

However, for the sh-wx region the inversion heterozygotes from either In2e^S or In3c proved to be heterogeneous.

For both regions the inversion heterozygotes and their normal sibs were pooled and tested for fit to a pooled $\bar{P}_{In2e^S+N2e^S}$ and $\bar{P}_{In3c+N3c}$. Among the pooled groups only two were not homogeneous, the c-sh region of In3c and the sh-wx region of In2e^S.

Table 1. Recombination and coincidence in testcrosses of inversion heterozygotes and normal sibs.

Inversion	Parental gametes	Reg. 1 singles	Reg. 2 singles	Doubles	Total	Recombination %		Coincidence
						Reg. 1	Reg. 2	
In3c	856	103	285	30	1274	8.1	22.4	1.30
Normal sibs	646	54	222	8	930	5.8	23.9	.62
In2e ^S	575	45	157	9	786	5.7	20.0	1.01
Normal sibs	1359	86	361	10	1816	4.7	19.9	.59

Table 1 shows the backcross results. Although total recombination in the two regions tested was unaffected, multiple exchanges appear to be increased in the inversion heterozygotes. In order to determine whether or not the increase in multiple exchanges was significantly different from the number of multiples in the normal sibs, the probabilities of occurrence of the observed number of double exchanges in the inversion heterozygotes were calculated from Stevens' binomial-Poisson distributions. The frequency of double exchanges in the normal sibs was used as the expected number. For both inversions the number of multiple exchanges was significantly increased. In In3c heterozygotes the probability of obtaining the observed number of double exchanges was less than .005, and in In2e^S, 0.39.

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Brown pericarp — We presently have two stocks giving brown pericarp, phenotypically similar if not identical. One stock was obtained from the Coop as Sh bp wx P-RR, and the other segregated from stocks originally obtained (also from the Coop) as P1 a A2 C R B. After growing out and testing these stocks for two years, we have concluded that the brown pericarp phenotype requires a a for its expression. We would be interested in knowing whether anyone else has information that would confirm this. Flavanones are present in fresh cob and pericarp

tissue of these plants prior to the formation of the brown colour, but it is not yet clear how this flavanone build-up is related to the formation of the brown pigment. As expected from a a tissues, there are no 3-deoxyanthocyanins or 3-deoxyleucoanthocyanidins, but there are substantial amounts of C-glycosyl-flavones present.

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The action of P1 in maize (an hypothesis) — There appear to be three main phenotypic effects of the P1 gene in our W22 stocks: 1) P1 greatly enhances the amount of anthocyanins produced in the mature leaves of plants capable of producing pigment but by itself has no ability to produce anthocyanins. 2) In stocks capable of producing anthocyanin, as a given leaf matures, pigment production commences earlier in p1 stocks than in otherwise similar P1 stocks. This has been termed a repression of pigmentation of P1 (MGNL 48:153). 3) Anthocyanin-producing P1 stocks demonstrate a sunlight-independent production of anthocyanins in leaves and pericarp.

Further examination of the P1 action in leaf tissue of the mature plant has provided information suggesting a possible biochemical function of the P1 gene. We have measured the effects of P1 on leaf sheath pigmentation in r-g B and r-r B-b stocks by weekly analysis of each leaf from plants of two replicate families. Samples were taken from 3-4 weeks pre-anthesis to 4 weeks post-anthesis. Optical density measurements were made on acidic methanol extracts from each leaf and were corrected for weight differences between families. We found that the maximum P1 effect in individual leaves (in terms of percent increase over p1) increased in magnitude up to the time at which the eleventh leaf had reached its maximum pigment-producing capability. This corresponds with anthesis on a developmental time scale. In leaves developing later than this, the percent increase due to P1 remains constant, despite an overall decrease in the maximum amount of anthocyanin present in these leaves.

We have postulated and are presently testing the following hypothesis for the biochemical action of P1. P1 may constitute a "by-pass" loop along the normal pathway of anthocyanin production. In tissues without P1 pigment is being produced at a rate determined by the steps of the normal pathway beyond the by-pass loop branch point; in this case, precursors to the branch point would not be limiting. If P1 is present, however, the alternate loop is engaged and the precursors are preferentially fed into the loop at the expense of the normal pathway. Due to the increased flow of precursors through the loop, the precursor concentrations are now limiting and the flow through the normal pathway is reduced.