of the model, and the need for simplicity and ease of manipulation. No suggestion is intended that allelic series are in fact present at all loci, or that such series are consistent in any attribute other than having more than two alleles. Furthermore, not every locus can be expected to be involved in epistasis, nor would every instance of epistasis necessarily involve only two loci. Certainly, too, one would expect to find few component traits conditioned by as few as two loci. The writer believes, however, that most features of the model, aside from those which exclude linkage and the possibilities of relationships among component traits, reflect genetic views favored by a majority of maize breeders. Even were this opinion incorrect, it would still appear that enough agreement was found between simulated and known yield behavior patterns to warrant further consideration of this approach, if only for illustrative purposes. Such schematic representations as may result can, in the writer's opinion, aid in a better understanding of the dynamics of yield heterosis.

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1. Restoration of A_1 gene action by crossing over.

Neuffer has undertaken an extensive study of a_1^{m-3} and a_1^{m-4}, two independent inceptions of control of A_1 gene action by the Ac system, to determine whether a controlling element, presumed to be associated with the A_1 gene in each case, could be removed by crossing over, thereby restoring A_1 gene action. His results were negative as are those that I have obtained during the course of studies of a_1^{m-3} and a_1^{m-4}. My data, however, are limited. My studies of a_1^{m-2}, on the other hand, have given quite different results. Restoration of A_1 gene action appears to arise from a crossover event which occurs relatively frequently with some states of a_1^{m-2} but infrequently, if at all, with others.

Nelson (personal communication) has shown that by means of a crossover, Wx gene action may be restored in tests conducted with wx^{m-1} and wx^{m-6}, two independent inceptions of control of action of the Wx gene by the Ac system, and also with wx^{m-8}, controlled by the Spm system. His method of analysis is precise in that it

allows placement of the component that is removed by the crossover.

Gene action at the *a1*$^{m-2}$ locus is under the control of the *Spm* system. Initially, *Spm* was associated with this locus. Later, it was possible to isolate a number of instances in which no evidence was given of the presence of *Spm* at the *a1*$^{m-2}$ locus. Action of the *A1* gene, nevertheless, remained under the control of the *Spm* system. States 7977B and 7995, Table 1, are instances of this. Many studies of *a1*$^{m-2}$ are conducted with plants that are *a1*$^{m-2}$ *Sh2*/*a1* *sh2* in constitution and many such plants have been crossed with plants that are homozygous for *a1* and *sh2*. The *a1* mutant utilized in these studies is the standard recessive that responds to *Dt* but not to *Ac* or *Spm*. The majority of the testcrosses that produced the data given in line 1 to 6 of Table 1 utilized the heterozygote as the ear parent. This table was constructed mainly to illustrate the frequency of appearance of the *A1* phenotype in the *sh2* class of kernels in some types of cross and their absence in this class in others. It should be stated that these data were obtained from crosses made in years in which no plants were present in the field that had *A1* and *sh2* in chromosome 3.

The data in line 1 of Table 1 were obtained from tests of *A1* mutants of *a1*$^{m-2}$. These mutations occurred in a chromosome carrying *a1*$^{m-2}$ and *Sh2* and in plants that had an *Spm* whose transposition-inducing component acts early in plant development. All of these *A1* mutants were stable in the presence of *Spm*. Line 2 is constructed from data obtained from tests of plants carrying a stable mottled mutant of *a1*$^{m-2}$. (This phenotype is described in Carnegie Institution of Washington Year Book No. 61, 1962.) These mutants do not produce a typical *A1* phenotype. However, in the testcrosses, 2 *sh2* kernels expressing a typical *A1* phenotype appeared. The data in line 3 came from testcrosses of plants that had *Spm* associated with the *A1* locus but the transposition-inducing component of this *Spm* acts late in plant and kernel development and, in this regard, it is very stable. (Kernels with this *Spm* are illustrated in B, Plate I of my report appearing in the Carnegie Institution Year Book No. 63, 1964.) It does not allow any germinal mutations to occur at *a1*$^{m-2}$ nor at *a1*$^{m-1}$ or *wx*$^{m-8}$ which have been tested for this. Nevertheless, 5 *sh2* kernels with very clearly expressed *A1* phenotypes appeared on the ears that contributed the data in line 3 in the table. None appeared in the *Sh2* class. In contrast to this, no kernels with this phenotype appeared in tests of plants having an inactive *Spm* associated with the *a1*$^{m-2}$ locus, either in the *Sh2* or *sh2* class.

Table 1

Phenotypes of Kernels on Ears Produced by Crosses of Plants That Were Homozygous for *a1* and *sh2* with Plants That Had *a1 sh2* in One Chromosome 3 and *Sh2* and the Markers Given in Column 1 in the Homologue

Constitution of *Sh2* chromosome	Phenotypes of Kernels					
	Sh2			*sh2*		
	A1		Colorless	*A1*		Colorless
1. *A1*; mutant of *a1*$^{m-2}$	8,959		13	12		8,906
2. Mottled: mutant of *a1*$^{m-2}$	Mottled 10,498	0	8	Mottled 15	2	10,399
3. *Spm*^{W} *a1*$^{m-2}$	*Spm*^{W} *a1*$^{m-2}$ 12,138	0	7	*Spm*^{W} *a1*$^{m-2}$ 10	5	12,131
4. Inactive *Spm* *a1*$^{m-2}$	(see text)				0	10,843*
5. State 7977B *a1*$^{m-2}$	" "				5	16,033*
6. State 7995 *a1*$^{m-2}$	" "				5	8,780*
7. *Spm*^{W} *a1*$^{m-5}$ (♀ in cross)	*Spm*^{W} *a1*$^{m-5}$ 3,757	0	3	*Spm*^{W} *a1*$^{m-5}$ 6	0	3,626
8. " " (♂ in cross)	4,370	2	5	0	0	3,930

* A few of these kernels received a crossover chromatid with *a1*$^{m-2}$ but its presence in most such kernels cannot be detected visually.

The phenotypes of the $\underline{Sh_2}$ class in this cross, line 4, and in those in lines 5 and 6, have been omitted from the table because there are a number of different types and these would be difficult to arrange in this table. None of these, however, is $\underline{A_1}$ in phenotype. States 7977B and 7995, lines 5 and 6, also produced some $\underline{A_1}$ $\underline{sh_2}$ kernels on the testcross ears. An active Spm was not present in the heterozygous parents. In some crosses, it was introduced into many kernels by the $\underline{a_1}$ $\underline{sh_2}$ pollen parent that also was homozygous for wx. Some of the plants in lines 5 and 6 had $\underline{wx^{m-8}}$ in one chromosome 9. Three of the 10 $\underline{A_1}$ $\underline{sh_2}$ kernels in lines 5 and 6 received $\underline{wx^{m-8}}$ from the ear parent and Spm from the pollen parent. The $\underline{A_1}$ expression in these three kernels was completely stable but that of the wx gene was not. $\underline{wx^{m-8}}$ responded to the introduced active Spm by producing a number of endosperm sectors exhibiting various levels of Wx gene action.

Lines 7 and 8 of Table 1 are included to illustrate that no $\underline{A_1}$ $\underline{sh_2}$ kernels appeared in testcrosses conducted with a state of $\underline{a_1^{m-5}}$ having an $\underline{Spm^w}$ associated with it. This $\underline{Spm^w}$ undergoes frequent mutation to a state that allows early occurring transposition and thus early occurring mutations to high alleles of $\underline{A_1}$.

Whether or not a controlling element may be removed from a locus by crossing over may well depend on the "state of the locus", as suggested by the data in Table 1, and also upon the organization of components in the comparable region of the homologue.

Two other studies aimed at removing a controlling element from the vicinity of the genes it can affect are reported below.

B. McClintock

2. Attempts to separate Ds from neighboring gene loci.

Early in the study of transposition of Ds to various locations within the short arm of chromosome 9, two instances of its insertion just distal to $\underline{Sh_1}$ were found, the first instance in 1948 and the second instance in 1949. In both instances, Ds remained in this location thereafter. Although it did not transpose away from this location, it responded to Ac by producing dicentric chromatids and also a series of changes affecting the genes located to either side of it. The types of change were described in the Carnegie Institution of