyacetic acid (8). Abeles (1) has shown that, among a similar group of compounds, phenoxyacetic acid and 2,4,6-trichlorophenoxyacetic acid had little or no capacity to promote the synthesis of ethylene and abscission of cotton cotyledonary explants. In contrast, 2,4-D and 2,5-D were both active promoters of ethylene synthesis and highly active promoters of debladed petiole abscission (1). All of the same compounds were not used in all three studies; however, it is apparent that there are two completely different phenomena involved. The earlier studies (1, 8) were dealing primarily with abscission of debladed petioles caused by auxin-induced ethylene synthesis; thus, the best auxins or poorest anti-auxins were the most active abscission stimulators. Here we are dealing with potentiation to abscission by reduction of auxin supply or activity in the abscission zone, so the poorest auxins or best anti-auxins were the most active abscission stimulators (Table II). These studies also indicate the danger of projecting explant findings to intact plants; the best materials for petiole abscission (in stage II) were the poorest for leaf abscission and the reverse was also true.

That the experiments here are dealing with the balance between auxin and ethylene is supported by the data in Table I. There are levels of ethylene so low that DPX-1840 will not enhance abscission. As ethylene levels were raised, a concentration of DPX-1840 was found that promoted abscission, and the degree of promotion was proportional to its concentration. At still higher ethylene levels the concentration of DPX-1840, within the limits shown in Figure 4, had little effect on the degree of abscission. In this latter situation, ethylene had exceeded the threshold necessary to act at some minimum auxin level and further reduction in the auxin level was without a major effect. Finally, at saturating levels of ethylene, DPX-1840 had only a slight effect on abscission (Fig. 6). One should note, however, that abscission with only 2.2 μl/l ethylene was increased by DPX-1840 to nearly the level that was achieved with 52 μl ethylene per liter air. Thus, by reducing the auxin supply with a transport inhibitor, abscission will occur nearly as rapidly at 2.2 μl/l ethylene as at 52 μl/l ethylene. This also argues for the importance of the relative levels of auxin and ethylene in abscission control.

The results in Figure 6 tempt one to do the reverse experiment, that is, determine if auxin transport inhibitors will promote abscission in the presence of levels of ethylene which completely inhibit auxin transport. Unfortunately, such experiments are neither straightforward nor would the results be easy to interpret. Ethylene-mediated inhibition of basipetal auxin transport begins after a few hours exposure and increases with time; at 24 hr, transport inhibition is not complete with ethylene levels up to 1 ml/l, while considerable abscission will occur by the same time with ethylene levels below 100 μl/l (See ref. 7, Figs. 1 and 2, and ref. 9 and 10 therein). Apparently, removal of all auxin transport capacity is not necessary for ethylene-induced abscission. It is not possible to experiment with plants fully devoid of auxin transport capacity due to exposure to ethylene and to approach this state brings one closer and closer to the actual event of abscission.

The results in Figure 6 raise the question of whether the events of the Beyer-Morgan model (7) are in series or parallel. One possibility is that 52 μl/l of ethylene is nearly as effective as DPX-1840 in reducing auxin transport. However, the DPX-treated plants presumably already had auxin transport inhibited, since DPX was applied 24 hr before ethylene; thus, one might consider that the inhibition of auxin transport and promotion of abscission layer events occurred simultaneously in the control plants at 52 μl/l ethylene nearly as rapidly as the abscission layer events occurred in the DPX-treated plants. This could mean either that in normal leaf abscission the inhibition of transport and induction of hydrolytic enzyme synthesis are parallel, a situation not entirely in harmony with our hypothetical model (7), or that levels of ethylene can be reached which saturate the enzyme-induction systems (abscission zone events) while overriding the effect of native auxin present. In this latter situation one would be dealing with nonphysiological levels of ethylene and a reduction of auxin levels would be less important. This explanation is favored by the fact that the ability of rather high levels of ethylene to cause leaf (11) or explant petiole (19) abscission can be blocked by supplying exogenous auxin.

Until tested further, we will follow the working hypothesis that inhibition of auxin transport (or other reduction of auxin levels) is a functional event controlled by ethylene in natural, intact leaf abscission which precedes the direct action of ethylene on events in the abscission zone. The failure of the auxin transport inhibitors to cause any promotion of abscission by themselves during the initial 2 to 3 days after application is suggestive that a rise in ethylene production rather than a decline in auxin levels is the key event which sets the abscission mechanism in motion.

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