FASCIATION AND HETEROSIS IN PEA

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Under the climatic conditions of Central Europe, our fasciated mutant 489 C always causes a strong heterotic effect in stem length, seeds per plant, and other characters when crossed with the initial line (IL) 'Dippes gelbe Viktoria', other lines, or different mutants (Fig. 1). Mutants 251 A, 123, and other fasciated forms genetically comparable with mutant 489 C cause a similar, but in some cases clearly weaker, heterotic effect (2).

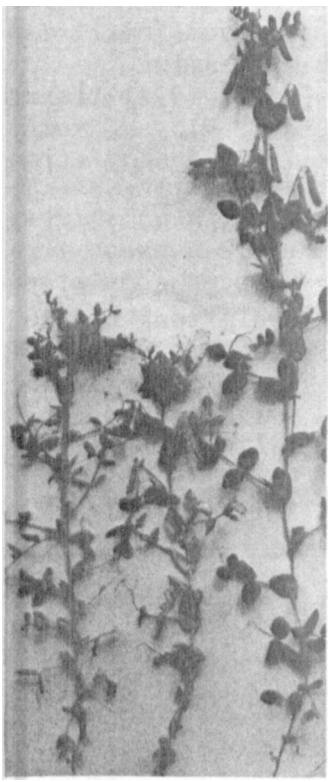

Fig. 1.

489C 1201A 489C x 1201A

As perhaps more than 15 genes are mutated in 489 C, it is not possible to say prima facie whether one or more of the genes causing fasciation or other unrelated mutant genes contribute to the heterotic effects. Nor is it clear whether the phenomenon is caused by a) heterozygosity per se, b) dominant factors "hidden" or impeded by detrimental recessive genes in the same mutant [most of the 15 genes have mutated to the recessive state, as, for instance, the genes for fasciation, but a few of them are dominant in relation to the IL, as lateness in flowering and ripening, sensitivity to day length (4), and, perhaps, the genes for increased stem length], c) gene interactions, or d) a combination of the above.

In seeking answers to these questions, we used the following mutants and recombinants for our studies (all forms are derived from Dippes gelbe Viktoria): a) strongly fasciated mutants 489 C, 33 A, b) linearly fasciated mutant 251 A, c) linearly fasciated recombinants R 859 (derived from 123 x 46 C), R 661 (derived from 489 C x 26), and R 667 (derived from 489 C x 169), d) weakly fasciated recombinants, stem bifurcated, R 161 (derived from 489 C x 1201 A) and R 177 (derived from 489 C x 1202 A), e) other mutants 176 A—narrow leaves, flowers, and pods, 1201 A—stem bifurcated, 1001—thousand-grain weight increased.

The results of these crosses are shown in Fig. 2 and can be summarized as follows: a) 33 A, R 161, R 661, and R 859 do not cause heterosis; the small deviations are well in the range of the IL. This means that the genetic causes for strong, linear, and weak fasciation are not necessarily correlated with the heterotic effects, b) R 177 x IL, etc., do not show heterosis in length although the character "seeds per plant" seems to show significant heterosis. For final analysis the crosses should be repeated with more extensive material. If verified, there would be no strict correlation between heterosis in length and yield, c) linearly fasciated R 667 seems to cause a strong heterotic effect, comparable to that of 489 C x IL, etc. However, 667 x IL is not very representative as we had only nine

plants in F1. According to the results, under a) the genetic cause for fasciation may not be the direct cause of this heterosis either.
Segregation: Wherever a marked heterosis effect was evident in F1, all the segregations studied so far in F2 showed a majority (sometimes more than three-quarters) of plants as long as the F1 hybrids. However, a simple 3:1 segregation is excluded by several hypostatic genes segregating for shorter plants of different length.

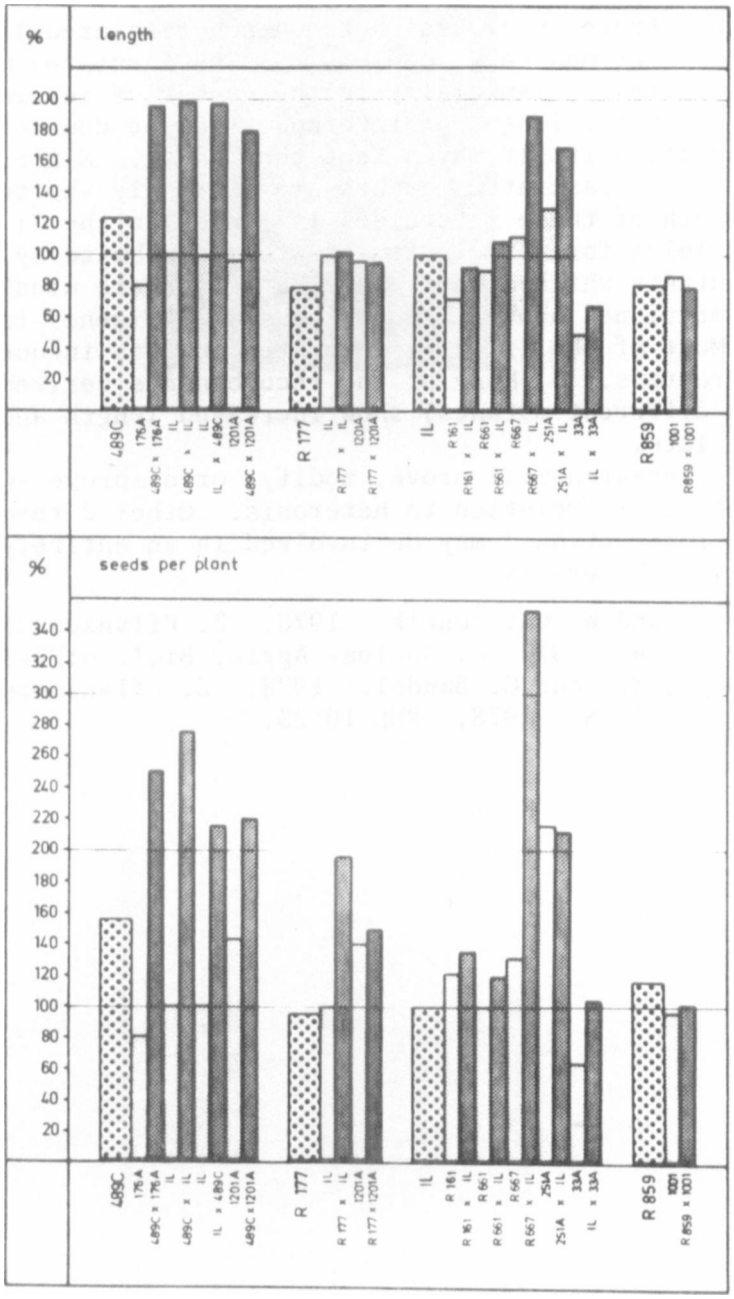

Fig. 2. F1's of the crosses

The following facts seem to support the working hypothesis for special importance of the genes for increased stem length and, perhaps, lateness: 1) Where there was no detectable heterosis in length in the F1, no long plants were found in F2; unusual length doesn't seem to be a simple hypostatic factor. None of these F1's was late. 2) The height of the hybrids was, of course, a function of internode number and—even more important in this case—internode length. Keeble and Pellew (1910) found both characters to show dominant inheritance in crosses between non-fasciated lines. The situation is, however, sometimes very complex. 3) The fasciated mutants causing heterosis show an increase, especially in the number of internodes (Milutinovic, 1972), but the total length of internodes may be counteracted by recessive genes. Fasciation itself may affect the length. Since the uppermost internodes of strongly fasciated mutants are extremely shortened, one could imagine that the length of these internodes is longer in the F1 as a result of the action of alleles for normal stem growth contributed by the IL. Strongly fasciated mutants which do not cause heterosis are usually smaller than the IL and, as mentioned above, may not possess the genes for special length and lateness. Most of the linearly fasciated mutants induce weaker heterosis in many characters. 4) Many of the recombinants derived from crosses of 489 C x IL (and different mutants) show increased length and yield (3, 4). Most of them are late.

Further research will prove, modify, or disprove some of the above-mentioned points in relation to heterosis. Other determinants (heterozygosity and/or gene interactions) may be involved in an entirely satisfying explanation of this case of heterosis.

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2. Gottschalk, W. 1977. J. Nuclear Agric. Biol. 6:27-33.
3. Gottschalk, W. and G. Bandel. 1978. Z. Pflanzenzuchtg. 80:117-128.
4. Hussein, H. A. S. 1978. PNL 10:23.