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Cytogenic Behavior of a Knobbed Chromosome 10 in Maize

Abstract. Unlike maize plants heterozygous for the abnormal chromosome 10 (K10) and the normal chromosome 10 (k10), those heterozygous for a knobbed chromosome 10 (K_T10/k10) do not undergo preferential segregation or "meiotic drive." Ting's chromosome 10, however, does show neocentric activity, although the extent of this is not as pronounced as is the case in K10 homozygotes. The K_T10 chromosome is considerably shorter than and is cytologically distinguishable from the K10 and the k10 chromosomes.

In the *A* genome of maize there exist several morphologically distinct forms of chromosome 10. The knobless form (k10) is the most frequently encountered one and is the shortest of the *A* chromosomes. The rare forms have been designated as "abnormal" and are longer than and uniquely different from the "normal" chromosome 10. The origin of these abnormal chromosomes 10 is still to be established, although Ting has proposed a possible mechanism (*1*).

The abnormal chromosome 10 designated as K10 is characterized by the presence of three small but conspicuous chromomeres in the region corresponding to the distal one-sixth segment of the long arm of the normal chromosome 10 and by the presence of a foreign segment of chromatin possessing a very large heterochromatic knob in the subterminal position. Of the several forms of abnormal chromosomes 10 present in the maize genome, this K10 form is the most common (*2*). In the presence of this abnormal chromosome, several intriguing events occur: (i) the formation of neocentromeres in regions other than the true centromeres (*3, 4*); (ii) the precocious anaphase movement of chromosomes possessing such newly arisen centromeres during meiosis with the neocentric regions leading the chromosomes to the poles (*3*); (iii) the preferential segregation or nonrandom recovery of certain chromosomes in the egg cells during megasporogenesis (*4, 5*); and (iv) an increase in the frequency of chiasma formation (*6*) and crossing over (*7, 8*) in the chromosomes thus far studied. The neocentric activity is especially pronounced in plants homozygous for the K10 chromosome, and it is not uncommon to observe highly attenuated chromosomes at meiotic anaphases.

A second form of abnormal chromosome 10, here designated as Ting's abnormal choromosome 10 (K_T10), is readily distinguishable from the K10 form. The K_T10 chromosome is longer than the normal chromosome 10 by approximately one-fifth the length of the latter. In the subterminal position of this abnormal chromosome is located a large knob, and in the region corresponding to the distal one-sixth of the long arm of the normal chromosome 10 is found a single chromomere which is very often quite conspicuous. Ting found this particular form of chromosome 10 in one of three plants from a cross between a Peruvian variety and a U.S. inbred strain. The plant possessing the K_T10 chromosome carried no supernumerary or *B*-chromosome, while each of the other two sister plants was homozygous for the normal chromosome 10 and possessed a single *B*-chromosome. According to Ting, the foreign piece of the K_T10 chromosome closely resembles the proximal region of the *B*-chromosome which includes the terminal centromere and the large, elongate, pycnotic region (*1*). He also reported the existence of a "high degree of 'homology'" between the knob of the K_T10 chromosome and the pycnotic regions of the *B*-chromosome (*9*). It is of interest to note that Rhoades and Dempsey found no appreciable frequency of association between the knob of the K10 chromosome and the *B*-chromosome (*10*). Because of the frequent association of the pycnotic regions of the K_T10 chromosome and the *B*-chromosome and the apparent morphological similarity between the foreign segment of the K_T10 chromosome and the proximal region of the *B*-chromosome, Ting has suggested that the K_T10 chromosome arose as a consequence of a simple translocation involving the normal chromosome 10 and the *B*-chromosome (*1*). As is the case with the K10 chromosome, neocentric activity was observed during the two meiotic divisions of microsporogenesis in plants carrying the K_T10 chromosome (*1*).

During the course of the present study it was found that Ting's abnormal chromosome 10 is considerably shorter than the K10 chromosome, the end of the long arm approximately coinciding with the position occupied by the most distal of the three conspicuous chromomeres of the K10 chromosome. Furthermore, the knob present on K_T10 is only about a third as large as that found on the K10 chromosome. Insofar as neocentric activity is concerned, plants homozygous for the K_T10 chromosome display activity not unlike that found in K10/k10 heterozygotes.

Inasmuch as both forms of abnormal chromosome 10 possess neocentric activity and since the preferential segregation hypothesis (*3, 4*) involves the action of these neocentromeres, it was deemed of interest to ascertain whether Ting's abnormal chromosome 10 is also capable of inducing preferential segregation during megasporogenesis. The data presented below represent the results of the initial experiment.

In this experiment the normal or k10 chromosome was genetically marked with the dominant *R* (aleurone color) factor, whereas the abnormal partner, K_T10, was marked with the recessive *r* (no aleurone color) allele. At the same time, one of the chromosomes 9 which

Table 1. Results from backcrosses of *R* k10 / *r* K_T10; $K^{M}9$ *Sh* / $K^{S}9$ *sh* compounds.

Family	Kernels (No.)						$\chi^2(1:1)$	*P*
	R	*r*	Total	*Sh*	*sh*	Total		
61 : 35	1366	1332	2698				0.428	> .50
61 : 36	1388	1380	2178				0.649	> .30
Totals	2704	2712	5416*				0.012	> .90
61 : 35				975	956	1931	0.042	> .80
61 : 36				1053	1049	2102	0.0076	> .90
Totals				2028	2005	4033*	0.132	> .70

* The two populations are not of identical size because a few of the plants were pollinated by *r* testers homozygous for the *Sh* factor.

possessed a medium-sized knob (K^M) was marked with the dominant *Sh* (plump kernel) factor, and the homolog possessing a small-sized knob (K^S) was genetically marked with the recessive *sh* (shrunken kernel) allele. Plants with the above cytogenetic constitution were employed as female plants in backcrosses to plants homozygous for both the *r* and *sh* alleles. It should be stated that preferential segregation is not confined to chromosome 10. Preferential segregation of other chromosomes occurs in K10/k10 heterozygotes when the two members of a pair differ by one possessing a knob while the other is knobless or if the two homologs have knobs of dissimilar size. If the K_T10 chromosome were as efficient as the K10 form in inducing preferential segregation, one would expect to find approximately 70 percent of the backcross kernels to be colorless (*3, 4, 7*) and about 60 percent to be "plump" in phenotype (*7*). The data presented in Table 1 clearly demonstrate the inability of the K_T10 chromosome to effect preferential segregation. Thus one would not expect to observe "meiotic drive" (*11*) in a population in which only the normal and the K_T10 chromosomes were segregating. Rather, these two forms of chromosome 10 should exist in stable proportions. On the other hand, a population containing equal numbers of the K10 and K_T10 forms should in time convert into a population in which the K10 chromosome is more prevalent.

Whether the absence of preferential segregation is related to the low level of neocentric activity is not known. Emmerling has noted that two modified forms of the K10 chromosome elicit a low level of neocentric activity and has suggested that this reduced neocentric activity could be responsible for the random segregation ratios she has observed in her experiments involving these modified chromosomes (*12*). A similar situation may very well exist here also (*13*).

GARY Y. KIKUDOME
Department of Field Crops, University of Missouri, Columbia

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Metabolic Block in Utilization of Galactose by Strain L Tissue Culture Cells

Abstract. Evidence is presented which suggests that the utilization of galactose by mouse strain L-cells is blocked by the absence of epimerase activity in this strain.

Abnormalities in the metabolism of galactose occur in a variety of organisms. The precise nature of these abnormalities has been studied in humans and, more recently, in bacteria. Thus Schwarz *et al.* (*1*) showed that when erythrocytes of carriers of the heritable defect galactosemia were incubated with galactose, galactose-1-phosphate (Gal-1-P) accumulated in the erythrocytes. Kalckar and his collaborators (*2*) demonstrated that this accumulation of Gal-1-P was caused by the lack of Gal-1-P uridyl transferase (transferase) activity in galactosemics. Different mutants of the bacterium *Escherichia coli* unable to utilize galactose as a source of carbon have been shown to be deficient in galactokinase (*3*), transferase (*3*), and uridine diphosphogalactose-4-epimerase (epimerase) (*4*) activity, respectively. Triply defective mutants have also been found (*4*).

Eagle *et al.* (*5*) have reported that galactose does not support the growth of L-cells, a strain of mouse fibroblasts (*6*). We confirmed this observation and also found that L-cells do not respire galactose. In this report it is shown that the inability of L-cells to utilize galactose as a source of carbon may be explained by the absence of epimerase activity in these cells.

L-cells were grown as described earlier (*7*); lactalbumin hydrolyzate medium (*8*) was substituted for tissue culture medium 199. The determination of the presence of the enzymes required for the metabolism of galactose was based in part on the identification of intracellular galactose and in part on enzyme assays. L-cells which had been incubated for 180 minutes at 37°C in lactalbumin hydrolyzate medium containing 100 μmole 1-C^{14} labeled galactose per milliliter were harvested by sedimentation, washed, and boiled in a small amount of water; and the cellular debris was removed by centrifugation. The supernatant was then freed of protein by ultrafiltration through a dialysis membrane and chromatographed with the use of Leloir's ethanol-acetate solvent (*9*). A portion of this protein-free extract was retained for the enzymic determination of Gal-1-P which had been formed (see below). The ethanol-acetate solvent provided excellent separation of the three compounds, galactose, Gal-1-P, and uridine diphosphogalactose (UDPGal). Thus, the R_F value of galactose is about 0.65, Gal-1-P has a R_F value of about 0.32, and uridine diphosphoglucose (UDPG) of 0.18. Since UDPG and UDPGal have the same R_F value in this solvent (*9*), UDPG was used as the reference standard for UDPGal.

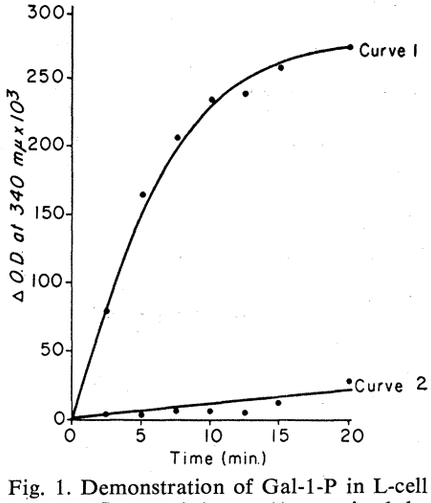

Fig. 1. Demonstration of Gal-1-P in L-cell extracts. Cuvette 1 (curve 1) contained the following assay mixture: 0.5 ml of buffer (0.006*M* histidine-HCl, *p*H 7.5); 1 μmole of $MgSO_4$; 0.6 μmole of TPN; 0.5 μmole of UDPG; yeast enzyme preparation, 0.05 ml; 0.2 ml of ultrafiltrate (see text) prepared from L-cells incubated in the presence of 0.005*M* galactose. Cuvette 2 (curve 2) contained 0.2 ml of boiled extract prepared from L-cells incubated in the absence of galactose. Cuvette 3 (control for endogenous reduction of TPN by yeast extract) contained 0.4 μmole of added Gal-1-P and no L-cell extract, and cuvette 4, which contained the yeast enzyme preparation (0.05 ml) and TPN, served as blank. The total volume in all cuvettes was 1 ml. The course of the reaction was followed by measuring TPN reduction at 340 mμ.