The flow through the by-pass loop must also involve systems that delay the production of the anthocyanin end product. The time-lag associated with the loop may be due to a secondary cycling pathway with developmental interactions or may simply be explained by enzyme kinetics. This suggests that the initial pigment-producing phenomenon may be due not to a repression by P1 but instead to a time lag in pigment production inherent in the by-pass loop system, coupled with a decrease in function of the normal pathway. The by-pass loop must rejoin the normal pathway before the point of action of the B gene because of two observations: 1) The magnitude of the P1 effect is proportional to the amount of pigment produced in the leaf tissue. 2) No leaf pigment is produced in b P1 plants. The sunlight-independent effect of P1 may result from the by-passing of a light-requiring reaction involved in the normal pathway of anthocyanin production.

In conclusion we suggest that an alternate biochemical pathway might explain the pleiotropic effects of P1 in repressing and enhancing anthocyanin production and at the same time allow for a single biochemical action initiated by P1.

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Primary non-homologue association among somatic chromosomes — From an extension of the study and refinement of the analysis presented earlier (MGCNL 48:165-167, 1974), we report evidence for primary non-homologue association. The "affinity distance values" (ADV) for the homologues and their grouping, based on the Tukey hsd procedure, from four cold-arrested stocks are:

Chromosome	ADV ± S.E.	Group*
X	34.9 ± 2.2	A
VII	35.7 ± 0.6	
II	35.9 ± 1.5	
III	37.9 ± 0.4	B
IV	38.4 ± 0.3	
IX	39.8 ± 0.3	
VI	41.2 ± 0.2	C
V	41.5 ± 0.8	
VIII	41.9 ± 0.4	
I	43.7 ± 1.0	D

*an hsd of 1.9 is required for $p \leq 0.05$

All non-homologue comparisons were also made. Eleven of 45 means exceeded the hsd value of 3.1 required to declare a mean significantly different from the

theoretical value of 45.3. This total of 11 means exceeds the two expected (5% level) and constitutes statistical argument for the presence in maize of primary non-homologue association.

In the presence of agents which disrupt microtubules, all the homologue ADV's were increased and the number of significant means of comparisons among non-homologues was reduced from 11 to one; i.e., the distributions became random.

J.D. Horn

Effects of cycloheximide on the frequency of somatic polar metaphase observed — We have found that a short pulse of cycloheximide resulted in a marked increase in the frequency of observed polar metaphase. The sensitivity of the cell to this treatment was cell-cycle time specific. A 15-minute cycloheximide treatment (75 ug/ml) at the beginning of prophase resulted in a marked increase in polar metaphases at 45-60 minutes post-treatment at 27°C and at 135 minutes post-treatment at 18°C (Table 1). We interpreted these results as being indicative of a cell-cycle time specific event.

Table 1. Dividing nuclei (%) following cycloheximide (15'; 75 ug/ml) treatment.

Control	Stage	Minutes following treatment							
		27°C				18°C			
		0	30	60	90	0	30	90	135
60	Prophase	42	61	55	68	57	68	76	63
21	Metaphase	36	18	15	5	24	18	18	6
6	Polar metaphase	6	15	25	27	7	6	6	31
7	Anaphase	8	4	3	0	6	4	0	0
6	Telophase	8	2	2	0	8	4	0	0

Cycloheximide is an inhibitor of protein synthesis. The proteins necessary for coiling of the somatic chromosomes are presumably already synthesized by the time of the onset of prophase since normal-appearing metaphase chromosome morphology is evident in cycloheximide-induced polar metaphase nuclei. Proteins necessary to uncouple the chromosomes from the nuclear membrane, for the breakdown of the nuclear membrane and for spindle fiber synthesis are not produced after the challenge with cycloheximide.

J.D. Horn

Description of chlorophyll mutants by in vivo spectrophotometry — Virescent chlorophyll mutants have long been recognized for their potential not only as genetic tools but also as vehicles for the study of development of the photo-