This means that certain chromosome arms may be marked with only one interchange. For the list, see M.N.L. 42:122-123 (1968).

Charles R. Burnham

8. "Discussions in Cytogenetics" reprinted.

A private reprinting of my book "Discussions in Cytogenetics" is available. Copies may be obtained for $9.80 plus mailing costs. Anyone wishing to order a copy should write to my home address: 1539 Branston St., St. Paul, Minnesota, 55108; the bill will be enclosed.

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1. An epistatic phenomenon resulting in aleurone color inhibition in maize.

In reciprocal crosses between individuals having the genotypes RR and rr, respectively, the expectations are: (1) self-colored kernels when the former is used as the female parent, and (2) mottled kernels when the latter is used as the female parent. We report here an exceptional case in which an interaction between a specific modifier, Ma, and a modifier-sensitive R allele, R*, results in the absence or near absence of anthocyanin in aleurone cells having the genetic constitution rrR*.

The epistatic phenomenon resulting in the absence or near absence of anthocyanin in rrR* cells was first observed in the cross: Inbred line KYS (BzBz, cc, rr) X "Bronze" (bzbz, CC, RR). The reciprocal of this cross produced only self-colored kernels. On the other hand, when our K^L-9 knob stock (BzBz, CC, RR) was reciprocally crossed to KYS, only self-colored or mottled kernels were obtained.

In order to determine the genetic difference between "Bronze" tester and K^L-9 stock plants, the F_1 and F_2 individuals of the cross, K^L-9 X "Bronze," were used as pollen parents in crosses to KYS. The results of the tests are shown in Table 1.

Table 1

cc, rr (Female) X CC, RR (Male)		Ear Phenotype: Per cent of kernels self-colored or mottled		
		100%	50%	0%
1) KYS	"Bronze"			x
2) KYS	K^L-9	x		
3) KYS	F_1		x	
4) KYS	F_2	x (19 ears)	x (49 ears)	x (17 ears)

The 1:1 colored to "colorless" kernel ratio obtained from crosses in Entry No. 3, and the 1:2:1 ear ratio obtained from those in Entry No. 4 of Table 1, indicate that the difference between the two RR stocks is due to a single gene pair. That this difference does not involve the bz_1 locus is clearly indicated by the fact that each of the bzbz, Bzbz, and BzBz F_2 individuals when crossed to KYS produced an ear which was either 100% colored kernels, 50% colored kernels, or 0% colored kernels. If the bz_1 allele was involved in the production of "colorless" kernels, then, only the bzbz plants in the F_2 generation when crossed to KYS should have produced "colorless" kernels.

That the c_1 locus is also not involved is deduced from the following line of evidence. The C-bearing chromosome 9 in the "Bronze" tester was marked with the sh_1 allele, while the C allele in the K^L-9 plants and the c allele in KYS were associated with the Sh_1 allele. Inasmuch as the c and sh loci are tightly linked, the two C alleles are traceable through the employment of the alleles found at the sh locus. On an ear obtained from a cross in Entry No. 3, 78 colored and 72 "colorless" kernels were obtained. Approximately 50% of the colored kernels (37) were found to be ShSh in genotype; similarly, about 50% of the "colorless" kernels (37) were found to be ShSh in genotype. Half of the "colorless" kernels, then, received the C allele from the "Bronze" tester, the other half receiving the C allele from the K^L-9 stock. Thus, it is obvious that the genetic difference between "Bronze" and K^L-9 does not involve the c locus.

Beckett's 1610A-2-30 (R allele from Acc. No. 749) and 1608-1 (R allele from Tama Flint) both give results identical to those of our "Bronze" tester when crossed to KYS. On the other hand, Coe Stock No. 3, Neuffer Stock No. 1, and our Abnormal Chromosome-10 (K10) stock all give results identical to those of K^L-9 when crossed to KYS. All of these five stocks are BzBz, CC, RR in genotype. Our conclusion that the genetic difference between "Bronze" and K^L-9 involves the r locus is based on the aforementioned observations. Specifically, our data indicate that the R allele found in the "Bronze" tester is not identical to the R allele found in the K^L-9 stock.

In order to determine whether or not the inhibition of aleurone color was strictly the function of a specific R allele (R^*), several other inbreds were examined. These inbreds were: W23, N6, Wf9, Oh43, Oh51A, K55, C103, M14, Hy2, L317 (all cc, rr), and Ky27, 38-11, and our tester g r sr (all CC, rr). Only the Inbred Line L317 gave results identical to those of KYS when crossed to either the "Bronze" or the K^L-9 stocks. The unavoidable conclusion is that the inhibition of aleurone color is either (a) an epistatic phenomenon, or (b) the result of allelic interaction between a specific r allele and a specific R allele.

To test the Allelic Interaction Hypothesis, KYS X W23 hybrids were produced and used as female parents in crosses to the "Bronze" tester. The r allele contributed to the hybrid by KYS was tagged with the plant color component "r" (r^r), while that contributed by W23 was labelled with the "g" component (r^g). The R allele in the "Bronze" tester was tagged with the "g" component (R^g). From the cross KYS, r^r / W23, r^g X R^g/R^g (Bronze), 136 colored and 125 "colorless" kernels were obtained. The 1:1 colored seedling (r^r/R^g) to colorless seedling (r^g/R^g) ratio realized in both the colored kernel class (70:66) and the "colorless" kernel class (64:61) negates the hypothesis that the inhibition of aleurone color results from a specific allelic interaction. What the data clearly indicate is that the R^* allele of "Bronze" is being influenced by a genetic factor other than specific r alleles in rrR^* aleurone cells.

The data in hand permit us to rule out the involvement of two modifiers, namely, M^{st}, the modifier of R^{st} which is tightly linked to the r locus (6 units), and M_r, the mutator of R^m which shows linkage to the c

locus (14 units). If $\underline{M}^{st}$ were involved in the inhibition of aleurone color and were the modifier in KYS and L317 interacting with the $\underline{R}^*$ allele, then the "colorless" kernels obtained from the cross KYS, $\underline{r}^r$ / W23, $\underline{r}^g$ X $\underline{R}^g/\underline{R}^g$ (Bronze) should have given rise to only colored plants, save for rare recombinants. The 1:1 seedling color ratio observed in both the colored and "colorless" kernel classes is contrary to the expectation of the hypothesis which invokes the $\underline{M}^{st}$ gene. If, on the other hand, the mutator $\underline{M}_r$ were involved, then the $\underline{M}_r$-sensitive $\underline{R}$ (in this situation, the $\underline{R}^*$) allele in both of the reciprocal crosses: KYS X "Bronze" and "Bronze" X KYS, should have responded to the presence of $\underline{M}_r$. And the response should have resulted in kernels having colorless aleurone patches in an otherwise colored background aleurone. We observed, it will be recalled, inhibition of aleurone color in only one of the reciprocal crosses and the kernel phenotype to be either colorless or near-colorless, not mosaic.

We, therefore, propose the existence of an aleurone color modifier, Ma, which interacts specifically with a modifier-sensitive $\underline{R}$ allele, $\underline{R}^*$, the result being the absence or near-absence of color in aleurone cells having the genotype $\underline{rrR}^*$.

That the dosage relationship between the Ma and the $\underline{R}^*$ genes is also critical in aleurone color inhibition can be gleaned from the following observations:

a) $\underline{rrR}^*$, MaMama : Colorless or near-colorless Aleurone

b) $\underline{R}^*\underline{R}^*$r , mamaMa : Self-colored Aleurone

It would be interesting to learn what are the necessary conditions that lead to complete absence of anthocyanin in $\underline{rrR}^*$ aleurone cells containing the Ma gene. Future experimentation should lead to a more precise characterization of both the $\underline{R}^*$ and Ma genes as well as of the epistatic phenomenon involved.

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