GENOTYPIC VARIATION IN THE FLOWER DELAYING EFFECT OF ETHEPHON

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The ethylene releasing compound ethephon is a potent inhibitor of flower initiation in the early developing line of peas, line 58 (6). However, an endogenous role for ethylene in the control of flowering in peas has not yet been found (7). For this reason it was decided to examine how a range of flowering genotypes responded to applied ethephon, with the hope that some correlation between the magnitude of the flowering response and the flowering genotype could be found.

The growing techniques used were similar to those previously used at Hobart (2, 5). Treatment with ethephon was performed by applying 10 mkl of ethanol containing the required quantity of ethephon to the dry testa. After the ethanol evaporated the seeds were planted 2 cm beneath the surface of the growth medium. Plants decotyledonized after 18 h imbibition were grown on White's nutrient agar medium until leaf 4 was fully expanded.

The results in Tables 1, 2, and 3 show that ethephon is capable of increasing the flowering node of lines 58 (flowering genotype lf e sn hr), 59 (lf E sn hr), 64 (lf E sn hr), 60 (lf E Sn hr), 53 (lf e Sn hr), 51 (lf E sn Hr), and 7 (lf^a E Sn hr) under an 8 h photoperiod and of lines 58, 68 (lf e sn hr), and 59 under continuous light (all delays significant at the 0.001 level when 480 ug of ethephon was used). However, although ethephon is general in its ability to delay the flowering node, the size of the delay varied considerably from one line to another within one experiment. For example, the three phenotypically similar lines, 58 (lf e sn hr), 68 (lf e sn hr), and 59 (lf E sn hr) differed significantly in the extent they were delayed by ethephon (Tables 1 and 2). These differences in the flowering response between the lines did not appear to be directly associated with the presence or absence of the individual genes E, Sn, or Hr. Further, the balance of the flowering hormones existing in the plant during the early growth did not appear to be implicated in the differential response in the early region since line 58 plants were delayed to a later node than were line 60 plants even though line 60 cotyledons and shoots have been shown to produce a more inhibitory balance of the flowering hormones than line 58 (2, 8). It appears other as yet undetermined genetic systems are responsible for the largest part of these different responses as illustrated by the degree of difference between lines 68 and 58 (both genotype lf e sn hr). Consequently, the present study does not indicate where, if at all, endogenous ethylene plays a role in controlling flowering in peas.

As well as having varying effects on the size of the flowering delay it is interesting to note that a particular concentration of ethephon also had differing effects on the vegetative growth of the different lines. This is illustrated by the fact that the length between nodes 1 and 6 was consistently reduced by the greatest percentage in line 58 (Tables 1 and 2). However, this measurement does not appear to tell the whole story, since plants of lines 58 and 55 were very "sick" in appearance when treated with 480 ug of ethephon even after 4 or 5 weeks growth, while lines 51y and 68 appeared almost unaffected by this treatment at this time. Lines 60, 59, and 64 were somewhat intermediate in their response between these two groups. Whether this differing vegetative response to ethephon is responsible for the differing flowering responses (e.g. between lines 58 and 68) is unclear.

Table 1. The mean node of first initiated flower (FI) ± S.E. and length between nodes 1 and 6 (LI-6) ± S.E. for lines 58 (lf e sn hr), 59 (lf E sn hr), 64 (lf e sn Hr), 60 (lf E Sn hr), 53 (lf e Sn hr) and 51y (lf E sn Hr). The plants were treated once with 0, 24, 96 or 480 µg of ethephon. The photoperiod was 8 h.

		58			59			64			60			53			51y		
Character	Treatment	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n
FI	0	10.06	±.06	17	9.06	±.06	17	9.80	±.11	15	11.00	±.00	17	20.53	±.75	17	9.22	±.10	18
FI	24/µg	12.44	±.22	18	10.47	±.12	17	10.82	±.36	17	12.06	±.06	17	21.38	±.50	13	10.59	±.15	17
FI	96/µg	14.44	±.22	18	10.94	±.10	18	12.13	±.56	15	12.89	±.21	18	22.94	±.44	16	11.06	±.19	18
FI	480/µg	15.25	±.18	12	11.69	±.21	13	13.09	±.51	11	13.38	±.14	13	23.13	±.65	15	12.29	±.19	17
LI-6	0	7.56	±.19	17	8.89	±.26	14	6.38	±.10	14	5.11	±.06	16	5.03	±.15	17	7.28	±.12	18
LI-6	24/µg	5.79	±.11	18	7.45	±.17	16	5.58	±.17	17	4.76	±.17	17	5.37	±.22	14	5.69	±.13	17
LI-6	96/µg	5.73	±.16	18	7.64	±.12	17	5.57	±.19	15	4.80	±.15	17	5.56	±.22	16	5.53	±.13	18
LI-6	480/µg	4.82	±.21	12	6.35	±.17	13	5.42	±.22	11	4.61	±.28	13	4.93	±.18	11	4.85	±.15	17

Table 2. The mean node of first initiated flower (FI)±S.E. and the length between nodes 1 and 6 (L1-6)±S.E. for lines 58 (lf e sn hr), 59 (lf E sn hr) and 68 (lf e sn hr) treated with either 0 or 480 µg of ethephon. The percent decrease in the internode length caused by Ethrel treatment is also indicated. The plants received continuous light from the time the plumules broke the surface of the growing medium.

		L58			L59			L68		
Character	Treatment	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n	$\bar{x}$	±S.E.	n
FI	0	10.29	±.11	17	9.11	±.08	18	9.89	±.08	1
FI	480/µg	13.25	±.37	8	11.58	±.15	12	11.56	±.18	1
L1-6	0	9.82	±.25	18	11.77	±.30	17	7.99	±.23	1
L1-6	480/µg	4.05	±.16	8	5.06	±.27	10	4.58	±.10	1
Percent decrease	L1-6	59			52			43		

Table 3. The mean node of first initiated flower ± S.E. for L7 plants (lf^a E Sn hr) either left intact (C), decotyledonized on day 0 (Decot 0) or day 5 (Decot 5), or treated on the cotyledons with 4 µg of ethephon. The photoperiod was 8 h.

C			Decot 0			Decot 5			Ethrel		
$\bar{x}$	± S.E.	n	$\bar{x}$	± S.E.	n	$\bar{x}$	± S.E.	n	$\bar{x}$	± S.E.	n
6.33	± .13	15	7.38	± .11	21	6.43	± .14	14	7.36	± .17	14

Two facets of the data are perhaps worthy of further comment. Firstly, apart from the untreated plants, the flowering node means for line 64 plants have large standard errors (Table 1), the flowering nodes varying from 10 to 16 with only one plant out of 43 flowering from node 14. This bimodality continued to occur even within the progeny from a single plant. It is suggested that a form of impenetrance is occurring in which ethephon either lowers the ratio of promotor to inhibitor reaching the apex to a level close to the threshold for flowering or lowers the threshold itself. Plants will then either flower in the early region (in this case nodes 10-13) when the cotyledon are the major source of the flowering hormones or not until the ratio coming from the shoot becomes promotory (nodes 15-18). This occurs because the cotyledons of line 64 under an 8 h photoperiod produce a more promotory balance of the flowering hormones than does the young shoot (5). A very small proportion of untreated plants of this genotype have also been reported to flower above node 15 (3) presumably for the same reason as given above. Although the range of the possible flowering nodes is smaller, this situation is analogous to that observed in intact line 61a (genotype lf e Sn hr) plants under an 8 h photoperiod (4,7).

The second point worth further comment is that the data in Table 3 indicate that the flowering node of line 7 (lf^a E Sn hr) is not determined (at least in all plants) before germination since treatment with 80 mkg of ethephon and decotyledonization after 18 h imbibition were both able to significantly delay the flowering node (at the 0.001 level). The number of nodes laid down in the apex after 24 hours imbibition was 6.11 +/- 0.11 (from a sample of 9 plants) indicating that an alteration in the flowering node is possible until very close to the time of initiation (node 6.33 +/- 0.13 in the controls). Decotyledonization on day 5 resulted in no significant alteration of the flowering node presumably because the plants had already initiated. Plants dissected on the 5th day possessed 8.13 +/- 0.13 nodes (sample of 8 plants). It should however be noted that in no case could the typical "bulge" of a flower primordium be seen in the leaf axil during these dissections suggesting axillary bud development lags substantially behind the development of leaf primordia in this particular line. This would seem different from the early developing and late lines dissected where the flower bud at a particular node is normally observable by the time the leaf primordium is initiated. It raises the possibility that the nature of the axillary bud (either vegetative or floral) may not be determined until after the leaf primordium has been initiated in line 7 although on the present evidence the determination would be made before day 5.

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