hence this gene probably is on chromosome 3. This
gene is not uncovered by TB-3a (3L.1); thus it is
in the short arm or proximal 10% of the long arm.

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1. A mutant of chromosomal behavior in mitosis and meiosis.

The isolation of a B^9 isochromosome from the TB-9b translocation
has been described (Carlson, 1970) as well as the isolation of mis-
division products from the B^9 isochromosome (Carlson, 1973). Following
conversion of the B^9 chromosome to an isochromosome, with apparent loss
of a minute short arm, the ability of the chromosome to undergo nondis-
junction at the second pollen mitosis was not lost and possibly not even
impaired. However, among six telocentric derivatives of the B^9 iso-
chromosome, four were found to be virtually incapable of nondisjunction.
The other two derivatives carried out nondisjunction at the second
pollen mitosis at a high rate. Differences between misdivision products
of the isochromosome may be related to the extent of damage done to the
centromere during misdivision. In any case, only the chromosomes which
do not undergo nondisjunction will be discussed. These B^9 chromosomes
will be referred to as mutants of nondisjunction. Experiments were
carried out to analyze what component of nondisjunction is missing from
the mutant chromosomes. In one experiment, the possibility that B chromo-
somes could restore nondisjunction to the mutant chromosomes was tested.
One of the derivative chromosomes (mul-1) was combined with two iso-
genic Black Mexican stocks which vary only in the presence or absence of
B chromosomes. Plants of $9\frac{Bz}{}9^B$ (mul-1) $B^{9\frac{Bz}{}}$ constitution, with and with-
out B chromosomes, were crossed as male parents onto a bz tester. If B
chromosomes restore nondisjunction to the mutant chromosome, generating
bronze kernels in the testcross, it may be assumed that a gene(s) which

acts in a _trans_ manner is missing from the mutant chromosome. However, as seen in Table 1, B chromosomes do not restore the normal non-mutant condition in which the frequency of _bz_ kernels ranges from about 20% to 30%. In the four plants lacking B chromosomes the rate of nondisjunction is quite low and, while it rises somewhat in the presence of B's, it does not attain anywhere near normal levels. The percent of kernels with fractional or mosaic (_Bz/bz_) endosperm phenotypes is also given in Table 1. In all crosses the unstable kernel types are more frequent than the _bz_ cases of nondisjunction, further demonstrating the inability of B chromosomes to restore "normal" behavior to the mutant B^9. In fact, the B chromosomes appear to destabilize the mutant chromosome, and one may wonder whether the apparent increase in nondisjunction in the presence of B's may not also result from a destabilization of the chromosome rather than true nondisjunction. At the present time, fractional (single event) and mosaic (multiple event) kernels have not been separated nor has the process of accepting or rejecting small fractional events been standardized. Thus, these data should be taken with some caution. However, the rise in _Bz/bz_ kernels with the addition of B chromosomes is unmistakable.

One interpretation of these findings is as follows. The centromere has lost a function which is vital to nondisjunction. The addition of B chromosomes destabilizes the mutant chromosome at the second pollen mitosis, but does not generally induce nondisjunction. Let us assume, as suggested by the results of Rhoades, Dempsey and Ghidoni (1967), that heterochromatic regions adjacent to the B centromere become "sticky" at the second pollen mitosis and initiate nondisjunction. This process may occur in the mutant B^9, but nondisjunction fails because the centromere is incapable of unipolar orientation. The behavior of the mutant chromosome may be likened to that of a univalent chromosome in meiosis: it is undivided but not capable of orienting properly to one pole. The mutant B^9 undergoes a possibly delayed splitting and disjunction to opposite poles. The addition of extra B chromosomes contributes a gene activity which increases the stickiness of the centromeric heterochromatin, but does not allow for unipolar migration. The competition between nondisjunction and disjunction destabilizes the chromosome.

Table 1

Comparison of nondisjunction in the presence and absence of added B chromosomes for the centromeric mutant of nondisjunction, mul-1

Plant numbers are given for male parents which were $9\frac{Bz}{}9^B$ (mul-1) $B9\frac{Bz}{}$. Female parent was a bronze tester. Since the appropriate $9^B B9$ gamete is not always transmitted through the pollen, the data should be multiplied by two (approximately) to obtain actual frequencies.

Plants without B's

Male parent	Percent bz	Percent Bz/bz	Total seeds
2071 A	0.2% (3)	1.9% (29)	1499
2071 B	0.5% (7)	1.2% (17)	1422
2071 C	0.2% (4)	1.6% (25)	1590
2071 D	0.2% (2)	2.0% (26)	1315

Plants with B's

Male parent	Number of B's	Percent bz	Percent Bz/bz	Total seeds
2073 - 4	3	0.2% (3)	2.2% (33)	1467
2073 - 7	4-5	1.9% (22)	4.4% (51)	1152
2074 - 7	7	2.4% (50)	8.8% (181)	2071
2073 - 6	7-8	3.5% (47)	6.7% (97)	1435

Table 2

Testcross data of $9\frac{bzwx}{}9^B\frac{Wx}{}$ mutant $B9\frac{Bz}{}$ ♀ x $bzwx$ ♂

Female parent	Bz Wx	Bz wx	bz wx	bz Wx	Ratio $9^B B9 / 9B9$
mul-1	388	306	905	54	1.58/1.00
1849-13	337	204	761	58	2.24/1.00

The analogy to a meiotic chromosome leads to the question of whether genes that control meiosis also control mitotic nondisjunction. The answer may be yes, at least for the centromeric mutants of nondisjunction. The meiotic behavior of two misdivision products of the B^9 isochromosome (mul-1 and 1849-13) were followed by crossing $9^{\underline{bzwx}}9B^{\underline{Wx}}$ (mul-1 or 1849-13) $B9^{\underline{Bz}}$ plants as female parents to a $\underline{bz}\ \underline{wx}$ tester. Formation of the major gametic classes ($9^{\underline{bzwx}}$; $9^{\underline{bzwx}}B9^{\underline{Bz}}$; $9B^{\underline{Wx}}B9^{\underline{Bz}}$) can be determined genetically (Robertson, 1967). The raw data are given in Table 2. The calculated frequencies of the relevant classes show that the 9^BB^9 gamete exceeds the $9B^9$ class considerably. These two gametic classes were found in equal frequencies by Robertson (1967) and the author when normal B^9 chromosomes were present.

The above data suggest that a change in mitotic orientational behavior at the second pollen mitosis is accompanied by a change in meiotic orientation. The meiotic behavior of normal B^9's, as described by Robertson, is considered anomalous. Equivalence of the 9^BB^9 and $9B^9$ gametes means that in the 99^BB^9 trivalent the B^9 chromosome disjoins at random from its pairing partner, chromosome 9. Rhoades (1940) had earlier found that disjunction of a telocentric chromosome 5 from its partner in 5 5 telo 5 trivalents was regular. The mutant B^9's, discussed here appear to be reverting toward "normal" chromosome behavior. The finding can be rationalized if chromosomes normally require that tension be applied to the centromere for orientational stability (Nicklas and Koch, 1969). During the second pollen mitosis a chromosome undergoing nondisjunction is essentially a univalent and tension cannot develop on the centromere. The result is instability. Perhaps the B chromosome possesses a specialized centromere which does not depend on tension for orientational stability. Nondisjunction can then occur regularly, but during meiosis segregation from a trivalent is abnormal. The mutant B^9's, derived by misdivision of the isochromosome, may have modified centromeres which respond to tension during orientation. They are incapable of unipolar orientation at the second pollen mitosis and they tend to disjoin from chromosome 9 during meiosis. Although the mutant B^9 does not always disjoin from its pairing partner in a trivalent, as

the telocentric 5 does, Maguire has found (1970) trivalent situations not involving B chromosomes in which disjunction is also not completely regular. In Rhoades' work with the telocentric 5, proximal exchanges between 5 and telo 5 were studied. This may have added another inducement to disjunction which is unrelated to tension. Nicklas discusses the tendency for centromeres to face in opposite directions (1967), and this tendency may be more prevalent in closely associated centromeres.

The findings given here are tentative but two conclusions may result. First, nondisjunction consists of at least two events: stickiness of the centromeric heterochromatin followed by unipolar orientation and migration. Second, genes involved in meiotic and mitotic chromosome segregation may in some cases be identical.

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