

4. Linkage data for five pigment deficient mutants.

Over the years I have accumulated linkage data on the following mutants:

w*-5625 - This is an off-white albino mutant that is on the long arm of chromosome 1, 11.3 (n = 159) crossover units from the breakpoint of T1-9a (1S.13). Placement in the long arm was indicated by the segregation of the mutant in crosses with TB-1a (1L.2).

w*-PI168013 - This is an off-white albino mutant isolated from a Plant Introduction accession from Turkey. Since this mutant shows 0.6% (n = 171) recombination with T1-9c (1S.48) and is uncovered by TB-1b (1S.05), it is located in the short arm of chromosome 1.

17 - This is a seedling mutant that varies from yellow-green to pale green in color. It is uncovered by TB-9b (9S.4), thus indicating it is on the short arm of chromosome 9. Linkage tests with wx and c indicate 21.6% (n = 282) recombination with the former and 16.3% (n = 270) recombination with the latter gene. Since the c - wx distance is approximately 33 crossover units, 17 must be located between c and wx.

nec*-PI1217486 - This is a yellow necrotic mutant that was isolated in a Plant Introduction accession of Dakota Flint. Allele tests have shown this mutant to be identical to sienna-7748, nec*-6697, and 1*-Blandy2. The following linkage data with three translocations involving chromosome 8 indicate it is located on the long arm of this chromosome: T8-9d (8L.09) - 14.0% c.o. (n = 358); T6-8 (6873) (8L.29) - 4.3% c.o. (n = 299); and T6-8 (6187) (8L.51) - 2.5% c.o. (n = 321).

w*-PI1228183 - This is an off-white albino that was isolated from a Plant Introduction accession from Russia. Linkage tests with T3-9c (3L.09) indicate 34.7% crossing over (n = 251). No linkage is indicated with waxy,

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 nondisjunction at the second pollen mitosis was not lost and possibly not even impaired. However, among six telocentric derivatives of the B^9 isochromosome, 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 chromosomes could restore nondisjunction to the mutant chromosomes was tested. One of the derivative chromosomes (mul-1) was combined with two isogenic Black Mexican stocks which vary only in the presence or absence of B chromosomes. Plants of $9^{\underline{Bz}}B$ (mul-1) B^9 ^{Bz} constitution, with and without 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