FASCIATION AND HETEROSIS IN PEA (II)

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In PNL 12:44-46, 1980, results of crosses between fasciated and nonfasciated pea lines were presented and reasons given for the hypothesis that dominant genes present in our fasciated mutant 489C (and others) were involved in the heterosis phenomena in F1. Flowering and ripening were usually delayed (5-10 days) in mutants causing heterosis, the delay showing dominant inheritance. Provided that only a few not-closely-linked genes were involved, further analysis would be expected to yield the following results:

1) Homozygous recombinants derived from heterotic F1 hybrids showing the same increased number and length of internodes and number of seeds per plant as the F1.
2) Such lines when crossed with 'Dippes Gelbe Viktoria' would show dominance for length and seed number.

About seven thousand plants were investigated in relation to this problem in 1980. Both expectations were fulfilled. Several non-segregating **F3** families were found showing the same length as the heterotic F1 hybrids planted nearby, and in addition two non-segregating families showed the same number of seeds per plant as F1 489 C x IL (Dippes Gelbe Viktoria). These were crossed back with the mother variety and the results so far obtained show full dominance in number and length of internodes and probably in the number of seeds per plant (but more material is needed).

These results suggest:

a) Heterozygosity for alleles causing fasciation does not appear to be the cause of heterosis.
b) Since the genes for fasciation cause growth anomalies, they hinder the full expression of dominant mutant genes in the respective mutants.
c) Recessive epistasis and dominance are the main causes of these examples of heterosis.