

Twisted tendrils (*Twt*) - a phenotype associated with a translocation involving chromosome 1

Gorel, F.L., Temnykh, S.V.,
Lebedeva, I.P., and Berdnikov, V.A.

Institute of Cytology and Genetics
Novosibirsk 630090, Russia

A new mutant displaying altered tendril morphology was found in 1988 after treatment of Sprint-2 seeds with 0.015% EMS. The tendrils of the mutant coil strongly soon after they form, even in the absence of contact with other objects (Fig. 1). Three of four plants in one M_2 family expressed the mutant phenotype, and one of these was used as the pollen parent in crosses with marker line WL1238 and with the initial line Sprint-2. Approximately half of the progeny in both cases had abnormal twisted tendrils and were semisterile; the other half were normal. We initially hypothesised that the pollen parent was heterozygous for a dominant gene that affects tendril morphology and fertility. The symbol *Twt* (Twisted tendrils) is proposed.

An F_2 population from the cross WL1238 (*A, twt, i, s, wb, k, b, le, gp, cp, tl, Ust, Bra, fna*) x Sprint-2 (*a, Twt*) was analysed, and a recombination fraction of about 30% was found between genes *Twt* and *a* (data not shown). For more accurate localisation of *Twt* a three-point test cross, involving *a, Twt*, and *His(2-6)*, was analysed. *His(2-6)* is a cluster of closely linked genes coding a set of four molecular variants of histone H1 (H1 haplotype) and has previously been shown to be 4 cM from *a* (1). The histone H1 phenotype is described by a numerical formula of four digits that reflects the electrophoretic mobility of histone H1 variants numbers 3, 4, 5, and 6 (Fig. 2).

A maternal plant taken from VIR accession K-3953 (Tadjikistan) with genotype H1-1133/H1-1133, *A/A, twt/twt* was crossed with a plant having genotype H1-1021/H1-1021, *a/a, Twt/twt* produced from the backcross mentioned above. Eight of the 15 F_1 plants had phenotype *Twt*. The remaining seven plants were normal. This result is consistent with the dominance of the mutant trait. All 8 F_1 plants having supercoiled tendrils were crossed reciprocally with tester line 5-11 (H1-1123/H1-1123 *a/a, twt/twt*). Progeny analysis of the testcross (Tables 1 and 2) permitted us to localise the genetic factor responsible for phenotype *Twt* at 8 cM from *a* and 13 cM from *His(2-6)*. These data suggest the following order of the three genes: *His(2-6)* -(5.3 cM) - *a* - (8.0 cM) - *Twt*.

However, it should be noted that situation is complicated by the presence among the testcross progeny of 28 plants that apparently had all three sets of the histone H1 haplotype (see Fig. 2, lane 4). All of them had phenotype *A, Twt*, H1-1133/1021/1123, and they could be distinguished from the rest by smaller size, much slower development, and stipules with pointed apices and undulating margins. These observations, particularly the presence of all three H1 haplotypes, suggested that these exceptional plants were trisomic at least with respect to the segment of chromosome 1 carrying *His(2-6)*. Indeed, the cytological analysis of one of these trisomics revealed the presence of one additional chromosome (Fig. 3a).

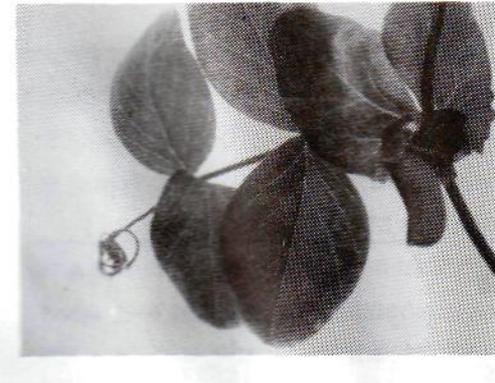


Fig. 1. The phenotype of the *Twt* (Twisted tendrils) mutant.

Fig. 2. Segregation of histone H1 phenotypes in testcross progeny. Lanes 1, 2, 3 and 5 show the heterozygous phenotype *1133/1123* and lanes 7 and 8 show the heterozygous phenotype *1021/1123*. Lane 6 depicts the *1021* H1 pattern of Sprint-2. The trisomic phenotype *1021/1133/1123* is represented by lane 4.

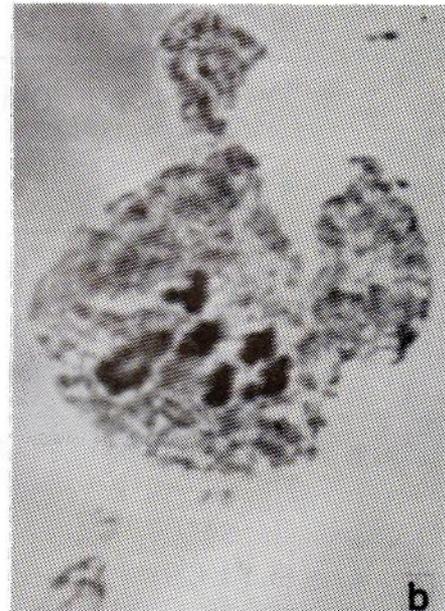
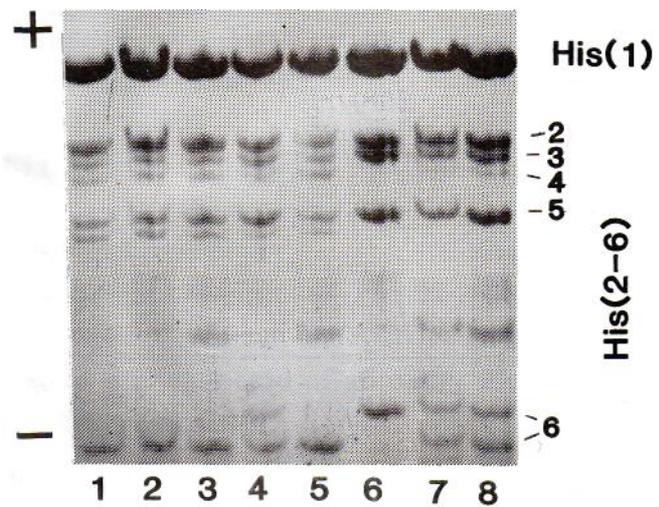


Fig. 3. Acetocarmine staining of different meiotic stages. a) Eight and seven chromosomes at anaphase I in PMC of a trisomic plant having phenotype A, *Twt*, H1-*1133/1021/1123*. b) Five bivalents and one ring of four at metaphase I in PMC of a *Twt/twt* plant from the cross K-3953 x Sprint-2 (*Twt*).

Table 1. Phenotypic distribution in the progeny from the testcrosses:

- 1) (*Twt/twt, A/a, 1021/1133*) x (*twt/twt, a/a, 1123/1123*).
 2) (*twt/twt, a/a, 1123/1123*) x (*Twt/twt, A/a, 1021/1133*).

Phenotype		Testcross 1	Testcross 2
Morphology	H1		
<i>twt, A</i>	<i>1133/1123</i>	171	91
<i>Twt, a</i>	<i>1021/1123</i>	150	107
<i>twt, A</i>	<i>1021/1123</i>	13	9
<i>Twt, a</i>	<i>1133/1123</i>	7	3
<i>twt, a</i>	<i>1021/1123</i>	22	6
<i>Twt, A</i>	<i>1133/1123</i>	7	13
<i>Twt, A</i>	<i>1133/1021/1123</i>	21	7

Table 2. Total number and fraction (in parentheses) of recombinant chromosomes in relation to gene pairs *A, His(2-6)*; *A, Twt*; and *Twt, His(2-6)*.

Test-cross	Gene pairs			Number of chromosomes tested
	<i>A-His(2-6)</i>	<i>A-Twt</i>	<i>Twt-His(2-6)</i>	
1	20 (5.4 ± 1.2)	29 (7.8 ± 1.4)	49 (13.2 ± 1.8)	370
2	12 (5.2 ± 1.5)	19 (8.3 ± 1.8)	31 (13.5 ± 2.2)	229
Σ 1+2	32 (5.3 ± 0.9)	48 (8.0 ± 1.1)	80 (13.4 ± 1.4)	599

The appearance of extra chromosomes may be generated from lines heterozygous for a translocation (2). The trisomy we observed in some of the *Twt* plants may have arisen as a result of a translocation involving chromosome 1, with *Twt* residing at the point of the chromosome break. This hypothesis is in agreement with two observations. First, the pollen fertility of the heterozygous *Twt/twt* plants was approximately 50-70% of that of the homozygous *Twt/Twt* and *twt/twt* plants. Second, five bivalents and one ring of four chromosomes were seen at metaphase 1 in pollen mother cells of heterozygous *Twt/twt* plants (Fig. 3b). The data suggest that EMS can induce structural aberrations in addition to the point mutations. EMS is known to have produced deletions in maize (3). The observations also could be interpreted to indicate that the mutant trait is directly caused by the presence of reciprocal chromosome exchange. At present we cannot determine if *Twt* is a phenotype produced by the translocation or simply reflects an altered DNA sequence near the breakpoint. In either event, *Twt* represents a unique case of an easily discernible dominant Mendelian character associated with a translocation.

1. Belyaev, A.I. and Berdnikov, V.A. 1981. *Genetika (USSR)* 17:498-504.
2. Sutton, E. 1939. *J. Genetics* 38:459-476.
3. Okagaki, R.E., Neuffer, M.G. and Wessler, S.R. 1991. *Genetics* 128:425-431.