

With regard to stability of location of Spm, the results obtained from the two experiments, outlined above, differ markedly. The first gave evidence of relatively frequent changes in location of Spm. This is in contrast to the experiment just described where an unusual degree of stability of location of Spm was made evident. Nothing is yet known about genetic or other factors that may be responsible for controlling the time during development of a tissue when change in location of Spm will occur, or the frequency of this.

2. Continued study of a structurally modified chromosome 9.

In last year's News Letter, a description was given of a modification affecting the organization of chromosome 9. Two chromosomes instead of one carry the substance of this chromosome. One of these is composed of the distal third of the short arm and it was referred to as the fragment chromosome. The centromere is situated at the proximal end of this component of chromosome 9. The longer segment is composed of the proximal two-thirds of the short arm of chromosome 9 and all of its long arm, and it was referred to as the deficient chromosome. Interest in this case was centered on the aberrant behavior of the fragment chromosome in somatic cells, and this was outlined briefly last year. Further examination of this case required more exact knowledge of the composition of the two components of this structural modification. Therefore, an extensive series of tests of this were continued during the past year. The fragment was known to carry the locus of C and preliminary evidence presented in the News Letter last year, suggested that it also carried the loci of sh and bz. Since the deficient chromosome was known to have the loci of Sh and Bz, with Sh situated very close to the end of its short arm, the genetic composition of the structurally modified chromosome 9 would then include a duplication of a segment composed of the region from the locus of sh to one that is proximal to bz. Recent tests have confirmed the presence of sh and bz in the original fragment chromosome and they also have revealed the relative length of the segment that extends from bz to the centromere of the fragment. It is equivalent to a segment in the normal chromosome 9 that is 5 crossover units proximal to Bz.

Genetic study of the constitution of the fragment and the deficient chromosome makes it clear that a segment in the fragment,--from the locus of sh to the centromere--, duplicates a segment in the deficient chromosome that is located at the very end of its short arm. Examination of the chromosomes at the pachytene stage in structural heterozygotes did not reveal the physical length of the duplicated segment with the desired degree of certainty. It can not include more than 1 or 2 small chromomeres, if matching chromomeres in synapsed regions may be used as a reliable criterion of homology.

In structural heterozygotes whose chromosome 9 components are appropriately marked for crossover studies (an example: normal chromosome 9 with I Sh Bz wx/ deficient chromosome 9 with Sh Bz Wx/ fragment

with C sh bz) an exchange occurring in the region between sh and bz of the fragment (region 1) or between bz and the centromere of the fragment (region 2) would give rise to a structurally normal chromosome 9 carrying sh and Bz or one carrying sh and bz. The presence of such a normal chromosome 9 was confirmed in 48 plants that were derived from kernels exhibiting a phenotype expected from a crossover in one or the other of these regions. In 9 of these 48 plants, an unmodified fragment chromosome carrying sh and bz was also present. (The fragment could not carry the reciprocal product of the crossover. If it had, the kernel would not have exhibited the crossover phenotype.) A normal chromosome 9 would be obtained from a crossover in region 1 or 2 either between the fragment and the normal chromosome or between the fragment and the deficient chromosome. Evidence obtained from the test crosses did not allow definite conclusions to be drawn regarding the relative frequency of the exchanges that occur in these regions between the fragment and the normal and deficient chromosomes. It did suggest, however, that most of the crossing over may take place between the fragment and the normal chromosome and that a crossover in either region 1 or 2 does not interfere with another occurring between the normal and the deficient chromosome. Evidence for the latter statement is conflicting, however, and some of the difficulties encountered in these analyses may derive from differences in behavior of the fragment among the tested plants, as illustrated below.

Four plants having two deficient chromosomes 9, each carrying Sh, Bz, and Wx, and a single fragment chromosome carrying C, sh, and bz, were used as pollen parents in crosses to plants that were homozygous either for C, sh, bz, and wx, or c, sh, bz, and wx, or for c, sh, Bz, and wx. The only functional pollen grains produced by such plants are those having either a deficient chromosome and the fragment, a deficient chromosome and the fragment that has become attached to the end of another chromosome (which sometimes occurs), or a structurally normal chromosome 9 produced by a crossover, however it may be initiated, between the homologous segments of the fragment and the deficient chromosome. Crossovers of this latter type would give rise to structurally normal chromosomes having either C sh Bz Wx or C sh bz Wx. The number of kernels having such phenotypes that appeared on the ears produced by test crosses with these four plants is given in A of table 4. In the cross entered in B of this table, only the sh kernels could be recorded for all of them received Bz from the female parent. Pollen used in the test crosses was collected from each plant over many days, and from tillers as well as from the main stalk. Regardless of the date or the part of the plant from which the pollen was collected, the frequency of appearance of the sh class of kernels on the test cross ears was the same for an individual plant. However, as table 4 shows, wide differences in this respect are exhibited among these plants. Such differences would not be anticipated unless it was known or suspected that some genetic system was controlling the type of behavior of the fragment chromosome. There is some evidence to suggest that this system may be related to the one that controls the behavior of the

fragment in somatic cells. In somatic cells, the fragment may undergo types of events that effect its non-disjunction or its removal during a mitotic cycle from one or both sister cells. Differences in type of genetic control exist and these may be recognized readily, for they give rise to different patterns of variegation in plant and endosperm cells when proper genetic markers are present to allow detection of those events that affect fragment distributions. The behavior of the fragment in the two plants in table 4 that produced the lowest percent of sh kernels, i. e., plants 6971A and 7174A-2, was similar in endosperm development. The pattern of variegation each produced indicated a low rate of loss of the fragment and these losses occurred late in endosperm development. On the other hand, the behavior of the fragment in plants 7169-10 and 7176B-3 resulted in a pattern of variegation in the endosperm that indicated frequent loss of the fragment and often this occurred early in development.

Table 4.

Plant No.	A				B		
	Total No. kernels	Phenotype of <u>sh</u> kernels		% <u>sh</u>	Total No. kernels	C <u>sh</u> Bz Wx	
		C <u>sh</u> Bz Wx	C <u>sh</u> bz Wx			% <u>sh</u>	
6971A	371	1	2	0.8	742	4	0.5
7167-10	3294	9	44	1.6	1589	23	1.4
7174A-2	3750	5	14	0.5	1361	5	0.3
7176B-3	1638	7	51	3.5	1763	63	3.5
	Totals	22	111				

That the kernels showing the crossover phenotypes received a structurally and functionally normal chromosome 9 from the male parent was demonstrated by cytological and genetical studies conducted with 2 plants derived from the C sh Bz Wx class of kernels and with 8 plants

derived from those in the C sh bz Wx class. Among the latter, two plants had received an unmodified fragment chromosome in addition to the structurally normal chromosome 9. It is of interest to note that the ratio of Bz to bz among the sh class of kernels in A of table 4 (22 : 111) is much the same as the ratio of these two phenotypes among the sh class that was obtained from heterozygotes (normal chromosome 9 with I Sh Bz wx/deficient chromosome 9 with Sh Bz Wx/fragment with C sh bz) when these were used as pollen parents in crosses to plants that were homozygous either for C, sh, bz, and wx, or for c, sh, bz, and wx. This ratio was 57 C sh Bz (6 Wx : 51 wx) to 206 C sh bz (27 Wx : 179 wx).

Barbara McClintock

CENTRO DI GENETICA DEL C. N. R.

Pavia, Italy

and

ISTITUTO DI GENETICA VEGETALE DELLA FACOLTA' DI AGRARIA

Piacenza, Italy

1. Defective endosperm factors from maize-teosinte derivatives.

Evidence is being accumulated that most of the defective endosperm factors from maize-teosinte derivatives are highly unstable. In several cases all sizes of kernels can be obtained from selfed de^t/de^t plant. In a few other types of de^t factors three distinct "states" seem easily distinguishable; besides the normal, a weak and an extreme defective class appear on the defective-segregating ear. At least a few de^t factors, when placed in a genetic background other than A158, seem to "recover." Apparently some genotypes "restore" de^t factors to De^t. Several de^t factors, which arose in different derivatives, turned out to be allelic, which, together with the instability, seems to support the hypothesis that the cause of such de^t factors could be of extragenic nature (in McClintock's sense). The factors de^t4, de^t5, de^t10, de^t11, de^t17, de^t18, de^t19, de^t23, de^t24 are probably identical or allelic; the same is possibly true for the series de^t13, de^t22, de^t26, de^t27, de^t29; and is well established for the series de^t14 and de^t20 (on chromosome 4).

2. Endosperm chimeras on ears segregating de^t factors.

Endosperm chimeras have been observed in derivatives of crosses to testers of the stocks showing the de^t factors. Their rate of appearance, when no teosinte segments are present, is unknown. The chimeras can be observed for characters whose genetic factors are carried by any chromosome, including the de^t carrier. Out of 17 chimeric kernels (12 Su-su, 3 De^t-de^t, 1 Pr-pr, 1 Wx-wx) 8 were found in ears segregating genetic.