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Auxin and Ethylene Regulation of Petiole Epinasty in Two Developmental Mutants of Tomato, *diageotropica* and *Epinastic*[1]

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ABSTRACT

The epinastic growth responses of petioles to auxin and ethylene were quantified in two developmental mutants of tomato (*Lycopersicon esculentum* Mill.). In the wild type parent line, cultivar VFN8, the epinastic response of excised petiole sections was approximately log-linear between 0.1 and 100 micromolar indole-3-acetic acid (IAA) and 2,4-dichlorophenoxyacetic acid (2,4-D) concentrations, with a greater response to 2,4-D at any concentration. When ethylene synthesis was inhibited by aminoethoxyvinylglycine (AVG), epinasty was no longer induced by auxin, but could be restored by the addition of ethylene gas. In the auxin-insensitive mutant, *diageotropica* (*dgt*), no epinastic response to IAA was observed at IAA concentrations that effectively induced epinasty in VFN8. In the absence of added IAA, epinastic growth of *dgt* petioles in 1.3 microliters per liter exogenous ethylene gas was more than double that of VFN8 petioles. IAA had little additional effect in *dgt*, but promoted epinasty in VFN8. These results confirm that tomato petiole cells respond directly to ethylene and make it unlikely that the differential growth responsible for epinasty results from lateral auxin redistribution. The second mutant, *Epinastic* (*Epi*), exhibits constitutively epinasty, cortical swelling, and root branching symptomatic of possible alternation in auxin or ethylene regulation of growth. Only minor quantitative differences were observed between the epinastic responses to auxin and ethylene of VFN8 and *Epi*. However, in contrast to VFN8, when ethylene synthesis or action was inhibited in *Epi*, auxin still induced 40 to 50% of the epinastic response observed in the absence of inhibitors. This indicates that the target cells for epinastic growth in *Epi* are qualitatively different from those of VFN8, having gained the ability to grow differentially in response to auxin alone. The *dgt* and *Epi* mutants provide useful systems in which to study the genetic determination of target cell specificity for hormone action.

Epinasty, broadly defined as growth in a direction away from the plant axis, is the result of differential cell growth in a plageotropic organ. Epinastic movement of petioles is the result of greater expansion of adaxial cells as compared to abaxial cells in specific regions of the petiole (10, 17). In the tomato, both auxin and ethylene will induce petiole epinasty (9, 11, 13), with auxin action being dependent upon induction of ethylene synthesis (2, 18). Contrasting theories for the mechanisms of ethylene and auxin action in the induction of epinasty have been proposed. In one hypothesis (reviewed in 10), ethylene alters lateral auxin transport, resulting in a transverse auxin gradient within the petiole; higher auxin levels in the adaxial portion of the petiole result in greater adaxial growth and hence, bending. Attempts to detect transverse auxin gradients of the magnitude required for differential growth have generally failed, leading to the proposal that epinastic growth is the result of a differential growth response of cells in the adaxial and abaxial halves of the petiole to ethylene (16). Osborne (15) has suggested that the adaxial petiole cells responsible for epinastic growth are so-called type III target cells, which elongate in response to ethylene, but growth is dependent upon the presence of auxin. In several semiaquatic plants, type III cells will also respond directly to auxin without a requirement for elevated ethylene synthesis, but the simultaneous presence of auxin and ethylene gives an additive response (20). It has not been thoroughly tested whether the petiole cells of tomato, which are highly sensitive to both auxin and ethylene, conform fully to this model for type III cells. The fact that auxin-induced epinasty in tomato petioles requires ethylene synthesis, while growth of other type III cells apparently does not, indicates that the tomato cells may differ from those of the semiaquatic plants wherein type III cells were defined.

Separation of the roles of auxin and ethylene in petiole epinasty has been complicated by the intimate relationship between auxin effects on ethylene synthesis and possible ethylene effects on auxin transport or synthesis ((10, 17). To avoid these difficulties, we have assessed epinastic growth responses to auxin and ethylene in a single-gene developmental mutant of tomato which exhibits altered hormone sensitivity. The *dgt*[3] mutant is partially dominant, isogenic with parent line VFN8, and characterized by horizontal growth of stems and roots, dark green, hyponastic leaves, and a lack of lateral roots (23, 24). The primary physiological lesion in this mutant is a marked insensitivity to exogenous auxin with respect to ethylene synthesis and hypocotyl elongation (12, 25). Since *dgt* tissues show only a slight stimulation of ethylene production in response to high auxin concentrations, the effects of auxin and ethylene on epinastic growth can be investigated independently without the use of inhibi-

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[3] Abbreviations: *dgt*, *diageotropica*; *Epi*, *Epinastic*; AVG, aminoethoxyvinylglycine; STS, silver thiosulfate; ethephon, (2-chloroethyl)phosphonic acid.

tors. In addition, since growth itself is highly insensitive to auxin in *dgt*, it should be possible to determine whether auxin action is required for the response to ethylene.

The second mutant utilized in this study is the *Epi* mutant of the tomato. This partially dominant mutant, also isolated from VFN8, is characterized by severe epinasty of leaves, swelling of stems and petioles, prolific branching of roots (our unpublished data), and overproduction of ethylene in the apical region of the plant (6). Sensitivities to exogenous auxin (induction of ethylene synthesis) and ethylene (inhibition of root growth) are similar in *Epi* and VFN8, but inhibitors of ethylene synthesis or action are unable to completely revert the *Epi* phenotype to the wild type (5, 6). Although the primary genetic lesion in *Epi* remains unknown, its constitutive epinasty and other morphological features are consistent with disrupted regulation of cell expansion in specific target tissues (our unpublished data). We have compared the auxin- and ethylene-induced epinastic growth responses of *Epi* petioles to those of VFN8 and *dgt* to further characterize the physiological consequences of the *Epi* mutation and to determine whether changes in auxin or ethylene action are responsible for its developmental features.

MATERIALS AND METHODS

Plant Material

The *Epi* mutant of the tomato (*Lycopersicon esculentum* Mill.) was discovered as a spontaneous mutant in cultivar VFN8. Seeds of VFN8 and the *dgt* mutant were obtained from Dr. C. M. Rick of the Department of Vegetable Crops, University of California, Davis. Seeds of each genotype were germinated in vermiculite and transplanted at the cotyledon stage into 0.5 L pots containing a soil mix and were fed weekly with a complete nutrient solution. Plants were grown in a greenhouse under ambient light conditions with day/night temperatures approximating 20 and 30°C, respectively.

Petiole Epinasty Assay

Stem sections with attached petioles were excised from the third, fourth, and fifth nodes above the cotyledonary node 6 weeks after sowing. Petioles were debladed 1 cm from the axil and the stems trimmed so that 1 cm of stem remained above the axil and 1 to 2 cm of stem remained below. Excised sections were incubated on a low-speed shaker in a volume of distilled water that kept petioles wet, but not submerged, for at least 1 h prior to use in the epinasty assay. Inhibitors of ethylene synthesis (AVG) and of ethylene action (STS) were supplied during this 1-h incubation period at appropriate concentrations in water. STS solutions were prepared by mixing $AgNO_3$ and sodium thiosulfate solutions at a concentration ratio of 1:4. The concentrations reported are those of the silver component.

After the incubation period, the excised stem-petiole sections were supported in an upright position in inverted serum caps and were either enclosed in an aerated humidity chamber or placed in 0.5-L jars or 6-L desiccators. Petiole angles were measured on the explants as the degrees from vertical of the adaxial portion of the first centimeter of the petiole. Angles were measured just prior to treatment and again after 8 h of treatment; epinastic growth was the difference between these measurements. Agar blocks (0.4%) were placed on the cut petiole surface, and auxins, when used, were added to the agar. Ethylene treatments greater than 1 μL/L were made by injecting ethylene into the sealed 0.5- or 6-L vessels to the desired concentration. The 1 μL/L ethylene treatment was imposed by flushing 6-L desiccators with air containing 1 μL/L ethylene, then maintaining a constant flow rate (1 L/h) of 1 μL/L ethylene in air throughout the treatment period.

Ethylene Measurements

Ethylene evolution rates of the excised stem-petiole explants during the epinastic growth response were determined by placing individual sections (approximately 0.7 g each) into sealed 8- or 22-mL vials for the final 2 h of the treatment period. Ethylene concentrations were measured in 1 mL samples of the gas phase by gas chromatography using a Carle 211 gas chromatograph with a flame ionization detector and an activated alumina column.

Ethephon Treatment

Five-week-old greenhouse-grown plants of VFN8 and *Epi* were sprayed daily with 250 mg/L ethephon. Petiole angles and petiole diameters 5 mm from the stem were measured on the second and third oldest leaves prior to ethephon treatment and again after 48 h. Control plants were treated identically using water instead of ethephon solution.

RESULTS

VFN8

Epinastic growth was measured in petiole explants of VFN8 in response to a range of concentrations of IAA and 2,4-D (Fig. 1). The epinastic response was approximately linear with the logarithm of the auxin concentration, with 2,4-D exhibiting significantly greater activity at each concentration (Fig.

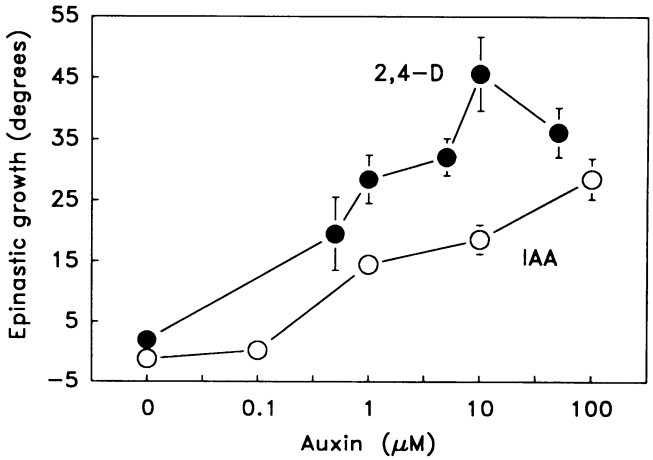

Figure 1. Epinastic growth responses of VFN8 petiole explants 8 h after treatment with IAA or 2,4-D. Data are means of eight observations ± SE.

1). The promotion of epinasty by 50 μM 2,4-D was completely inhibited by pretreatment with 100 μM AVG, an inhibitor of ethylene synthesis, but could be fully restored by 5 μL/L exogenous ethylene (Table I).

dgt

The epinastic growth responses of VFN8 and *dgt* to a range of IAA concentrations were compared (Fig. 2). The response of *dgt* to auxin was only significant at 10 μM IAA, where the response was still approximately half of that observed in VFN8 (Fig. 2). This slight promotion of epinasty by high IAA levels in *dgt* was completely inhibited by pretreatment with AVG (data not shown), indicating that it was dependent upon ethylene synthesis. Exogenous ethylene (1.3 μL/L) promoted an epinastic response in auxin-depleted VFN8 petioles which was approximately one-half as large as the response promoted by 10 μM IAA alone, and the effect of added ethylene on auxin-treated petioles was synergistic at 0.1 μM IAA and additive at higher auxin concentrations (Fig. 2). The response of *dgt* to ethylene in the absence of exogenous auxin was more than double that of VFN8 (Fig. 2). Again, a response to IAA was observed only at 10 μM, which was likely due to endogenous ethylene production, since 1.3 μL/L was not a saturating level of ethylene (Table I).

Table I. *Effect of AVG and Ethylene on 2,4-D-Induced Petiole Epinasty in VFN8*

Petiole explants from VFN8 were pretreated for 1 h with or without the ethylene synthesis inhibitor AVG and then received 2,4-D (applied in agar to the cut distal end of the petiole) and ethylene as indicated. Epinasty was determined 8 h after 2,4-D treatment. Results represent the means of eight observations ± 95% confidence limits.

2,4-D	AVG	C_2H_4	Epinasty
μM		μL/L	degrees
0	0	0	7 ± 5
50	0	0	43 ± 5
50	100	0	10 ± 5
50	100	1	24 ± 5
50	100	5	43 ± 14

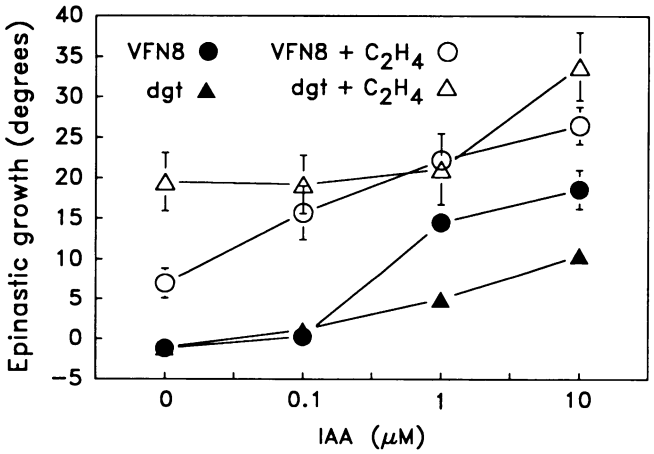

Figure 2. Epinastic growth responses of VFN8 and *dgt* petiole explants to IAA with and without 1.3 μL/L added ethylene. Data are means of eight observations ± SE.

Epi

Epinastic growth at 1 and 10 μM IAA without added ethylene did not differ significantly between VFN8 and *Epi* (Table II). In both genotypes, increasing ethylene alone also promoted equivalent levels of epinastic growth, with saturation occurring between 3 and 10 μL/L (Table II). Ethylene enhanced the effectiveness of auxin in both genotypes, indicating that auxin-induced ethylene did not saturate the growth response (Table II; Fig. 2).

In addition to epinasty, petioles of *Epi* also exhibited pronounced swelling in response to ethylene. When severe epinasty was induced in intact VFN8 plants by ethephon application, no increase in petiole diameter occurred beyond that due to normal growth (Table III). In *Epi*, on the other hand, diameters of ethephon-treated petioles increased at almost three times the rate of untreated petioles over the 48-h period (Table III). The data of Table III also illustrate the greater initial petiole angles and diameters of *Epi* as compared to VFN8.

Inhibition of ethylene synthesis by pretreatment with 100 μM AVG prevented the induction of epinasty by 2,4-D in VFN8, whereas in *Epi*, only 50 to 60% of the auxin-induced epinastic growth was inhibited by AVG (Table IV). Increasing the AVG concentration to 500 μM had no additional effect, and ethylene synthesis rates were approximately one-tenth of control levels in both *Epi* and VFN8 (Table IV). Identical results were obtained using IAA instead of 2,4-D (data not shown).

The effectiveness of STS, an inhibitor of ethylene action,

Table II. *Comparison of the Epinastic Growth Responses of VFN8 and Epi to IAA and Ethylene*

Epinastic growth was determined in petiole explants of VFN8 and *Epi* 8 h after treatment with IAA or ethylene. Differences in treatment responses were analyzed by *F*-test (n = 8). Differences between genotypes were not significant; for comparison among individual means, LSD = 9.

Treatment		Genotype	
IAA	C_2H_4	VFN8	*Epi*
μM	μL/L	epinasty, degrees	
0	0	3	2
0	1	13	13
0	3	24	24
0	10	27	12[a]
1	0	8	13
1	1	26	27
1	3	27	26
1	10	33	28
10	0	16	21
10	1	33	32
10	3	36	30
10	10	27	33

[a] This mean was spuriously low in the experiment shown. In other experiments, 10 μL/L ethylene saturated the epinastic response of *Epi*.

Table III. *Epinasty and Petiole Swelling Induced in VFN8 and Epi Tomato Plants by Ethephon*

Plants were sprayed with either 250 mg/L ethephon or water three times during a 48-h period. Measurements were made prior to treatment and after 48 h. Values are means ± 95% confidence limits of 10 replicates from the second and third oldest leaves.

Character	Time After Treatment	VFN8		*Epi*	
		Control	C_2H_4	Control	C_2H_4
	h	*degrees*			
Epinasty	0	53.3 ± 3.4	53.5 ± 2.0	62.0 ± 5.6	60.0 ± 4.7
	48	59.7 ± 3.8	131 ± 7.2	71.4 ± 7.2	117 ± 3.6
	Δ[a]	6.4 ± 1.8	77.5 ± 7.5	9.4 ± 2.3	57.0 ± 3.6
		mm			
Petiole diameter	0	3.89 ± 0.14	3.95 ± 0.09	5.34 ± 0.36	5.06 ± 0.27
	48	4.32 ± 0.41	4.31 ± 0.25	5.65 ± 0.36	5.97 ± 0.18
	Δ[a]	0.44 ± 0.38	0.36 ± 0.16	0.32 ± 0.14	0.91 ± 0.18

[a] Difference between the initial and 48-h measurements.

Table IV. *Effect of AVG on 2,4-D-Induced Epinasty in VFN8 and Epi*

Petiole explants from VFN8 and *Epi* were pretreated for 1 h with 0, 100 or 500 μM AVG. Epinasty and ethylene production were measured 8 h after treatment with 100 μM 2,4-D. Control petioles received neither AVG nor 2,4-D. Mean separations by Duncan's multiple range test ($P < 0.05$, $n = 8$).

Genotype	Control	100 μM 2,4-D		
		0 AVG	100 μM AVG	500 μM AVG
		epinasty (degrees)		
VFN8	2 e	37 a	6 de	4 e
Epi	1 e	30 b	12 cd	16 c
		ethylene (nL/g·h)		
VFN8	0.9 cd	2.6 b	0.16 de	0.11 e
Epi	1.0 c	8.6 a	0.34 cde	0.12 e

in preventing epinasty was also assessed. VFN8 exhibited dosage-dependent inhibition by STS of epinasty induced by 10 μM IAA, with complete inhibition at 5 mM STS (data not shown). Significant inhibition of epinastic growth by STS was also observed in *Epi*, but approximately 25% of the maximum response remained at 5 mM STS (data not shown). To assess whether auxin was acting in a dosage-dependent fashion on epinasty in the presence of 5 mM STS, increasing levels of auxin were applied after STS pretreatment. No response to auxin was observed in VFN8 at any IAA concentration following treatment with 5 mM STS, while a significant linear response to increasing IAA concentrations occurred in *Epi* (Fig. 3). STS was completely effective in preventing ethylene-induced epinasty in both VFN8 and *Epi* (data not shown).

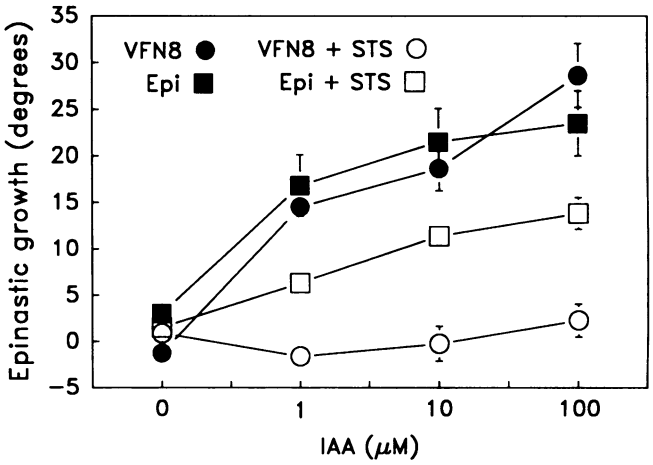

Figure 3. Comparison of epinastic growth responses to IAA in petiole explants of VFN8 and *Epi* after pretreatment with 5 mM STS. Data are means of eight observations ± SE.

DISCUSSION

Our results confirm that in normal tomato genotypes, induction of petiole epinasty by auxin is dependent upon ethylene synthesis (Tables I, III) or action (Table IV; Fig. 3), in agreement with previous reports (2, 11, 21). The experiments with 2,4-D and *dgt* are pertinent to the debate concerning the mode of action of ethylene in inducing epinasty. One view (reviewed in 10) is that ethylene alters the lateral transport of auxin within the petiole, resulting in its accumulation in the adaxial side, where it stimulates differential growth. The alternative view (reviewed in 17) is that ethylene acts directly on the petiolar cells to induce differential expansion. The former model assumes that the cells are uniformly responsive to auxin, with concentration differences resulting in differential growth rates. The latter hypothesis assumes a target cell model (15) wherein individual cells can respond differentially to uniform hormone concentrations. The greater epinastic response of VFN8 petioles to 2,4-D as compared to IAA (Fig. 1) argues against the lateral auxin transport model and for the ethylene action model, since polar transport of 2,4-D is much slower than transport of IAA (14), but 2,4-D is more effective than IAA in sustaining elevated rates of ethylene synthesis (1).

Further evidence for the lack of an auxin requirement for petiole epinasty is provided by the *dgt* mutant. The genetic lesion in *dgt* results in a markedly decreased capacity of hypocotyl cells to grow or synthesize ethylene in response to exogenous auxin within a concentration range which is highly effective in VFN8 (12). The relative insensitivity of petioles of *dgt* to high auxin concentrations (Fig. 2) can be attributed

to the poor ability of auxin to stimulate ethylene synthesis in this mutant (12, 25), but also makes it highly unlikely that the differential growth responsible for epinasty results from small lateral changes in IAA content within the petiole. It is also unlikely that failure of auxin transport in *dgt* prevented auxin applied distally on the cut petiole surface from reaching the responsive cells at the base of the petiole, as it has recently been shown that the capacity for auxin transport in *dgt* hypocotyls actually exceeds that of VFN8 (3). Exposure to ethylene increased both auxin uptake and retention in *dgt* relative to VFN8 (3). The ability of *dgt* petioles to become epinastic in response to ethylene, despite their insensitivity to auxin, demonstrates that epinasty in the tomato is a direct response to ethylene. Since ethylene will at least partially normalize the *dgt* phenotype (25), it could be argued that exposure to ethylene restores auxin sensitivity to the tissue. However, this was not the case with *dgt* hypocotyls, where the presence of ethylene did not alter their insensitivity to growth stimulation by auxin (12).

In VFN8, endogenous auxin may be required for a maximal epinastic response to ethylene. The petiole explants used in our assay were incubated to deplete endogenous auxin levels prior to treatment. Without added auxin, VFN8 petioles showed only a weak epinastic response to ethylene, and auxin concentrations too low to elicit epinasty alone promoted it in the presence of ethylene (Fig. 2; Table II). This could mean that auxin is required for cell expansion, but the differential growth leading to epinasty is controlled by ethylene. Interestingly, *dgt* petioles responded markedly to ethylene regardless of the presence of added auxin (Fig. 2). This may represent a quantitative enhancement in sensitivity of *dgt* tissues to ethylene, in agreement with the results of Zobel (25, 26). It may also indicate that *dgt* tissues do not require auxin action for growth or that only some aspects of auxin action are defective in *dgt* (23).

We also investigated the epinastic growth response in the *Epi* tomato mutant. Many constitutive features of *Epi* are similar to symptoms of ethylene exposure in the tomato, suggesting that altered regulation of ethylene-induced cell growth may be a key factor in the distinctive morphology of *Epi* plants (our unpublished data). Endogenous IAA content is not significantly greater in *Epi* than in VFN8 (7), but overproduction of ethylene, within the plant apex, has been detected (6). While the threshold auxin concentration for the induction of ethylene synthesis was similar in *Epi* and VFN8, the rate of ethylene synthesis was greater in *Epi* than in VFN8 at equivalent auxin concentrations (6). Since *Epi* produced more ethylene in response to auxin (Table IV), greater epinastic growth might be anticipated at each auxin level. Contrary to expectation, no significant differences between VFN8 and *Epi* in response to 1 and 10 μM IAA were observed (Table II). Similar levels of epinastic response also occurred at each ethylene concentration in the two genotypes, although *Epi* appeared to saturate at somewhat lower ethylene concentrations (Table II). Consistent with the observation of Kazemi and Kefford (11) in normal tomato petioles, auxin did not induce additional epinastic growth in saturating ethylene levels.

In addition to the normal epinastic response to ethylene, target cells in *Epi* petioles show two additional responses not evident in VFN8. The cells of the lateral and adaxial cortex of the petioles swell upon exposure to ethylene in *Epi*, but not in VFN8 (Table III). In some cases, this swelling extends completely around the node as well (data not shown). Enlarged and irregular cells are present in the stem cortex in *Epi*, even without exogenous ethylene (our unpublished data). Ethylene appears to exacerbate this constitutive tendency toward swelling of cortical cells. Petiole cells of *Epi* also exhibit differential growth resulting in epinasty in response to auxin even when ethylene synthesis or action are prevented, while in VFN8, auxin-induced epinasty is completely dependent upon ethylene synthesis and action (Tables I, IV; Fig. 3). Petiole epinasty in *Chrysanthemum* and poinsettia (*Euphorbia pulcherrima* Willd.) and epinasty of tomato leaf midribs can also be induced by auxin when ethylene synthesis is prevented (8, 11, 19). Thus, there are precedents for the existence of tissues with the capacity to grow differentially in response to auxin alone. It appears that in *Epi*, the normal differential growth response to ethylene is present, but is superimposed on an abnormal (for tomato petioles) direct response to auxin. If this change in the auxin/ethylene regulation of cell expansion is generalized throughout *Epi* plants, it could well explain the abnormal morphological features of the plant.

Auxin and ethylene are thought to control the rate and direction of cell expansion by effects on the extensibility and orientation of cell wall microfibrils (4, 22). *Dgt* cells have lost sensitivity to auxin while retaining sensitivity to ethylene. *Epi* cells may have a constitutive ethylene-like response and a qualitatively altered response to auxin. Elucidation of the normal molecular or cellular functions of these genes should provide insight into how the extent and orientation of cellular growth is controlled or coordinated.

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