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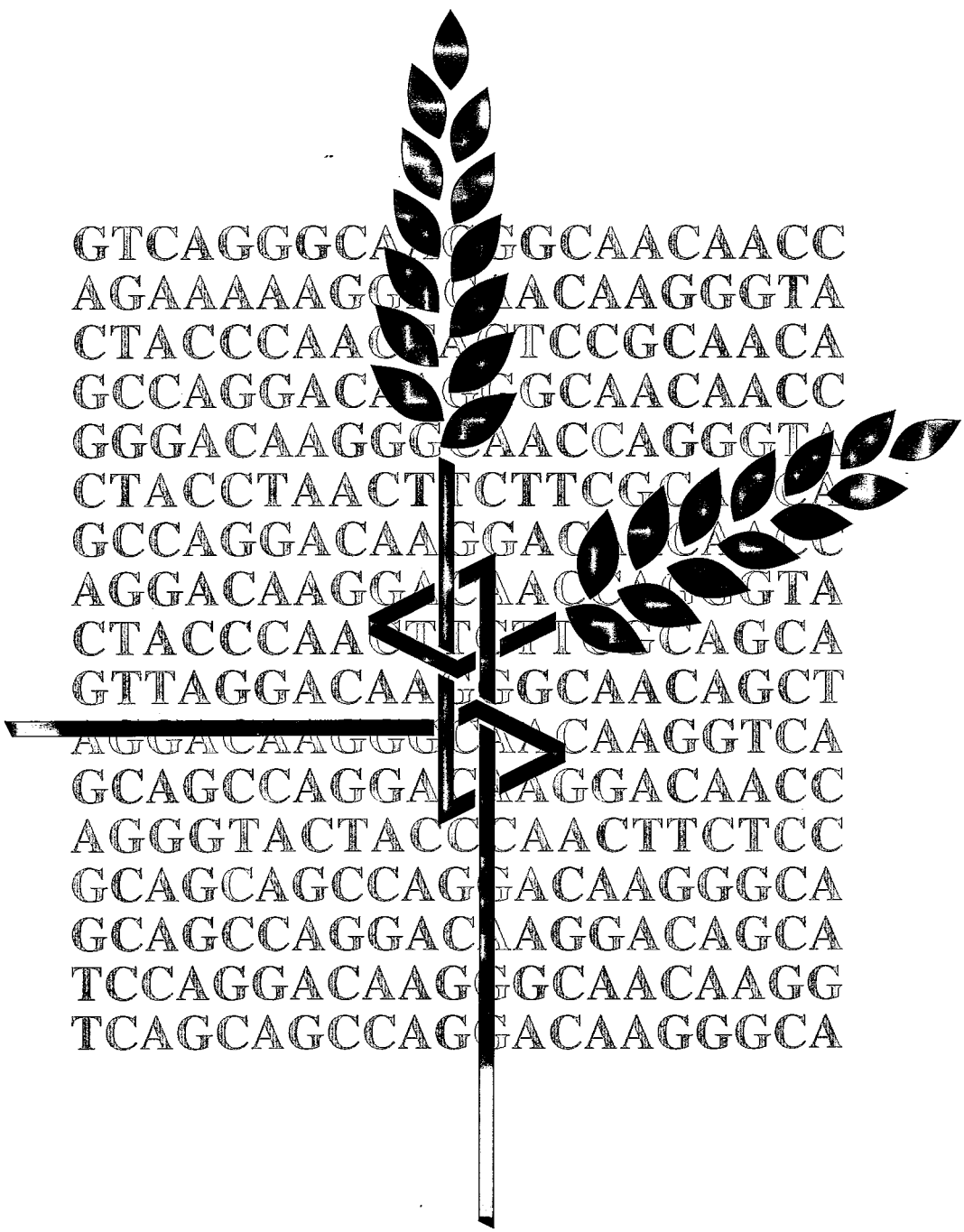

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Research article

Colchicine-induced chromosome doubling in wheat haploids

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Summary

A large number (2,596) of wheat x maize hybridization-derived haploids from 38 genotypes of soft white spring wheat were evaluated for their response to colchicine treatment. Overall, 85.9% of the treated plants survived treatment and 89.9% of these produced viable seed, with a range of 46.7% to 100% among genotypes. The differential response of genotypes to colchicine treatment was highly significant ($P<0.01$).

Key words: wheat haploids, chromosome doubling, colchicine, wheat x maize hybridization

Introduction

Haploid technology offers a useful breeding tool to enhance the speed and efficiency of cultivar development (Baenziger et al. 1984). However, the successful application of this technology to plant breeding depends not only on reliable methods for the production of haploids in large numbers, but also on a means of achieving a high frequency of chromosome doubling to restore their fertility.

Two methods have been used to produce haploids in wheat: anther/microspore culture and wheat x maize hybridization (Laurie and Bennett 1986, 1988; Sadasivaiah et al. 1999). The use of anther culture is limited by an overall low level of haploid production, strong genotype dependency and frequent albinism in regenerants (Orshinsky and Sadasivaiah 1994; Lefebvre and Devaux 1996). The wheat x maize hybridization technique, on the other hand, is less genotype-dependent with no albinism, and the ease with which it can be applied makes it more efficient than anther culture for the production of haploids in common wheat (Sadasivaiah et al. 1999).

Although the occurrence of spontaneous chromosome doubling in anther-derived wheat haploids has been reported, it is generally an infrequent and inconsistent event (De Buyser and Henry 1980; Metz et al. 1988; Orshinsky and Sadasivaiah 1994; Mentewab and Sarrafi 1997; Hansen and Andersen 1998). Furthermore, no such phenomenon has been observed in wheat haploids derived from wheat x maize crosses (Sadasivaiah et al. 1999). Therefore, there is the need for an efficient technique for inducing a high level of chromosome doubling in order to achieve the full potential of haploids in wheat breeding programs.

A number of antimitotic/antimicrotubule agents have shown potential for chromosome doubling in plants (Subrahmanyam and Kasha 1975; Hansen et al. 1988; Hassawi and Liang 1991; Thomas et al. 1997; Hansen and Andersen 1998; Hansen et al. 1998). Of these, colchicine is the most widely used chemical agent for chromosome doubling.

In the present study, the wheat x maize hybridization technique was used to produce haploids from a diversity of genotypes used in our soft white spring wheat breeding program. This paper reports on the success rates of chromosome doubling in these haploids following a colchicine treatment.

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Materials and methods

The genotypes from which the haploids were produced are listed in Table 1. All material was grown in a growth room under a 16/8 h day/night photoperiod and 19/16°C temperature. Haploids were produced using the wheat x maize hybridization technique described earlier (Sadasivaiah et al. 1999). Haploid seedlings with 5-6 tillers (about 8 weeks old) were washed free of soil, and the roots trimmed to about 4-6 cm. The seedlings were then placed in a beaker with the crowns immersed in 0.2% colchicine solution for 5 h at ambient laboratory temperature under low intensity light. After the treatment, the seedlings were thoroughly rinsed in running tap water. The shoots were then trimmed to about 10-15 cm before transplanting into 2.2 in (6.3 cm) square pots containing Cornell mix (Boodley and Sheldrake 1977) and returned to the growth room for further development. The presence of viable seed at maturity was used as the criterion of chromosome doubling due to the colchicine treatment. The percentage of plants with doubled sectors was calculated as the number of plants that produced seed, divided by the number of plants that survived the colchicine treatment and developed spikes. The chi-square test was used to compare frequencies of chromosome doubling. Pearson correlation coefficient was calculated to evaluate the relationship between the number of haploids treated and percent survived and fertile.

Table 1. Genotypes used for the production of haploids with the wheat x maize hybridization technique

Code	Pedigree
F_1 and F_2 material	
L92017	Fielder/89B-18
L92045	AC Reed/89B-18
L96102	AC Reed/Ji-91-4232//AC Reed/U92036
L96120	SWS-179//Fielder/U92036/3/AC Reed/U92036
L97012	AC Reed/SWS-179//AC Reed/Jimai 30
L97103	AC Reed/SWS-179//L87246/SWS-182/3/SWS-214
L97107	AC Reed/SWS-189//AC Reed/Ji-91-4232/3/SWS-214
L98017	Fielder/SWS-214
L98019	AC Phil/SWS-214
L98031	SWS-207/SWS-214
L98032	Centennial/SWS-214
L98033	Penawawa/SWS-214
L98048	FB-42/B-109//AC Phil
L98220	SWS-179/SWS-214//AC Reed/RL4555
L98236	SWS-179/SWS-214//AC Reed/SWS-192
L98243	SWS-132/SWS-179//AC Phil/SWS-214
L98252	AC Reed/SWS-214//SWS-207/SWS-179
L98262	SWS-132/SWS-214//SWS-207/SWS-179
L98265	Centennial/SWS-214//SWS-179/AC Reed
L98266	Penawawa/SWS-214//SWS-179/AC Reed
L99004	L98027/L98037//SWS-223/AC Vista
L99009	SWS-214/SWS-178//AC Vista/AC Domain
L99010	SWS-214/SWS-178//AC Vista/Grandin
L99016	SWS-223/SWS-214//AC Vista
L99016 F2	SWS-223/SWS-214//AC Vista
L99026	SWS-223/SWS-214//AC Barrie/Grandin/3/AC Vista
L99028	SWS-223/SWS-214//AC Domain/Grandin/3/AC Vista
Advanced breeding lines	
B-353	AC Reed//Owens/IDO159
B-359	AC Reed//SWS-103/SWS-18
B-700	IDO236/L2631-19//AC Reed
B-788	Dirkwin/Treasure//Blanca/Fielder
B-792	Dirkwin/Treasure//Blanca/Fielder
B-799	Dirkwin/8021-V2//Treasure/Blanca
B-812	HY355/L(IND)-19//Wadual/L(IND)-21
B-854	IDO236/L2631-19//AC Reed
B-878	SWS-15/SWS-18//Blanca/Treasure
B-91034	AC Reed/Centennial
SWS-179	FB-42/B-109

Results and discussion

The haploids varied with regard to vigor and tillering habit both within and among genotypes. Data on the number of seedlings treated, post-treatment survival and chromosome doubling frequencies (based on seed set) are presented in Table 2. Only 14.1% of the plants failed to survive

colchicine treatment, with a range of 0 to 34.8% among the genotypes. In most cases the mortality appeared to be due to poor seedling vigor resulting in an inability to overcome the toxic effect of colchicine (Jensen 1974; Hansen et al. 1988; Mentewab and Sarrafi 1997; Hansen and Anderson 1998). Of the 85.9% that survived the treatment, only 10.1% were completely sterile, with a range of 0 to 53.3% among the genotypes.

In most cases the 89.9% of plants that survived

Table 2. Fate of wheat x maize hybridization-derived wheat haploids treated with colchicine

Code	No. of seedlings				
	Treated	Dead	Survived	Sterile	Fertile
L92017	39	4 (10.3)†	35 (89.7)	6 (17.1)	29 (82.9)
L92045	29	2 (6.9)	27 (93.1)	1 (3.7)	26 (96.3)
L96102	91	16 (17.6)	75 (82.4)	12 (16.0)	63 (84.0)
L96120	104	24 (23.1)	80 (76.9)	8 (10.0)	72 (90.0)
L97012	136	13 (9.6)	123 (90.4)	10 (8.1)	113 (91.9)
L97103	138	13 (9.4)	125 (90.6)	18 (14.4)	107 (85.6)
L97107	139	21 (15.1)	118 (84.9)	16 (13.6)	102 (86.4)
L98017	20	2 (10.0)	18 (90.0)	7 (38.9)	11 (61.1)
L98019	61	6 (9.8)	55 (90.2)	8 (14.5)	47 (85.5)
L98031	57	4 (7.0)	53 (93.0)	9 (17.0)	44 (83.0)
L98032	78	7 (9.0)	71 (91.0)	5 (7.0)	66 (93.0)
L98033	23	3 (13.0)	20 (87.0)	4 (20.0)	16 (80.0)
L98048	13	4 (30.8)	9 (69.2)	3 (33.3)	6 (66.7)
L98220	102	13 (12.7)	89 (87.3)	9 (10.1)	80 (89.9)
L98236	8	0 (00.0)	8 (100.0)	1 (12.5)	7 (87.5)
L98243	27	5 (18.5)	22 (81.5)	11 (50.0)	11 (50.0)
L98252	23	4 (17.4)	19 (82.6)	6 (31.6)	13 (68.4)
L98262	38	6 (15.8)	32 (84.2)	14 (43.8)	18 (56.2)
L98265	9	1 (11.1)	8 (88.9)	1 (12.5)	7 (87.5)
L98266	23	8 (34.8)	15 (65.2)	8 (53.3)	7 (46.7)
L99004	116	23 (19.8)	93 (80.2)	9 (9.7)	84 (90.3)
L99009	42	10 (23.8)	32 (76.2)	7 (21.9)	25 (78.1)
L99010	167	26 (15.6)	141 (84.4)	12 (8.5)	129 (91.5)
L99016 F_1	123	22 (17.9)	101 (82.1)	14 (13.9)	87 (86.1)
L99016 F_2	206	24 (11.7)	182 (88.3)	12 (6.6)	170 (93.4)
L99026	37	6 (16.2)	31 (83.8)	3 (9.7)	28 (90.3)
L99028	46	4 (8.7)	42 (91.3)	3 (7.1)	39 (92.9)
B-353	106	10 (9.4)	96 (90.6)	1 (1.0)	95 (99.0)
B-359	52	2 (3.8)	50 (96.2)	1 (2.0)	49 (98.0)
B-700	29	8 (27.6)	21 (72.4)	0 (0.0)	21 (100.0)
B-788	44	4 (9.1)	40 (90.9)	0 (0.0)	40 (100.0)
B-792	77	3 (3.9)	74 (96.1)	0 (0.0)	74 (100.0)
B-799	51	2 (3.9)	49 (96.1)	1 (2.0)	48 (98.0)
B-812	187	45 (24.1)	142 (75.9)	4 (2.8)	138 (97.2)
B-854	21	6 (28.6)	15 (71.4)	0 (0.0)	15 (100.0)
B -878	49	8 (16.3)	41 (83.7)	0 (0.0)	41 (100.0)
B-91034	52	3 (5.8)	49 (94.2)	1 (2.0)	48 (98.0)
SWS-179	33	5 (15.2)	28 (84.8)	0 (0.0)	28 (100.0)
Total	2596	367 (14.1)	2229 (85.9)	225 (10.1)	2004 (89.9)

† Figures in parentheses represent percentages

the colchicine treatment and subsequently produced seeds showed only partial fertility, although occasional heads were fully fertile. There was a very low correlation between the number of haploids treated and percent survived (r = 0.03) and the number of haploids treated and percent fertile (r = 0.31). Similarly, the correlation coefficient for the percent survived and the percent fertile was also low (r = 0.37). This suggests that there is no relationship between the number of haploids treated and the rate of doubling. The frequency of surviving plants setting seed varied with the genotype, ranging from 46.7 to 100%, with a differential response that was highly significant ($P<0.01$). In a study of the effect of colchicine treatment in two winter wheat lines, Metz et al. (1988) found that 98% of the treated plants of one line, Centurk, produced seeds, whereas only 43% of another line, NB88, did. A similar genotypic difference in response to colchicine treatment was observed in the wheat haploids studied by Mentewab and Sarrafi (1997).

The seed harvested from colchicine-treated plants was viable and produced phenotypically normal, fully fertile progeny. The colchicine treatment technique used in this study is simple and effective in producing a high frequency of doubled haploids from a diversity of genotypes and facilitates the attainment of instant homozygosity thereby enhancing the efficiency of selection in breeding programs.

Acknowledgments

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Wheat Information Service
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Research article

A one-gene system of cytoplasmic male sterility-fertility in durum wheat

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Summary

The nuclear genome of durum wheat (*Triticum turgidum* L.) is incompatible with *Aegilops longissima* cytoplasm [(*lo*) cytoplasm] and nucleocytoplasmic compatibility is improved by a species cytoplasm specific (*scs*^{t}) nuclear gene located on chromosome 1A of durum. The resulting durum line having (*lo*) cytoplasm and one copy of the (*scs*^{t}) gene is male-sterile and when crossed to normal durum produces plump and viable seeds carrying *scs*^{t}, while seeds without *scs*^{t} are shriveled and non-viable. Our objectives were (1) to transfer the *scs*^{t} gene from (*lo*) male-sterile durum to the cytoplasmic background of normal durum, (2) to determine if the euplasmic durum with a *scs*^{t}/*scs*^{t} gene pair is fertile, and (3) to use the selected euplasmic durum line (if fertile) as a recurrent male parent to propagate a (*lo*) male-sterile durum line. We crossed a 1D(1A) disomic-substitution line of euplasmic Langdon durum as a female to a male fertile line having (*lo*) cytoplasm and *scs*^{t} *scs*^{t} + *ViVi* (vitality) gene pairs. The resulting 1A+1D double-monosomic F_1's were partially fertile. The F_2's were cytologically examined. One euploid F_2 plant was obtained. It was male fertile and when crossed to the (*lo*) *scs*^{t} male-sterile durum produced all plump seeds and male-sterile progeny, indicating that F_2 plant had a *scs*^{t} *scs*^{t} gene pair and no *Vi*. Thus, an euplasmic F_2-derived line carrying a *scs*^{t} *scs*^{t} pair was produced and used as a maintainer B-line to produce a cytoplasmic male-sterile A-line having (*lo*) cytoplasm and a *scs*^{t} *scs*^{t} gene pair.

Key words: *scs*^{t}, *Vi*, cytoplasmic male sterility

Introduction

A cytoplasmic male sterility system (CMS) derived from *Triticum timopheevi* Zhuk., used in research for producing hybrid cultivars of common wheat (*T. aestivum* L.), can also be used for producing hybrid cultivars of durum wheat (*T. turgidum* L.). This CMS system requires labor intensive selection procedures for breeding male fertility restoring lines (R-lines) with a potential to produce fully fertile hybrid wheats, because the native wheat genes that are expressed as sterility in the alien cytoplasmic background also affect fertility in the hybrid wheat cultivars.

The *Triticum* species differ in regards to compatibility with the cytoplasm from some related species (Maan 1983; Sasakuma and Maan 1978). For example, the nuclear genomes of common wheat and *T. timopheevi* are compatible with the cytoplasm of *Aegilops longissima* or *Ae. uniaristata* [(*lo*) or (*un*) cytoplasm, respectively], and the resulting alloplasmic common wheat lines have normal fertility and plant vigor (Maan 1975). In contrast, the nuclear genome of durum wheat is incompatible with the (*lo*) or (*un*) cytoplasm (Maan 1992a, b, 1994).

A species cytoplasmic specific (*scs*^{t}) nuclear gene derived from *T. timopheevi* improves compatibility between the nuclear genome of durum wheat and (*lo*) or (*un*) cytoplasm (Maan 1992a, b). The resulting durum lines are male sterile and maintained by crossing to normal durum wheat. In the successive crosses with normal durum, *scs*^{t} remains heterozygous (or hemizygous) and is transmitted through 50% plump seeds that are viable, while seeds without *scs*^{t} are shriveled and inviable.

The *scs*^{t} gene is closely linked with the centromere on the long-arm of chromosome 1A (1AL) (Anderson and Maan 1995; Maan et al. 1999). A *scs*^{t} gene with an effect similar to the one on 1AL is also located on the long-arm of chromosome 1D (1DL telo) from common wheat and a telocentric chromosome from *Ae. uniaristata* [(*un*) telo] (Maan 1994, 1995). The 29-chromosome durum plants having an alien telo and (*lo*) or (*un*) cytoplasm are male sterile and when crossed to the normal durum produce about 15% plump and viable seeds having the alien telo, while the seeds having euploid embryos are shriveled and inviable. These results show that the alien telocentrics also have a *scs*^{t} gene that improves compatibility with the (*lo*) or (*un*) cytoplasm and the resulting 29-chromosome plants are male sterile (Maan 1992a, b).

A dominantly inherited vitality (*Vi*) gene located on the short-arm of chromosome 1B (1BS) has a positive xenia effect on seed plumpness and seed viability, and produces male fertility in the (*lo*) or (*un*) durum male-sterile lines having *scs*^{t} (Anderson and Maan 1995; Maan 1992a, b). The (*lo*) or (*un*) durum plants having *scs*^{t} *scs*^{t} and *ViVi* gene pairs produce true breeding fertile progeny and when crossed as a male to the (*lo*) or (*un*) *scs* male-sterile durum lines produce plump-viable seeds and fertile plants with the *scs*^{t} and *Vi* genes (Anderson and Maan 1995; Maan 1992b). However, the affects of *scs*^{t} or *Vi* in the cytoplasmic background of the normal durum wheat have not been reported.

Our objectives were (1) to transfer the *scs*^{t} gene from a (*lo*) durum line to the cytoplasmic background of normal durum wheat, (2) to determine the fertility of the euplasmic durum with a *scs*^{t} *scs*^{t} gene pair, and (3) to use euplasmic durum line having a *scs*^{t}/*scs*^{t} gene pair (if fertile) as a recurrent male parent to propagate a (*lo*) male-sterile durum line.

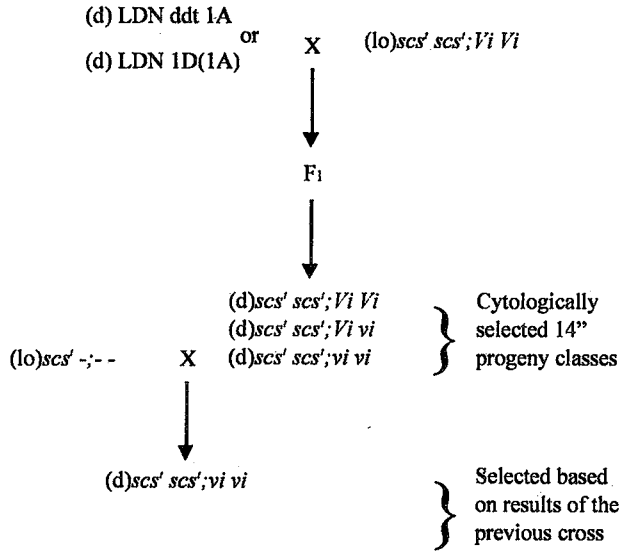

Fig 1. Procedure to transfer a *scs*^{t} *scs*^{t} gene pair to the cytoplasm of normal durum line from *Ae. longissima* cytoplasm. In each cross, the *scs*^{t} gene with whole chromosome 1A is exclusively transmitted through heterosexual gametes.

Materials and methods

The breeding behavior of the genetic stocks used in this study are described below; (1) The (*lo*) durum line carrying one copy of the *scs*^{t} gene is maintained by crossing as the female to control durum. The *scs*^{t} gene is maternally transmitted through the plump and viable seeds, while the seeds without *scs*^{t} are shriveled and inviable (Maan 1992a, b, 1994, 1995). (2) A true breeding fertile durum line has (*lo*) cytoplasm and *scs*^{t} *scs*^{t} and *ViVi* gene pairs (Anderson and Maan 1995). (3) A 1D (1A) disomic-substitution line of Langdon durum (Jappa 1988) (Note: Langdon double-ditelosomic can also be used, because chromosome 1A with *scs*^{t} is exclusively transmitted through heterosexual gametes). (4) The control durum selection 56-1.

We crossed a 1D(1A) disomic-substitution line of Langdon durum as a female parent to the fertile durum line having (*lo*) cytoplasm and *scs*^{t} *scs*^{t} and *ViVi* gene pairs (Fig. 1). The 1A+1D double-monosomic F_1's were partially fertile. The F_2 individuals were cytologically examined. All disomic F_2 individuals were expected to carry a paternal 1A chromosome pair with *scs*^{t} *scs*^{t}, while others may or may not have *Vi*.

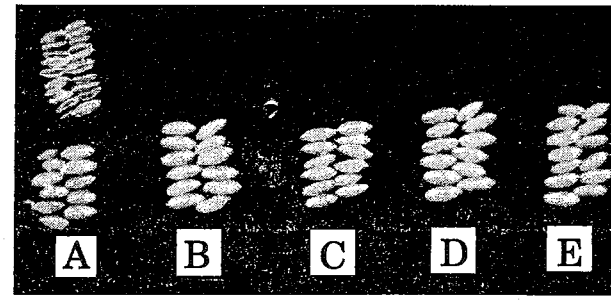

Fig 2. Seeds from the lines tested.
A: (*lo*)*scs*^{t}– durum/ *27 durum (bottom: plump seeds, top: shriveled seeds), B: (*lo*)*scs*^{t} - durum/ (*lo*)*scs*^{t} *scs*^{t}; *Vi Vi* (plump seeds), C: Langdon 1D(1A) disomic substitution line/ (*lo*) *scs*^{t} *scs*^{t} (F_1 seeds), D: Langdon *scs*^{t} *scs*^{t}; *vi vi* (F_2 plump seeds), E: (lo)*scs*^{t} –/ Langdon *scs*^{t} *scs*^{t}; *vi vi* (plump seeds)

An F_2 plant with *scs*^{t} *scs*^{t} (but without *Vi*) was selected by testcrossing to the (*lo*) male-sterile line. The F_2-derived progeny was used as a recurrent male parent to maintain male-sterile durum line having (*lo*) cytoplasm.

The presence of a *scs*^{t} *scs*^{t} gene pair in the male-sterile A-line was confirmed by test-crossing to normal durum. Similarly, the presence of a *scs*^{t} *scs*^{t} pair in the maintainer B-line was confirmed by test-crossing to (*lo*) male-sterile line having one copy of *scs*^{t} gene. The test-crosses were expected to produce plump seeds and male-sterile progeny provided both the A- and B-lines carried a *scs*^{t} *scs*^{t} gene pair (Fig. 2).

Results and discussion

The partially fertile F_1's were obtained from a cross between the 1D(1A) disomic-substitution of Langdon durum as female parent and the (*lo*) durum line having *scs*^{t} *scs*^{t} and *ViVi* genes pairs. In the F_2 generation, *scs*^{t} and *Vi* genes were expected to segregate independently of each other, because *scs*^{t} is located on chromosome 1A and *Vi* on chromosome 1B (Table 1). A cytologically selected disomic F_2 plant was male fertile. This plant was crossed to the (*lo*) *scs*^{t} male-sterile line and produced all plump seeds and male-sterile progeny, indicating the paternal F_2 had a *scs*^{t} *scs*^{t} gene pair but did not have *Vi* (Table 1). Thus, the (*lo*) male-sterile A-line and the euplasmic maintainer B-line both sharing the same *scs*^{t} *scs*^{t} pair were produced.

A similar procedure crossing the euplasmic durum line with the *scs*^{t} *scs*^{t} gene pair with (*un*) durum having *scs*^{t} *scs*^{t} and *ViVi* produced a male-sterile line having the *Ae. uniaristata* cytoplasm [(*un*) cytoplasm] (Maan unpub). These results also showed that the euplasmic durum line with the *scs*^{t} *scs*^{t} gene pair was male fertile but the (*lo*) or (*un*) durum lines with the *scs*^{t} *scs*^{t} gene pair were male sterile.

To maintain cytoplasmic male-sterile A-line, it is essential that a *scs*^{t} *scs*^{t} pair is present in the male-sterile A-line as well as in the fertile maintainer B-line. Crosses were repeated from time to time to make sure that the male-sterile A-line as well as the maintainer B-line carry a *scs*^{t} *scs*^{t} pair (Fig. 2): (a) A cross between (*lo*) male-sterile durum carrying a *scs*^{t} *scs*^{t} gene pair and the normal durum produced plump seeds and male-sterile progeny, because all female gametes carried a *scs*^{t} gene. In contrast, a cross between (*lo*) male-sterile line carrying one copy of the *scs*^{t} gene and normal durum produced plump and viable seeds and shriveled and inviable seeds. These results indicate that the euplasmic maintainer line carried a *scs*^{t} *scs*^{t} gene pair. (b) A cross between a (*lo*) male-sterile line having one copy of *scs*^{t} and an euplasmic maintainer line having an *scs*^{t} *scs*^{t} gene pair produced all male gametes carrying a *scs*^{t} gene.

The *scs*^{t} and *Vi* genes can be distinguished from the *Rf* genes by the differential effects they produce in the durum lines having (*lo*), (*un*), or other alien cytoplasms. For example, *Vi* and *Rf* produce fertility in the durum lines carrying cytoplasm from several species, including *T. timopheevi*, *T. araraticum*, or *Ae. speltoides* (Maan unpub). In contrast, the male fertility restorer lines (R-lines) having *Rf* genes from the above species when crossed to the (*lo*) male-sterile line having one copy of *scs*^{t} produced plump seeds and male fertile progeny having *scs*^{t} and *Rf*, while seeds with *Rf* alone (without *Vi*) were shriveled and inviable (Maan unpub), like those from the cross with control

Table 1. Testing of alloplasmic (*lo*) *scs*^{t} *scs*^{t} and euplasmic (*d*) *scs*^{t} *scs*^{t} durum† lines for the presence of *scs*^{t} *scs*^{t} gene pair, and increase of alloplasmic male sterile lines

Cross No.‡	Female	Male	Number		Seeds/spike	
			Plants	Spikes	Plump§	Shriveled
1§	(lo) *scs*^{t} –	control durum	257	381	14.8	11.1
2	(lo) *scs*^{t}*scs*^{t}	control durum	17	53	24.5	0
3	(lo) *scs*^{t} –	(d) *scs*^{t}*scs*^{t}	23	23	12.3	0
4	(lo) *scs*^{t}*scs*^{t}	(d) *scs*^{t}*scs*^{t}	15	27	27.4	0

† Lines with *scs*^{t} *scs*^{t} pair and *Aegilopis longissima* or durum cytoplasm.

‡ In cross numbers 1 and 3 female parents have one copy of *scs*^{t} gene and in numbers 2 and 4 female parents have two copies of *scs*^{t} gene.

§ Pooled data from 27 successive backcrosses with cultivated durum selection 56-1.

§ Plump seeds produced male-sterile progeny.

durum. These results show that *Vi* produces plump seeeds that result in fertile progeny, while *Rf* alone (without *scs* or *Vi*) produces shriveled and inviable seeds in durum with (*lo*) or (*un*) cytoplasm. The *Rf* genes are dominant to *rf* as such a single copy restores fertility by epistatic interaction with *scs*^{t} in the alloplasmic wheat lines.

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Research article

Genetic identification of an amphiploid between *Triticum aestivum* and *Aegilops variabilis*

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Summary

An amphiploid between *Triticum aestivum* and *Aegilops variabilis* was identified by observation of morphology, cytology and seed storage protein electrophoresis as well as disease resistance surveys in order to evaluate its potential use for wheat improvement. Most morphological traits of the amphiploid are intermediate between its parents, but the plant height of the amphiploid and its selfed progenies is identical to its *Aegilops* parent, with significantly shorter than that of the wheat parent. It is deduced that the *Ae. variabilis* parent would carry a new type of dwarfing gene(s), which is strongly effective in the wheat background. The complete amphiploid contained 2n=70 chromosomes including eight noticeable satellited chromosomes and a great reduction of the chromosome number was observed in amphiploid selfed generation. Giemsa C-banding technique enabled the identification of *Ae. variabilis* chromosomes in wheat genetic background and revealed that the cytological instability of the amphiploid resulted in the loss of *Aegilops* and sometime wheat chromosomes. The most seed storage protein of the amphiploid overlapped those from the parents and the resistance to powdery mildew and stripe rust of *Ae. variabilis* generally expressed in the amphiploid.

Key words: wheat, *Aegilops variabilis*, amphiploid, genetic identification

Introduction

Aegilops variabilis Eig (*Triticum peregrinum* Hackel) is an annual allotetraploid species with genome $U^vU^vS^vS^v$. Genes for resistance to root knot nematode (Yu et al. 1990), kernel bunt (Williams and Mujeeb-Kazi 1996), eyespot (Bang and Hulbergen 1992) and powdery mildew (Spetsov et al. 1993) from this species were successfully transferred to common wheat, *Triticum aestivum* L. Moreover, Dhaliwal et al. (1993) found that most *Ae. variabilis* accessions exhibited high resistance to leaf rust and stripe rust. To exploit the agronomically desirable genes from *Aegilops*, and to investigate their expression in wheat genetic background, we produced a group of hybrids or amphiploids between wheat lines and 41 accessions of nine tetraploid *Aegilops* species (Yang and Liu in press).

Production of amphiploid is an important step for successful gene introgression, and the amphiploid also allows reliable evaluation of genomic interaction between the alien species and wheat (Jiang et al. 1994). To obtain the amphiploid, the crossability of wheat genotype should be considered. A wheat line J-11 was reported to have high crossability genes including *kr1*, *kr2*, *kr3* and *kr4*, with alien species (Zheng et al. 1992). Furthermore, Yang et al. (1998) introduced ph^{1b} gene to J-11 background, named J-11ph^{1b}, which can improve the effectiveness of J-11 to alien gene transfer. An amphiploid between J-11ph^{1b} and *Ae. variabilis* was developed through colchicine

treatment of hybrid F_1. In the present paper, we attempted to identify the J-11ph^{1b} - *Ae. variabilis* amphiploid from morphology, cytology, seed storage protein and disease resistance, in order to evaluate its potential use to wheat improvement.

Materials and methods

Ae. variabilis accession 13E was provided by Dr. Mujeeb-Kazi, CIMMYT, Mexico. Wheat line J-11ph^{1b} was developed by Yang et al. (1998). An amphiploid between J-11ph^{1b} and 13E was obtained from the colchicine treatment of hybrid F_1.

For somatic chromosome counts, root tips of seedling were pretreated with water at 0°C for 24h and fixed in ethanol-acetic acid (3:1) for at least 1 week, and stained by the conventional Feulgen method. Giemsa C-banding procedure was carried out according to Ren and Zhang (1995). Identifications of C-banded *Ae. variabilis* chromosomes mainly followed Friebe et al. (1996).

Endosperm gliadin protein and glutenin subunits were separated by acid polyacrylamide gel electrophoresis (APAGE) and sodium dodecyl sulphate - polyacrylamide gel electrophoresis (SDS-PAGE), respectively. Powdery mildew resistance in seedling and stripe rust resistance in adult-plant were assessed. The methods were identical to those described by Yang et al. (2000).

Results and discussion

Morphologic observation : The amphiploid and its selfed progenies showed upright growth habit with the plant height of 65-70cm. This was identical to that of *Ae. variabilis* parent but lower than wheat parent J-11ph^{1b} with the plant height of 120-130cm. It is likely that the plant height of amphiploid was strongly influenced by *Aegilops* genome. On the other hand, the amphiploid had a mean of six spikes per plant indicating that the tillering ability was affected by its wheat parent, because its *Aegilops* parent always produced more than 20 spikes per plant. The spike characteristics of the amphiploid were intermediate between the parents but showed rachis brittleness and black tenacious glume at maturity (Fig. 1). The fertility of the amphiploid was considerably lower than those of parents and the seed-set rate was less than 30 per cent.

When the wheat and alien genomes are brought together, genomic interaction would affect genetic expression of qualitative and quantitative traits in the new background. The black glume of the amphiploid shows that the genes controlling the color pigmentation located on *Ae. variabilis* chromosomes (Spetsov et al. 1997) was expressed in amphiploid background. The quantitative traits such as plant height were frequently controlled by multigenes. Therefore, such quantitative traits are expected to show intermediate values between the two parents. We developed hybrid plants between wheat lines with other 12 *Ae. variabilis* and *Ae. kotschyi* accessions and found the plant height of the hybrids close to those of taller parents (Yang and Liu unpub.). But the height of the present amphiploid resembled *Aegilops*, a shorter parent. This indicates that *Ae. variabilis* accession 13E may carry a novel dwarfing gene(s) which easily expressed in the wheat background. Pichl (1996) reported that a number of selections from the progenies from crosses between wheat and alien species including *Aegilops* possessed new types of dwarfness. It is likely that the dwarfing system in 13E of *Ae. variabilis* in present study is different from those in common wheat and can be exploited in wheat improvement to enrich the dwarfing resources.

Cytological identification: *Ae. variabilis* has 28 chromosomes. Two pairs of these are satellited

Fig. 1. Spikes of wheat J-11ph^{1b} (left), the amphiploid (middle) and *Ae. variabilis* (right).

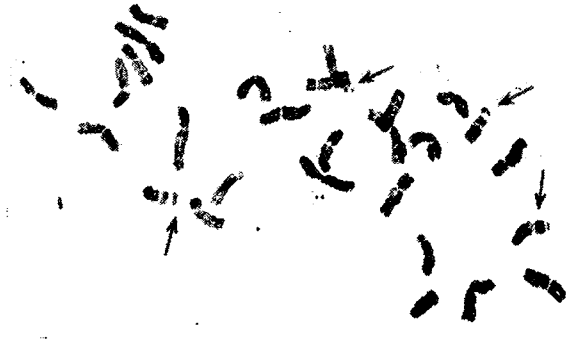

Fig. 2. Somatic metaphase of *Ae. variabilis* (2n=28) with four satellited chromosomes (arrows).

chromosomes, one pair is bigger than the other (Fig. 2). This agrees with the chromosomes $1U^v$ and $5U^v$ carrying satellites in *Ae. variabilis* revealed by Friebe et al. (1996). A complete amphiploid containing 70 chromosomes was identified by somatic chromosome counting (Fig. 3.), and eight satellited chromosomes were observed. Wheat parent J-11ph^{1b} possessed four satellited chromosomes of 1B and 6B. Therefore, the satellite from both J-11ph^{1b} and *Aegilops* chromosomes were totally expressed in its complete amphiploid. This differs from the finding that nucleolar competition existed in many other artificial amphiploids such as primary hexaploid triticale (Lukazaweski and Gustafson 1987).

In the two subsequent generations, the amphiploid displayed high cytological instability. The chromosome number varied from 46 – 66 and averaged 52 in 24 plants derived from the 70-chromosome amphiploid. It is evident that the high ploidy amphiploid always presented continuous decrease of chromosomes after its polyploidization. Spetsov et al. (1993) stated that a 70 chromosome amphiploid between a winter wheat and *Ae. variabilis* exhibits a high level of aneuploidy. But the present amphiploid produced aneuploid at a high level, which may have resulted from the effective gene *ph^{1b}* of its wheat parent.

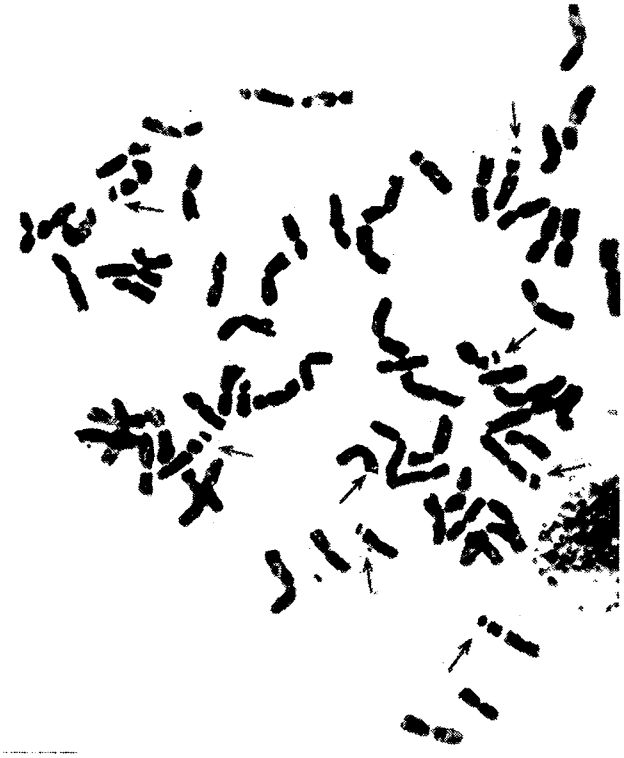

Fig. 3. Somatic metaphase of the amphiploid (2n=70) with eight satellited chromosomes (arrows).

Though interspecific and intraspecific C-banding pattern polymorphisms is present in genus of *Aegilops* (Teoh 1983), it does not prevent the identifications of their chromosomes in wheat background, after the establishment of standard karyotypes of corresponding genome (Friebe et al. 1996). In the present study, the C-banding of S^v genome of *Ae. variabilis* accession 13E is the most similar to that of the B genome in wheat. But these strong telomeric bands in S^v chromosomes allows for their identification. Moreover, the band patterns together with their length and arm ratio make U^v genome chromosomes easily distinguishable from those of wheat chromosomes. Giemsa C-banding patterns of the *Ae. variabilis* chromosomes in the amphiploid and its selfed progenies were similar to those of its diploid state. In C-banded chromosomes of a F_3 plant of amphiploid (2n=48), eight *Ae. variabilis* chromosomes were observed (Fig. 4). It can be concluded that the chromosomes from wheat or *Aegilops* parents may have opportunity to be lost when the amphiploid is selfed, which further confirms the cytological instability of the amphiploid. Moreover, the cytological instability together with the effectiveness of *ph* gene may be beneficial for creating the desirable gene recombination between wheat and *Aegilops* chromatin.

Seed storage protein analysis: APAGE of seed gliadin revealed that the strong bands of *Ae. variabilis* 13E existed in w, r and b zone (Fig. 5B). *Ae. variabilis* contained quite strong and some aggregated bands in w zone. These are totally expressed in amphiploid. The additive electrophoresis patterns of gliadin permit the genetic identification of amphiploid and chromosome markers for directed genetic

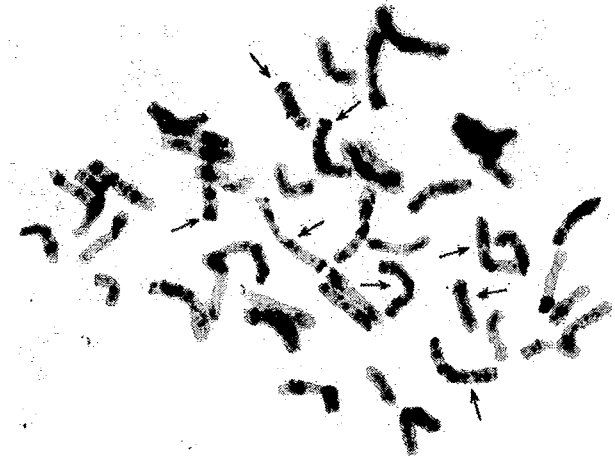

Fig. 4. C-banded karyotype of an amphiploid selfed aneuploid plant (2n=48). Arrows show the *Ae. variabilis* chromosomes.

manipulation. By using the different band patterns as biochemical markers including seed gliadin, Williams and Mujeeb-Kazi (1996) and Spetsov et al. (1998) identified a wheat-*Ae. variabilis* amphiploid and wheat-*Ae. kotschyi* substitution lines, respectively.

The composition of glutenin was analyzed by SDS-PAGE (Fig. 5A). The high molecular weight glutenin subunits (HMW-GS) of J-11ph^{1b} contained null subunit of *Glu-A1*, and subunits 7 + 8 of *Glu-B1*, as well as subunits 2+12 of *Glu-D1*. *Ae. variabilis* also exhibited two strong slower-migrating bands. The slowest band with electrophoretic mobility as the subunit 2.2 of *Glu-D1* can also be observed in the amphiploid selfed plant. Other faster-migrating group of two closely located bands of *Ae. variabilis* were between subunit 8 and subunit 12 of wheat. However, those two bands were modified in amphiploid selfed plant. The faster-moving one was absent, and a new band moving slightly slower than subunit 8 emerged. Furthermore, the amphiploid selfed plant lost the glutenin subunits of both *Glu-B1* and *Glu-D1* from wheat parent, indicating the corresponding chromosomes or segments were lost. This also demonstrated that the wheat chromosomes were lost or modified in the selfed amphiploid background. In addition, the other bands in HMW and LMW regions of the amphiploid are mostly from its *Aegilops* parent.

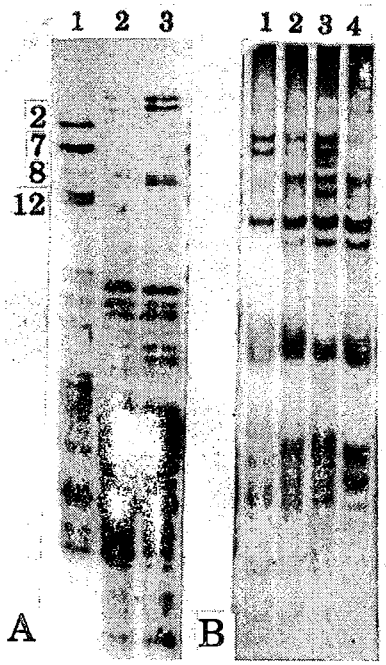

Fig. 5. Electrophoretic patterns of seed storage proteins.
A: SDS-PAGE patterns of glutenin, 1; J-11ph^{1b}, 2; amphiploid selfed plant, 3; *Ae. variabilis*.
B: APAGE patterns of gliadin, 1; J-11ph^{1b}, 2 and 3; amphiploid selfed plant, 4; *Ae. variabilis*.

Spetsov et al. (1997) implied that two of the three HMW subunits of *Ae. variabilis* were expressed in the wheat-*Ae. variabilis* disomic addition and substitution lines. However, in the wheat-*Ae. kotschyi* substitution lines, Spetsov et al. (1998) found that only one of the three HMW subunits were expressed and a new band from *Ae. kotschyi* was produced. The present study also showed that a HMW subunit gene modified its expression in the amphiploid background. Therefore, it is supposed that the variation of genetic expression of HMW subunits of *Aegilops* in wheat background may be caused by gene recombination between the closely homologous chromatins between wheat and *Aegilops*.

Recently, Wan et al. (2000) stated that some HMW-GS of several *Aegilops* species exhibit subunits closely or distantly related to wheat. It is possible that we can use the wheat-*Aegilops* amphiploid to create new glutenin gene recombination between wheat and *Aegilops*. Moreover, gliadin structural genes from *Ae. variabilis* were also quite different from those of wheat. The relationship of the gliadin and glutenin introduced from *Aegilops* to wheat background for improving wheat quality is worth exploring further.

Disease resistance survey: Resistance investigation of the amphiploid were conducted with references to its parents when inoculated by powdery mildew isolates and stripe rust races. *Ae. variabilis* showed high resistance to these tested isolates in seedling and adults plant, respectively. Whereas the wheat parent J-11ph^{1b} was highly susceptible, the amphiploid plant with 2n=70 displayed high resistance to powdery mildew and intermediate resistance to stripe rust. These results indicated that powdery mildew resistance from *Ae. variabilis* was expressed easier than stripe rust resistance in the amphiploid .

The present study suggested that the amphiploid can serve as a donor to transfer the disease resistance from *Ae. variabilis* to wheat breeding. When examining the hybrids of wheat and other *Aegilops* species, we found that about half of them did not express the stripe rust resistance from *Aegilops* accession (Yang and Liu in press). It seems that the expression of resistance from *Ae. variabilis* in the wheat background was independent of the specific wheat genotype or genomic interaction of both parents.

Several plants were recovered from wheat-*Ae. variabilis* amphiploid backcrossed with common wheat. It is expected that some of the spontaneous translocation between wheat and *Aegilops* would be produced following the effects of ph^{1b} gene existed in the amphiploid and the resulting generation. On the basis of present data, the morphological, cytological and biochemical results are beneficial to trace the *Ae. variabilis* chromatin for transferring the novel resistance to powdery mildew and stripe rust to wheat background.

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Research article

An *Aegilops speltoides*-derived *scs*^{spt} gene located in T2BL.2S translocation chromosome of durum wheat

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Summary

The nuclear genome of durum wheat (*Triticum turgidum* L.) is incompatible with *Aegilops ventricosa* cytoplasm [(*vent*) cytoplasm]. A species cytoplasm specific (*scs*^{spt}) gene from *Ae. speltoides* L. improved nucleocytoplasmic compatibility and the resulting (*vent*) durum line was male sterile and partially female fertile. When crossed to normal durum, (*vent*) durum produced 28-chromosome plants with *scs*^{spt} and a meiotic metaphase I configuration indicating that some of the plants had a translocation chromosome. Our objectives were to identify the translocation chromosome and determine the chromosome carrying *scs*^{spt}. We crossed (*vent*) durum to a set of 14 double-ditelosomics of Langdon durum, and crossed the double-monotelosomic (dMt) F_1's to normal durum, and cytologically examined the hybrid progeny. Of the seven A-genome dMt F_1's, six (except dMt 4A) had two type of plants with 13"+ t1t"' or 11"+1^{IV}+t1t"'. Of the eight 6A dMt F_1's, five had 13"+t1t"', two had 11"+(1"')t^{IV}+t', and one had 11"+ t(1"')t^{V}, while the progeny of dMt 2A included one plant with 12"+2t1", indicating that (*vent*) durum had a 2A.6A translocation. Seven B-genome F_1's had only 13"+t1t"' and produced disomics and dMts from crosses to normal durum, while one (dMt2B) produced only disomics, and one plant with a spontaneous maternal telosome that remained unpaired with the paternal chromosome 2B (2n=28; 13"+1'+t'). In summary, the (*vent*) durum has (a) two, T2A.6A and T2B.2S, translocation chromosomes, (b) *scs*^{spt} is located in *Ae. speltoides*-derived 2S that is homoeologous to 2BS of durum, and (c) recombinants with *scs*^{spt} and durum chromosome 2B were not obtained.

Key words: *scs*^{spt} vs. *Gc* genes, T2A.6A and T2BL.2S translocation chromosomes.

Introduction

The cytoplasms from several species of *Aegilops*, including *Ae. longissima*, *Ae. uniaristata*, and *Ae. ventricosa* [(*lo*), (*un*) and (*vent*) cytoplasms, respectively] are fully compatible with the nuclear genome of common wheat (*Triticum aestivum* L.) (Maan 1975, 1978). The resulting common wheat lines were fertile and used as the sources of alien cytoplasms to produce alloplasmic durum wheat (*T. turgidum* L.) lines, because cytoplasmic genes have exclusive maternal inheritance in the Triticeae species. The (*lo*), (*un*) or (*vent*) durum lines, thus produced, were male sterile and retained a long-arm telocentic of chromosome 1D (telo 1DL) from common wheat. Telo 1DL has a species cytoplasm specific (*scs*^{ae}) gene that improves compatibility between these cytoplasms and nuclear genome of durum wheat (Tsuji and Maan 1981; Maan and Endo 1981, 1991). The resulting 29-chromosome male-sterile lines when crossed to normal durum produce plump and viable seeds having embryos with 1DL yielding 29-chromosome male-sterile progeny, while the fertilization of 14-chromosome female gametes without 1DL (*scs*^{ae}) by 14-chromosome male gametes of normal durum produce shriveled and inviable seeds. Telo 1DL remained unpaired during meiosis in 29-chromosome plants, and therefore, *scs*^{ae} in 1DL was not transferred to a durum chromosome. Male-sterile lines with a T1AL.1DL (Maan et al. 1999) or T1BS.1DL translocation chromosome carrying *scs*^{ae} have been

obtained (Maan unpub).

A *scs*^{t} gene from *T. timopheevi* also improved compatibility between the nuclear genome of durum wheat and (*lo*) or (*un*) cytoplasm (Maan 1992a). The resulting alloplasmic durum lines were male sterile, produced plump and viable seeds having embryos with *scs*^{t} resulting in 28-chromosome male-sterile progeny, while seeds without *scs*^{t} were shriveled and inviable. The *scs*^{t} gene is located on the long arm of chromosome 1A (1AL) and closely linked with the centromere (Anderson and Maan 1995; Maan et al. 1999). But, the *scs*^{t} gene was not transferred to telo 1AL of Langdon durum by recombination in the progeny of double-monotelosomic 1A F_1's (dMt; 2n=29; 13"+t1t"') from crosses of (*lo*) *scs*^{t};- durum to double-ditelosomic 1A of Langdon durum (2n=30; 13"+2t"; dDt 1A) nor misdivision of complete chromosome 1A produced telo 1AL with *scs*^{t} in the progeny of 1A+1D double-monosomic (dM; 2n=28; 13"+2') F_1's from a cross of (*lo*) *scs*^{t};- durum to 1D(A) double-disomic of Langdon. Possibly, a gene or genes in the short arm of chromosome 1A (1AS) may be essential for the functioning of *scs*^{t} in 1AL.

A vitality (*Vi*) gene of spontaneous origin located on 1BS produced fertility in the (*lo*) durum lines carrying *scs*^{ae} in 1DL on the 29-chromosome plants as well as 28-chromosome plants carrying *scs*^{t} on the complete chromosome 1A (Maan 1992b). The *Vi* gene also produced fertile (*lo*) durum or euplasmic durum lines having heterozygous or homozygous *scs*^{ae} in T1AL.1DL or T1BS.1DL translocation chromosome (Maan unpub).

Similarly, a *scs*^{spt} gene from *Ae. speltoides* produced compatibility between (*vent*) cytoplasm and the nuclear genome of durum wheat (Maan unpub). The resulting (*vent*) durum line was male sterile and when crossed to normal durum produced plump and viable seeds having embryos with *scs*^{spt}, while female gametes without *scs*^{spt} did not function. The *Vi* gene did not produce fertility in the (*vent*) *scs*^{spt} durum, indicating that *scs*^{t} and *scs*^{spt} differ in regards to interactions with the (*lo*), (*un*), or (*vent*) cytoplasm, even though these cytoplasms are similar to one another with regards to compatibility with the nuclear genomes of durum or common wheat (Maan 1975, 1978).

In the progeny from successive crosses of (*vent*) *scs*^{spt};- durum to normal durum, some of the plants had pollen mother cells (PMC's) with a meiotic metaphase I configurations (2n=28; 14" and 12"+1^{IV}), indicating that the (*vent*) durum carried a translocation chromosome (Maan unpub). The objectives of this study were to identify the translocation chromosome and determine the chromosomal location of *scs*^{spt} in the (*vent*) durum line.

Materials and methods

A durum line (Selection 56-1) with a *scs*^{spt} gene from *Ae. speltoids* and cytoplasm from *Ae. ventricosa* [(*vent*) *scs*^{spt} durum] was produced by S. S. Maan at North Dakota State University, Fargo, ND (Maan unpub). A set of 14 double-ditelosomics of Langdon durum (CS-LDN dDt) were produced by Joppa (1988).

The (*vent*) *scs*^{spt} durum, maintained by crossing to durum Selection 56-1, was crossed to a set of 14 CS-LDN dDt (2n=30; 13"+2t"). Some of the (*vent*) *scs*^{spt} durum plants were cytologically examined to assure the presence of a translocation chromosome. The resulting double-monotelosomic (dMt) F_1's (2n= 29; 13"+t1t"') were crossed to normal durum. Hybrid progeny were cytologically examined to determine the meiotic chromosome number and chromosome pairing at meiotic metaphase I in the pollen mother cells (PMC's).

The laboratory and greenhouse procedures were the same as described by Maan et al. (1999). The experimental plants were grown in a greenhouse at Fargo, ND. The spikes of male-sterile plants were covered with glycine bags prior to anthesis and recovered after pollination to prevent out-crossing with stray pollen from other wheat plants in the greenhouse.

Results and discussion

In a set of 14 dMt F_1's, the six A-genome F_1's (except dMt 4A) were of two types; type-1 had PMC's with 13"+t1t"' and type-2 had PMC's with 13"+t1t"' as well as 11"+1^{IV}+t1t"' (Table 1). Eight 6A dMt F_1's were examined; five had 13"+tlt"', two had 11"+111t^{IV}+t" and one had 11"+t(1"')t^{v}. In contrast, each of the seven B-genome dMt F_1's were of only type-1 having PMC's with 13"+tlt"' and no type 2 plants (Table 1). These results indicate that (a) the A-genome dMt F_1's had a multivalent configuration that was absent in the B-genome dMt F_1's, and (b) chromosome 6A was involved in a translocation that produced the quadrivalent configuration including telocentrics. Perhaps, (*vent*) durum plants used in crosses with the dDt's of the B-genome chromosomes did not have a 2A. 6A translocation chromosome and, therefore, did not produce a multivalent in the PMC's of F_1 plants. Five dMt 7B F_1's were examined; four had 13"+t1t"' and one had 13"+t1t"'+tS' in which the short arm telo (tS) remained unpaired.

Of the 14 dMt F_1's crossed to normal durum, 13

produced disomics (2n=28; 14" or 12"+1^{IV}) and dMts (13"+t1t'"), while 1 (dMt2B) produced 21 plants having PMCs with 14", 12"+1^{IV}, 13"+t1"+1', or 13"+1'+tS' but no dMt (Table 2), indicating that the female gametes having maternal chromosome 2B were transmitted. Thus, the *scs^{spt}* gene is located on chromosome 2B and female gametes without *scs^{spt}* did not function. However, one exceptional plant had a maternal telocentic 2BS (tS) that remained unpaired (in all 11 PMC's examined) with the paternal chromosome 2B from normal durum, indicating that tS was homoeologous to the short-arm of chromosome 2B of durum and did not pair with it. Therefore, chromosome 2B in (*vent*) durum consists of the long arm of chromosome 2B of durum and short-arm (tS) of 2B from *Ae. speltoides*. Additionally, the presence of two plants with 12"+t1t'", one from a cross with dMt 2A and one from a cross with dMt 6A, indicate that the (*vent*) durum line has a 2A.6A translocation chromosome (Table 2).

Of the 79 plants from a cross of dMt 1A F1 to normal durum, 19 had 14", 58 had 13"+t1t'", one had 13"+t1" and 1 had 12"+1'"+t1t'", indicating that the 15-chromosome female gametes having telocentrics 1AL+1AS and 13 normal chromosomes had a functional advantage over those having 14 maternal chromosomes of normal durum (Table 2). Our explanation is that the telocentrics 1AL and 1AS in the (*vent*) dMt F1 have certain gene(s) from the Chinese Spring double-ditelosomic 1A (CS-dDt 1A) that are not present in chromosome 1A of normal durum. The CS-dDt 1A is the progenitor of Langdon-dDt 1A (Joppa 1988). The residual CS gene(s) in Langdon dDt 1A may have improved nucleocytoplasmic compatibility and provided functional advantage to gametes carrying telocentic 1AL+ 1AS, because CS is fully compatible with (*vent*) cytoplasm (Maan 1975). However, of 41 plants from a cross of dMt 2A to normal durum, 15 had 14" or 12"+1^{IV}, 22 had 13"+t1t'" or 13"+t1" indicating that the female gametes of dMt 2A F1 carrying 14 normal chromosomes of durum functioned nearly as well as those with 13 normal durum chromosomes and 2AL+2AS telocentrics (Table 2).

Table 1. Meiotic chromosome pairing in F1's from crosses of (*Ae. ventricosa*) durum to a set of 14 double-ditelosomics of the Langdon durum

Cross†	No. of cells examined	Chromosome configurations	
		13"+t1t'"	11"+1^{IV}+t1t'"
1A	6	4	2
2A	9	6	3
3A	10	6	4
4A	4	3	1‡
5A	17	16	1
6A	8	5	3§
7A	9	5	4
1B	9	9	0
2B	4	4	0
3B	8	8	0
4B	4	4	0
5B	2	2	0
6B	4	4	0
7B	5	4	1¶

†Cross is of the type (*Ae. ventricosa*) durum x double ditelosomic Langdon durum. The double ditelosomic chromosome is listed here.

‡The plant was haploid

§Of the 3 plants, 2 had 12"+111t^{IV}+t' and 1 had 12"+t111t^{V}

¶The plant was 13"+t1t'"+t'S

The dMt 2A progeny included additional seven plants; one each with 13"+1' (from 13-chromosome female gamete) or 13"+1'" (showing a 6A.2A/2A.6A translocation configuration), or 9"+6'"+t1"+4' (from a 28-chromosome unreduced female gamete), respectively, fertilized by the 14-chromosome male gametes, and 14' or 12"+2t" (a haploid and diploid produced by apomixis, respectively), and 10"+2^{IV}, or 11"+2^{IV} (having 4 chromosomes in each translocation configuration) (Table 2). Similarly, dMt 1B produced two additional plants; one had PMC's with 12"+ 2t1" (Table 2), indicating that an additional translocation involved chromosome 1B and another chromosome, and one having PMC's with 4"+t1"+18' (with reduced meiotic pairing), indicating that this plant had a deletion of the asynaptic gene in chromosome 3A. The gametocidal (*Gc^{spt}*) gene in the T2BL.2S may have produced plants with unexpected chromosome numbers and meiotic configurations that resulted from chromosomal breakage and deletions, because *scs^{spt}* was hemizygous in the male-sterile plants with a T2A.6A. Also, certain genes producing apomixis and meiotic non-reduction may have been occasionally expressed in certain florets of (*vent*) *scs^{spt}*;- durum (Table 2). In a similar study (Maan et al. 1999) crosses between a (*Ae. longissima*) durum line having a *scs^{t}* gene from *T. timopheevi* and a set of 14 double-ditelosomics of Langdon durum (LDN-dDts) produced dMt F1's with the expected meiotic chromosome number and pairing (2n=29; 13"+t1t'") and neither dMt F1's nor the progeny from crosses to normal durum produced PMC's with a multivalent

configuration. Therefore, there was no chromosomal structural heterozygosity between LDN-dDts and durum Selection 56- 1 , while (*vent*) scs^{spt};- durum had one or more translocation chromosomes.

The scs^{t} and scs^{spt} genes produced compatibility between the nuclear genome of durum wheat and the cytoplasm of *Ae. longissima* (*lo*) or *Ae. ventricosa* (*vent*), respectively. Similarly, scs^{ae} in 1DL from common wheat produced compatibility with the (*lo*) and (*vent*) cytoplasms (Maan 1992b). Therefore, the scs^{spt}, scs^{t}, and scs^{ae} genes are similar in regards to producing compatibility with the (*vent*) cytoplasm and the resulting durum lines are male sterile. From crosses with normal durum, the female gametes without scs^{t} or scs^{ae} were functional but produced shriveled seeds, while female gametes without scs^{spt} did not function. Either scs^{spt} on 2S produced nucleocytoplasmic compatibility as well as gametocidal activity or 2S has two genes; *Gc* and scs^{spt}.

A common wheat line with a T2B.2S translocation chromosome has a gametocidal gene in 2S from *Ae. speltoides* (Tsujimoto and Tsunewaki 1988) but the action of *Gc* in the alien cytoplasm has not been examined. Telosome 2S also has a scs^{spt} (Table 2) and female gametes without scs^{spt} did not function in the (*vent*) durum. Similarly, durum wheat and common wheat lines having chromosome 4SL from *Ae. longissima* or *Ae. sharonesis* have a *Gc* gene (Maan 1975, 1976), and a T4AL.4SL translocation chromosome, like 2BL.2S, has *Gc* as well as scs^{t} genes that produce compatibility with the cytoplasm from the donor *Aegilops* species and gametes with T4AL.4SL are exclusively transmitted in the euplasmic as well as alloplasmic durum wheat lines (Maan unpub). The long arm of chromosome 1D (1DL) has a scs^{ae} gene in a T1AL.1DL or TIBS.1DL chromosome that produces compatibility between the nuclear genome of durum wheat and the *Ae. longissima* cytoplasm (Maan et al. 1999; Maan unpub). Similarly, a gene(s) in chromosome 1D from *Ae. squarrosa* produce distorted segregation favoring alien chromosome 1D over native chromosome 1D of common wheat (Dvorak pers comm), indicating that the alien chromosome 1D had *Gc*-like activity. Thus,

Table 2. Meiotic chromosome number and pairing in the progeny from crosses of (*Ae. ventricosa*) durum double-monotelosomic F1's and normal durum.

Cross†	No. of cells examined	Chromosome configurations				
		14"	12"+1^{IV}	13"+t1t"' or 11"+1^{IV}+t1t"'	13"+t1" or 11"+1^{IV}+t1" or 13"+1'	Others
1A	79	19	0	58	1	1 (12"+1"'+t1t")
2A	41	10	5	19	3	4 (11"+2^{IV}; 13"+1"'; 13"+1'; 14')
3A	12	7	0	3	1	1 (12"+t1t"'+t1")
4A	4	1	2	0	1	0
5A	25	13	1	7	1	3 (13"+1'; 13"+11t"'; 3x=2"+12"'+2')
6A	32	17	1	7	3	4 [(1)12"+2t1"; (2)13"+1"'; (1)12"+t1"+t1t"']
7A	31	14	5	9	1	2 (12"+t1"+tS')
1B	41	31	3	4	1	2 (12"+2t1"; 4"'+t1"+18')
2B	21	15	4	0	0	2 (13"+t1"+1'; 13"+1'+tS')
3B	16	7	1	7	0	1 (13"+t1"+tS')
4B	23	12	0	8	1	2 (13"+tS1"+tS'; 3x)
5B	9	4	0	4	1	0
6B	14	3	1	9	0	1 (13"+2')
7B	14	7	0	5	1	1 (12"+t1t"'+1')
11"+1^{IV}+t1t" 1A	8	0	0	8	0	0
11"+1^{IV}+t1t" 2A	13	0	5	0	5	3 (12"+2t1"; 10"+2^{IV}; 3x=9"+6"'+4'+t1")
11"+1^{IV}+t1t" 3A	5	3	1	1	0	0
11"+1^{IV}+t1t" 4A	7	1	0	4	2	0
11"+1^{IV}+t1t" 7A	5	0	1	4	0	0
13"+t1"+tS 6A	4	0	1	1	2	0

†Cross is of the type 13"+t1t"' (telosomic chromosomes are listed) x durum except for where noted.

2S from *Ae. speltoids*, 4SL from *Ae. longissima*, and 1D chromosomes from *Ae. squarrosa* have *scs* genes that are expressed differently in the different wheat genotypes. Alternatively, two genes, *scs* and *Gc*, are located in the three non-homoeologous chromosomes from three diploid species. In addition, certain chromosomes of other *Aegilops* species have *Gc* genes with different degrees of reduced *Gc*-like effects but their interactions with the alien cytoplasms have not been examined.

The action of the *scs* genes is cytoplasm specific. While *Gc* appears to be non-cytoplasm specific, even though *Gc* is more effective in certain alien cytoplasms than others; *scs* genes produce interspecific nucleocytoplasmic compatibility and female gametes having *scs* are functional, while female gametes without *scs* either do not function or when fertilized by male gametes of normal durum (not having *scs*) produce aborted seeds. Functionality of the male gametes, female gametes, and seeds without *scs* (or *Gc*) may represent different degrees of nucleocytoplasmic compatibility, and the female gametes function better because of the maternal effect(s) than male gametes.

According to the *scs* gene hypothesis (Maan 1995), different forms of native ancestral *scs* genes produce nucleocytoplasmic compatibility and fertility within species having differentiated nuclear and cytoplasmic genomes. The parental *scs* genes are expressed as sterility when hemizygous in the interspecific hybrids. Some of the alien *scs* genes, when experimentally transferred into the nuclear genomes of the tetraploid or hexaploid *Triticum* species (with or without the alien cytoplasms), also function as *Gc* genes that in certain interspecific combinations impair DNA repair mechanism and produce chromosomal breakage, numerical and structural chromosomal aberrations, including deletions, male and female sterility, self- or cross-incompatibility, exclusive preferential functioning of the male gametes and female gametes, differential survival of zygotes and/or seeds.

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Research article

Effectiveness of *Triticum tauschii* (*Aegilops squarrosa*) derived *Lr* genes in conferring resistance to Indian races of leaf rust (*Puccinia recondita tritici*) of wheat

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Summary

Effectiveness of nine *Triticum tauschii* (syn. *Aegilops squarrosa*) derived *Lr* genes in conferring resistance to nine prevalent leaf rust (*Puccinia recondita tritici*) races at the seedling stage in the glass-house and against the most virulent Indian race 77-5 (known to attack all the *Lr* genes originating from *T. aestivum*) at the adult stage in the field was studied. *Lr22b* was ineffective while *Lr41* was effective against all the races tested at the seedling and adult stage. *Lr38* behaved differentially while *Lr42* showed susceptibility against race 104-2 only. *Lr21*, *Lr22a* and *Lr43* appeared to confer adult plant resistance against race 77-5. Potential of these alien genes in exploiting diverse resistance for strategic use in wheat breeding is discussed.

Key words: *Triticum*, *Aegilops*, leaf rust, resistance, *Puccinia recondita*

Introduction

Leaf rust caused by *Puccinia recondita* Rob. ex Desm. f .sp. *tritici* Eriks and Henn. is a common disease of wheat, *Triticum aestivum* L. in India. Breeding for disease resistance in this crop is almost synonymous with breeding for leaf rust resistance in all the regions in India. Genetic studies have led to the naming of nearly 45 leaf rust resistance genes (*Lr* genes) globally (McIntosh et al. 1995; Singh et al, 1998). The spread of many leaf rust pathotypes showing combined virulence for the most commonly occurring genes *Lr23* and *Lr26* in Indian cultivars resulted in the susceptibility of several commercial wheat cultivars (Nayar et al. 1994). This has necessitated search for new sources of resistance to leaf rust effective against Indian races.

According to Kerber and Dyck (1979), *Aegilops squarrosa* being a donor of D genome to hexaploid wheat, the transfer of leaf rust resistance from this diploid progenitor to hexaploid wheat is relatively easier and also permits selection against deleterious genes closely linked with the resistance genes. The present report analyzes the effectiveness of several *Lr* genes transferred from Asian goat grass, *Triticum tauschii* (syn. *Aegilops squarrosa*) to common wheat (Cox et al. 1994) against the pathotypes prevalent in different wheat growing regions in India.

Materials and methods

Seeds of nine Thatcher (Tc) near-isogenic lines carrying the genes *Lr21, Lr22a, Lr22b* and *Lr38* through *Lr43* were obtained from Dr. P.L. Dyck (195-Dafoe Road, Manitoba, Canada) and used for the present work. Two Indian cultivars Agra Local and WL711 (*Lr13*) were used as leaf rust susceptible cultivars. The wheat lines possessing *Lr* genes transferred from *T. tauschii* and the two susceptible cultivars were sown in plastic trays in the glass-house. First leaf of seven day-old seedlings was inoculated with uredospore-talc mixture of each of the races, 12, 77, 77-1, 77-2, 77-3, 77-4, 77-5, 77-5, 77-6 and 104-2, separately. The seedlings were inoculated at 100% relative humidity for 24 hr and kept in separate glass-houses maintained at 20 ± 1°C. The infection types (ITs) were recorded 14 days later, following McIntosh

et al. (1995).

For adult plant tests 15-20 seeds of the line were sown in 2 m long paired rows, spaced 30 cm apart. These lines were surrounded by two spreader rows of each of the susceptible cultivars Agra Local and WL711. The spreader rows as well as the lines carrying genes from *T. tauschii* were sprayed with water suspension of uredospores of the race 77-5 on alternate days. Race 77-5 is the most frequent and virulent amongst the Indian races and attacks all Indian cultivars carrying *Lr* genes originating from *T.aestivum* (Saini et al. 1998; Sawhney et al. 1998). The inoculations were continued till the leaf rust started appearing on susceptible cultivars. The field was irrigated adequately to ensure high humidity needed for the leaf rust development. Disease reaction was recorded as per cent rust severity on modified Cobb scale (Peterson et al. 1948) as well as pustule type (Roelfs et al. 1992) during 1996-1999.

Results and discussion

Nine *Lr* genes transferred to *T. aestivum* from *T. tauschii*, as already mentioned, were evaluated for resistance against nine prevalent pathotypes of *P. recondita tritici* at the seedling stage in the glasshouse and against race 77-5 at the adult stage in the field during 1996-1999 (Table 1). Lines carrying the genes *Lr22a* and *Lr22b* and cultivars Agra Local and WL711 showed high (susceptible) infection types (3 or 3+) against all the leaf rust races used. On the other hand, the line carrying the gene *Lr41* showed low (resistant) reaction against all the pathotypes. The line carrying *Lr42* mainly differed from *Lr41* in showing susceptibility against pathotype 104-2. The lines carrying the genes *Lr39* and *Lr40* showed high infection types against race 12, 77 and 104-2 only. The reaction pattern of these two lines was different from that of the other *T. tauschii* derived *Lr* genes.

At the adult stage, the leaf rust reaction during the years 1996-1999 was 60S on the line carrying the gene *Lr22b* which is comparable to the susceptible cultivars Agra Local and WL711. The disease reaction ranged from free to traces (Tr) on *Lr41* to 20MS–40 MS on line carrying *Lr42* during this period. The field reaction on lines with the genes *Lr21, Lr22a* and *Lr43* against race 77-5 was low even though these lines showed high seedling reaction. These three genes appear to confer adult plant resistance against race 77-5. Since 77-5 attacks all the *Lr* genes originating from *T. aestivum*, *Lr* genes effective against this pathotype can provide useful and diverse resistance for strategic use in breeding programs. According to McIntosh et al. (1995), *Lr21* has potential for use in breeding but it remained largely unexploited. Sawhney (1997) reported the successful use of *Lr21* in wheat for leaf rust management. The genes *Lr22a, Lr40* and *Lr41* derived from *T. tauschii* conferred high level of adult plant resistance to race 77-5 and thus have potential for use in wheat improvement. Except for 104-2, the line carrying the gene *Lr42* has shown seedling resistance against all the races including 77-

Table 1. Reaction of some *T. tauschii* derivatives with known *Lr* genes against some Indian leaf rust races

Wheat line/cv.	Seedling reaction† and race									Adult plant reaction‡
	12	77	77-1	77-2	77-3	77-4	77-5	77-6	104-2	
Tc+*Lr21*	;1-	3c	3	3	X†-	X-	33+	33+	;1	Tr–10MR
Tc+*Lr22a*	33+	33+	33+	33+	33+	3	33+	33+	3	5MR–10MS
Tc+*Lr22b*	33+	3	33+	33+	33+	33+	33+	33+	3	60S
Tc+*Lr38*	33+	3	33+	;1	;	–	;	33+	33+	Tr–40S
Lr39 (WGRC2)	33+	3	X	;1	X at base 3 at tips	– –	;12	–	33+	5MR–30MS
Lr40 (WGRC7)	33+	3	X	;1	X†-	?	;1N	–	33+	Tr–10MR
Lr41 (WGRC10)	0;	0;	;	;	0;	0;	;	0;	0;	Tr
Lr42 (WGRC11)	–	;	0;	0;	;	0;	;	0;	3	20MS–40MS
Lr43 (WGRC16)	–	;	3	;1+	;1*-	3	33+	;1-	33+	5MR–30MS
Susceptible cultivars										
Agra Local	33+	33+	33+	33+	33+	33+	33+	33+	33+	60S–80S
WL711 (*Lr*13)	33+	33+	33+	33+	33+	33+	33+	33+	33+	60S–80S

†According to McIntosh et al. (1995). ‡According to Peterson et al. (1948) and Roelfs et al. (1992). – : Not tested.

5 but this line has shown relatively low adult plant response of 20MS–40MS. Such response is unlikely if the gene *Lr42* is stable for expression at high temperature (30°C and above) which is prevalent when leaf rust reaction is recorded in this part of the country.

Access to diverse genetic stocks possessing different *Lr* genes is an essential pre-requisite for a dynamic crop improvement program. Therefore, the genes *Lr21*, *Lr22a*, *Lr*40 and *Lr41* derived from *T. tauschii* can be generally utilized against the *P. recondita tritici* races prevalent in India.

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Research article

Breaking yield barriers in wheat – new plant type designed

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Summary

To achieve a quantum jump in wheat productivity in India, there is a need to design a new plant type combining negatively correlated yield components in a single genotype of very high yield potential. In this endeavor, a new plant type combining two negatively correlated traits (high tiller numbers with high grain weight and high grain weight with high grain number per ear) has been successfully developed resulting in significantly high yield. The second phase advanced generation materials, in pipeline, have optimum combination of all three yield components and carry alien genes (*Lr24/Sr24*) for resistance to leaf, stem, and stripe rusts giving further boost to yield potential. The newly designed plant type has characteristic features of moderate tillering (375 per m^2 and above), all productive tillers, 50 and above grains per spike, grain weight between 45 and 50 g per 1000 grains, higher biomass, dark green, thick and broad leaves, thick stem, maturity duration between 120 to 135 days and plant height between 85 and 100 cm.

Key Words : wheat, yield components, plant type, rust resistance

Introduction

India witnessed the Green Revolution in mid 1960's due to large scale planting of high yielding, semi dwarf wheat varieties developed at CIMMYT, Mexico. The semidwarf wheats reduced the yield loss due to lodging resistance and were responsive to higher levels of inputs. The new plant architecture replacing tall types was responsible for increasing wheat yields from one tonne per hectare in early 1960's to nearly 2.7 tonnes per ha in late 1990. However, to keep pace with the population growth, India will need 109 m tonnes of wheat by the year 2020. To achieve this target, the average yield must be increased from 2.7 tonnes per ha to 4.0 tonnes per ha. To achieve this quantum jump in wheat productivity, the Indian Agricultural Research Institute initiated a strategic research in 1994 designing a new plant type. Optimally the new plant type has achieved the objective of three yield components, i.e., grain weight, grain number per spike and tillers per plant along with dark green thick and broad leaves and thick stems. This plant type is the first of its kind in the country and probably in the world.

Materials and methods

The materials involved as parental lines in the development of high yielding genotypes were local germplasm (SFW) and two released wheats (Vaishali and Vidisha) with bold, lustrous grains and carrying tightly linked resistance genes for leaf and stem rust from *Agropyron elongatum* (*Lr24/ Sr24*).

SFW has very long ear-heads with high spikelet number but unfilled middle spikelets, long and shriveled grains, and fewer tillers per plant and high susceptibility to rusts. This germplasm line has been included as one of the parents in crossing program with a view to filling all the spikelets with bolder grains and combining other desirable traits from the second parent.

The other parents Vaishali and Vidisha which were crossed with the local type are released cultivars for timely sown irrigated conditions and late sown irrigated conditions in North Eastern Plains Zone and

Central Zone, respectively. These parents were chosen with the objective to combine genes for resistance to stem and leaf rusts and also to look for optimum combination of tillering habit, grain weight and grain number in single genotype. Along with this material, four varieties, namely, PBW343, HD2687, HD2329 and UP2338 were included as checks in the present investigation for comparing yield and yield components.

The new efficient plant type combining desirable yield components along with resistance to stem and leaf rusts was developed through modified pedigree method of selection from F_2 to F_5 generations. Two crosses of SFW with Vaishali and Vidisha were attempted and F_1's were bulked. A very large F_2 population (approx. 2500 plants) of these two crosses was planted. The spreader rows were planted all around and in between, at regular interval. The artificial epiphytotic of leaf rust was created by inoculating the spreader rows with the urediospores of most virulent and prevalent pathotype 77-5 with the help of hypodermal syringe. The selection in F_2 generation was exercised for plants combining optimum tillering, long and well filled ear-heads and resistance to leaf rust. Selected F_2 plants were individually harvested and screened for well-filled, bold and lustrous grains; F_3 families were raised from F_2 plants in 2.5 m x 2 rows plots. In selected F_3 families exhibiting resistance to rust and good tillering, long and well filled ear-heads were picked up and threshed individually. In F_4 and F_5 generations, ear row progenies were planted in epiphytotic conditions of leaf rust, and long, well-filled ear-heads were selected from the selected progeny rows having desired plant type and grain selection was exercised. The finally selected ear-heads from a progeny row were bulked and evaluated in a yield trial comprising 6 rows of 5 m length with 3 replications evaluated in randomized block design during 1999-2000. One square meter plot from the middle of the plot of each entry in all replications was cut from the ground level when it is fully matured. The data on biological weight, grain yield, number of tillers were recorded from this one square meter plot area. The

Table 1. Mean values of yield and yield components

Varieties	Biological yield/m² (g)	Grain yield/m² (g)	No. of tillers/m²	1000-grain weight (g)	Grains/ ear	Plot yield (kg)
DL 1266-2	2160	708 (1)§	335	45.60	46.4	4.320
DL 1266-1	1883	696 (2)	274	51.47	49.8	3.829
DL 1280-1	2113	668 (3)	611	46.13	23.8	4.134
DL 1266-6	1953	649 (4)	341	40.27	47.7	3.603
Vidisha†	2007	643 (5)	541	36.00	33.6	3.969
PBW 343‡	1957	625 (6)	515	33.47	36.4	4.125
DL 1267-3	1803	625 (7)	376	39.73	41.9	3.765
DL 1267-2	1957	625 (8)	377	39.73	41.9	3.778
HD 2329‡	1820	620 (9)	482	34.93	37.4	3.861
DL 1270-4	1950	610 (10)	487	38.00	33.1	3.830
DL 1266-3	1940	609 (11)	279	44.93	48.7	3.995
Vaishali†	1917	604 (12)	532	37.20	30.8	3.771
DL 1266-5	1807	601 (13)	222	48.67	56.0	3.781
HD 2687‡	2037	584 (14)	467	29.60	41.5	3.497
UP 2338‡	1930	553 (15)	446	29.47	42.5	3.627
SFW†	1723	402 (16)	304	33.33	40.4	2.702
CV (%)	8.8	11.6	13.0	5.1	10.9	-
SE	42.5	17.8	13.3	0.49	1.11	-
CD at 5%	283.4	118.5	88.6	3.330	7.39	-
CD at 1%	381.6	159.6	119.3	4.483	9.95	-

†Parents. ‡Checks.

§Values within parenthesis indicate ranks.

number of grains per ear-head were calculated from randomly selected 50 ear-heads from the harvested plot. The data collected on various traits were analyzed for variance (ANOVA) and correlation among these traits.

Results and discussion

All the traits (grain yield/m^2, number of tillers/m^2, 1000-grain weight and number of grains/ear) except biological yield were highly significant which indicate very high variability in the material under study.

Out of the nine newly developed lines, four lines yielded higher than all checks including the most popular variety PBW343 in northern and western parts of the country (Table 1). The line DL1266-2 significantly out yielded the best check variety PBW343 and also both parental lines (SFW and Vaishali). This genotype is the ideal genotype with respect to optimum combination of tillers (335/m^2), high grain weight (45.6 g) and number of grains per ear (46). This genotype also occupied first rank on mean yield per plot basis. Breeding genotypes combining optimum tillers per plant and higher yield contributing traits (grain weight and number of grains), DL1266-2 is the most suitable example.

Another genotype DL1266-1 from the same cross also yielded higher than all the four checks and parental lines. However, it ranked 8th on the basis of grain yield per plot. This genotype has the best combination of 1000-grain weight (51.5 g) and grains per ear (50) but has low tillering habit. This type of genotype becomes the base material for further improvement in tillering habit. DL1266-6 is the third genotype of the same parentage, which is similar to DL1266-2 in optimum combination of yield contributing traits.

The fourth genotype DL1280-1 from a cross between HD2329 and Vaishali is also superior to all the check varieties. However, this genotype has a different combination of yield contributing traits. It has the highest number of tillers (611/m^2) and also very high 1000-grain weight (46.1 g) but less number of grains per ear (24). This genotype ranked second on the basis of grain yield per plot.

DL1267-3 (SFW x Vidisha) that yielded on a par with the best check (PBW 343) has optimum combination of all the three yield contributing traits.

SFW has low tillering (304/m^2) and low 1000-grain weight (33.3 g) and moderate grains per ear (40) but the number of spikelets per spike were very high, though poorly filled. It has been observed that the grains were very long but shriveled resulting in low grain weight. The two parents Vaishali and Vidisha have higher tillers per plant and high grain weight and low grain number per ear.

With the new strategy, DL1266-2, DL1266-1 and DL1266-6 (with common parentage) have been developed with new plant type (Fig. 1) where in the physiological efficiency of partitioning of dry matter to economic yield has increased. This increase in physiological efficiency is due to increased availability of photosynthate for proper filling of sink leading to very high grain weight and proper filling of all the grains in all the spikelets resulting in higher number of grains per ear. In fact, in SFW, the number of spikelets/spike are very high but grain formation is low because the poorly filled grains are highly shriveled and unaccountable. The grains per ear in line DL1280-1, which do not have SFW as one of the parents, are very low (24), further suggesting the role of SFW in contributing to high grain number in newly developed genotypes. It is generally observed by several workers (Gandhi et al. 1964; Bhatt 1973; Chaudhary et al. 1977; Sinha and Sharma 1979; Balyan and Singh 1987; Pawar et al.1990) that two yield contributing traits, grain weight and grain

Fig. 1. A designed new plant type.

Table 2. Correlation coefficients among yield and yield components

Characters	Biological yield	Grain yield	No. of tillers	1000-grain weight	Grains/ ear
Biological yield	-	0.727**	0.402**	0.120	-0.129
Grain yield		-	0.212	0.479**	0.086
No. of tillers			-	0.418**	0.874**
1000-grain weight				-	0.327*

* and ** : Significant at 5%and at 1%, respectively

number per spike are negatively correlated. It is also known that the increase in tiller number per plant leads to decrease in grain weight and number of grains per ear. However, the newly constituted plant type (in the form of DL1266-1, DL1266-2 and DL1266-6) has shown increase both in grain weight and grain number per ear with moderate tillering capacity (Figs. 2 and 3). The significant positive correlation between grain weight and grains per ear is evident from this study (Table 2).

It was also possible to successfully combine high tiller number and high grain weight in a genotype DL1280-1, which are otherwise negatively correlated components of yield.

Concerted efforts are on in the direction of amalgamating two positive combinations of yield components present in DL1266 and DL1280-1, and optimising selection criteria leading to maximization of productivity. This advanced material in pipeline has passed through preliminary yield trials and are under testing in replicated multilocation trials.

The *Agropyron elongatum* derived leaf rust resistance gene *Lr24* is effective till today in the Indian subcontinent. This leaf rust resistance gene is linked with stem rust resistance gene *Sr24* which is effective to an array of virulent and prevalent races in the country. All the newly developed genotypes in the study carry this combination of leaf and stem rust resistance genes, introgressed through two released wheats Vaishali and Vidisha, the carrier of linked genes *Lr24/Sr24*. All these genotypes were found highly resistant to leaf and stem rusts when tested as seedlings in the glasshouse and as adult plants at hot spots.

Conclusion

The strategy adopted to design a new plant type has resulted in the development of wheat varieties having 15.2% more yield than PBW343, HD2329 and UP2338, the most popular wheat varieties in the wheat belt of the country. The new plant type possesses moderate tillering, higher number of grains per spike, high grain weight (above 45 g/1000 grains) and higher biomass with dark green thick broad and

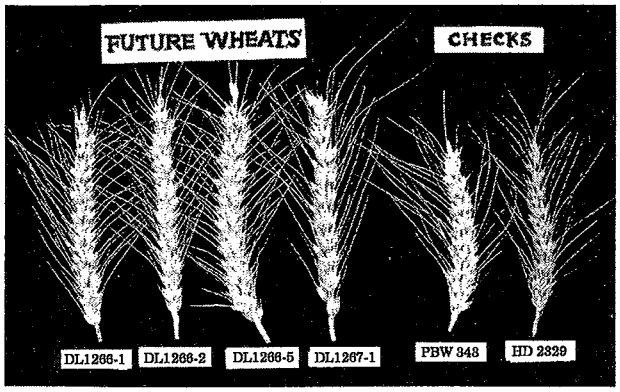

Fig. 2. Spikes of new plant type wheats and check varieties.

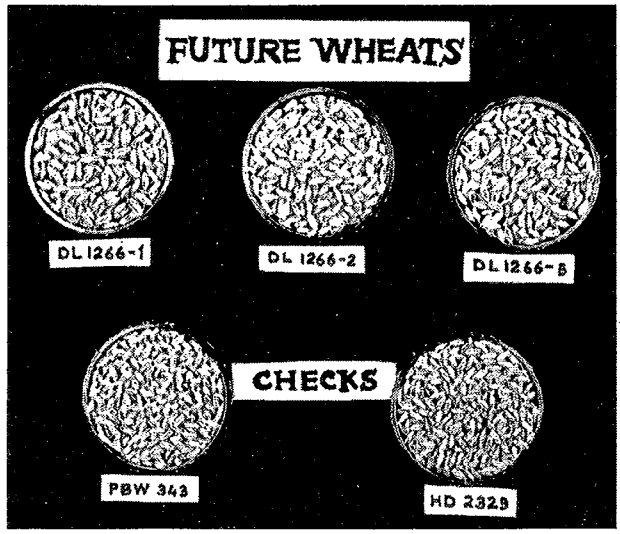

Fig. 3. Grains of new plant type wheats and check varieties.

erect leaves. It also has thick stem having a plant height between 85 and 100 cm. The newly constituted lines have maturity duration between 120 and 135 days. Some of the early maturing lines like DL1266-1 and DL1266-6 also have more per day productivity than the check varieties and fit well in areas planted late after the harvest of rice. The yield levels of three lines may increase by 20-30% in improved production management so as to harness maximum yield potential. The second generation material developed by utilizing the above genotypes (DL1266 group) are more promising as they combine all the three yield components viz. high number of grains per spike, high grain weight and high number of tillers per plant along with resistance to leaf, stem and also to stripe rust.

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Research article

Third dominant male sterility gene in common wheat

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Summary

The mutants producing dominantly inherited male sterility (*Ms*) survive in the polyploid species. Hexaploid wheat (*Triticum aestivum* L.) has two *Ms* genes, *Ms2* on chromosome 4D and *Ms3* on chromosome 5A. Our objective was to determine the identity of a putative third *Ms* gene. First, we crossed an euploid *Msms* plant to double-ditelosomic 5A (dDt; 2n=44; 20"+2t") of Chinese Spring (CS), a monoditelosomic 5A (mDt) *Msms* F_1 (2n=43; 20"+t1t"' 5A) to nullisomic 5A-tetrasomic 5D [(5A) 5D; 2n=42; 19"+1^{IV}] of CS, a 43-chromosome *Msms* plant (19"+1"'5D+2t'5A) to CS, and produced a 1:1 ratio of fertile and sterile plants, showing that *Ms* was not located on chromosome 5A. Second, we crossed an euploid *Msms* plant to (4D)4A of CS, a tri 4A-mono 4D *Msms* (2n=43; 19"+1"'+ 1') plant to dDt 4D of CS, and produced *Msms* plants (20"+t1t"'4D) or dMt (20"+2t'4D) and fertile Dt plants (20"+ 2t') or Mt (20"+t'), showing that *Ms* is located on chromosome 4D. Third, a *Msms* dMt 4DL (20"+1t") plant was crossed to CS and produced 21 sterile plants with 21", 21 fertile plants with 20"+1t"4DL but no recombinants (*msms* with 21" or *Msms* with 20"+1t"), showing that *Ms* is located in the short-arm of chromosome 4D (4DS). Fourth, two *Msms* mDt 4D (20"+t1t"') plants (one with *Ms* in whole chromosome 4D and the other with *Ms* in telo 4DS) were crossed to CS and produced 1:1 ratios of fertile and sterile plants with 21" as well as 20"+t1t", indicating that the new *Ms* (*Ms4*) gene is located in the distal portion of 4DS, while 4DS also has *Ms2* with 31.16 crossover units from the centromere.

Key words: *Triticum*, polyploidy, male sterility, mutation, diploidized genes

Introduction

Common wheat has several male sterile mutants, but only three have been located to specific chromosomes. One X-ray induced *ms1* recessive mutant is a null locus in the short arm of chromosome 4A (previously chromosome 4B) (Sears 1954; Driscoll 1977). Franckowiak et al. (1976) and Sasakuma et al. (1978) reported one dominant (*Ms3*) and several recessive male sterile (*ms*) mutants that were produced by EMS treatment of seed in the common wheat cultivar, Chris, which has cytoplasm from *Aegilops squarrosa* L. The recessive *msms* gene pair produced male sterility in the euplasmic as well as alloplasmic lines of common wheat. Sasakuma et al. (1978) examined allelic relationships among the *Ms* and *ms* mutants. Klindworth and coworkers (pers comm) determined that two of those EMS-induced *ms* mutants studied by Sasakuma et al. (1978) were allelic to *ms1* located on chromosome 4A.

The *Ms3* mutant producing dominantly inherited male sterility is closely linked to the centromere in the short-arm of chromosome 5A of alloplasmic common wheat (Maan et al. 1987). Maan and Williams (1984) transferred *Ms3* to euplasmic common wheat by using limited functional pollen in the male-sterile plants grown under higher than normal greenhouse temperature conditions. The *Ms3* gene is equally effective in producing dominantly inherited male sterility in the euplasmic and alloplasmic common wheat cultivars and can be maintained by backcrossing with common wheat or sib-mating sterile and fertile segregants.

Genesis of the *Ms2* gene is not known for certain. The *Ms2* gene is 31.16 crossover units from the centromere in the short arm of chromosome 4D (4DS) and is presumably carried in the euplasmic common wheat (Liu and Deng 1986a, b; Deng and Huang 1988).

The objective of this study was to determine the identity of a putative third dominant *Ms* gene and to distinguish it from those that are known to be located on chromosomes 5A, 4A and 4D of common wheat.

Materials and methods

A common wheat line carrying a putative third *Ms* gene producing dominantly inherited male sterility was received from Professor C.F. Konzak, Washington State University, Pullman, Washington, USA. Several aneuploid stocks of Chinese Spring wheat (CS) were used (Sears 1954, 1963). These included nullisomic 4D-tetrasomic 4A (nulli 4D-tetra 4A) and nulli 5A-tetra 5D, double-ditelosomic 4D (dDt 4D; 2n=44; 20+2t"), and dDt 5A that were originally received from E.R. Sears, University of Missouri, Columbia, Missouri, USA and were available from a previous study (Maan et al. 1987). The euploid CS was used as a control in some of the crosses.

The male-sterile plants can be crossed only as female and, therefore, the *Ms* genes can not be tested for allelism by conventional methods involving reciprocal crosses between wheat lines with *Ms* genes from different sources. Therefore, a modified monosomic analysis (Maan et al. 1987) involving the chromosomal location, chromosome arm location and gene-centromere distance was used to determine the relationship of the putative third *Ms* gene to those in the short-arms of chromosomes 5A and 4D (Maan et al. 1987; Liu and Deng 1986a, b; Deng and Huang 1988).

First, the progenies from a series of four crosses involving a euploid male-sterile plant with the putative new *Ms* gene were examined along with aneuploids of chromosomes 5A and 5D to determine if the new *Ms* gene was located on chromosome 5A; (a) a male-sterile plant carrying a *Msms* gene pair was crossed to dDt 5A, (b) a resulting male-sterile F_1 (dMt 5A; 2n=43; 20"+t1t'"5A) to nulli 5A-tetra 5D, (c) a resulting male-sterile segregant with 19"+1'"5D+2t'5A to CS. and (d) a resulting male-sterile plant with 20"+t1t'"5A to CS (Table 1).

Second, the progenies from crosses involving an euploid *Msms* male-sterile plant and aneuploids of chromosomes 4A and 4D were examined to determine if the putative third *Ms* gene was located in chromosome 4D; (a) a male-sterile plant carrying a *Msms* gene pair was crossed to nulli 4D-tetra 4A and (b) a trisomic 4A-monosomic 4D male-sterile F_1 (20"+1'"4A +1'4D) to dDt 4D (Table 2).

Third, the cytologically identified male-sterile plants of specific chromosomal constitutions from above progenies were crossed to CS; (c) a plant with 20"+t1t'"4D carrying *Ms* on whole chromosome 4D was crossed to CS; (d) a plant (from a above) with 20+ t1" 4DL carrying *Ms* on whole chromosome 4D was crossed to CS; and (e) a plant with 20"+t1t'"4D but carrying *Ms* in telocentric 4DS was crossed to CS (Table 2). The purpose of these crosses was to locate the *Ms* gene to a specific arm of chromosome 4D and determine its linkage to the centromere.

The experimental plants were grown in a greenhouse in Fargo, North Dakota. One or more spikes of the segregants were examined for anther extrusion and seed set in the bagged spikes, and meiotic chromosome number and pairing was evaluated in the pollen mother cells (PMC's) at the metaphase I of meiosis. The plants were assigned idealized chromosomal constitutions according to the maximum observed meiotic pairing involving chromosomes being tested for the presence or absence of the *Ms* gene as described by Maan et a1. (1987). The Chi-square test was used to compare the probability of fit between the observed and expected ratios of the fertile and sterile segregants in the hybrid progenies.

Results and discussion

First, the segregation for chromosomal constitution, fertility, and sterility in the hybrid progeny from a series of four crosses was examined starting with a cross of an euploid male-sterile plant to the aneuploids of group 5 chromosomes to determine if the putative new *Ms* gene was the same as *Ms3* gene that is closely linked with centromere in the short-arm of chromosome 5A (Maan et al. 1987). The results are described below (Table 1).

A cross of an euploid male-sterile plant to dDt 5A produced 20 fertile and 15 male-sterile F_1's (a 1:1 ratio; P=0.398), and a cross of a male-sterile F_1 (2n=43; 20"+t1t'"5A) to nulli 5A-tetra 5D produced 20 fertile and 19 male-sterile plants (a 1:1 ratio; P=0.875), including 7 fertile and 7 male-sterile segregants with 19"+1'"+1' having maternal chromosome 5A and 11 fertile and 7 male-sterile plants with 19"+1'"+2t'5A having maternal telocentrics. Similarly, a cross of a male-sterile plant with 19"+1'"+2t' 5A to CS produced 38 fertile and 43 sterile plants (a 1:1 ratio; P=0.575), including 14 fertile and 18 sterile plants with

Table 1. Segregation for chromosomal constitution and fertility or sterility in progeny from crosses of common wheat plants having a dominant gene for male sterility and Chinese Spring wheat (CS) or CS aneuploids

Cross‡	Meiotic chromosome number and meiotic chromosome pairing†										Total	Ratio tested	Probablity§
	21"	19"+1'"+1'	20"+t1t'"	19"+1'"+2t'	20"+1'	19"+1'"+t'	20"+1tL"	20"+1tS"	19"+1'"+t1t'"	20"+(1")t'"			
21" x (20"+2t"5A)			20/15								20/15	1:1	0.398
(20"+t1t'"5A) x [19"+1'"5D(5A)]		7/7		11/7		2/1				0/4	20/19	1:1	0.873
(19"+1'"5D+2t'5A) x CS		14/18			15/10	2/9	3/4	3/0	1/0	0/2	38/43	1:1	0.579
(20"+t1t'"5A) x CS	9/8		5/13				2/0				16/21	1:1	0.411

†The ratio indicates the number of fertile/ number of sterile plants.

‡In all crosses the maternal plants carry the *Ms* gene.

§In each cross a ratio of 1 fertile : 1 sterile total plants (and with individual types of chromosomal constitution) shows that the new *Ms* gene is not located in the maternal chromosome 5A, because *Ms3* is located in the short-arm of chromosome 5A closely linked to the centromere and recombination are rarely produced (Maan et al. 1987)

Table 2. Segregation for chromosomal constitution and fertility or sterility of progeny from crosses of male sterile plants of different chromosomal constitution and nullisomic 4D-tetrasomic 4A, double-ditelosomic 4D, or Chinese Spring (CS) wheat

Cross‡	Meiotic chromosome number and meiotic chromosome pairing†								Total	Ratio tested	Probablity§
	21'	19"+1'"+1'	20"+t1t'"	19"+1'"+1t" or 20"+tS'	20"+1tL"	19"+1'"+2t' or 20"+2t'	20"+tL' or 20"+tS'	19"+1'"+tL' or 19"+1'"+tS'			
a 21" x [19"+1'"4A(4D)]		12/10							12/10	1:1	0.670
b (19"+1'"4A+1'4D) x (20"+2t"4D)			0/15	0/5		39/0	32/0	20/0	91/20	3:1	0.089
c (20"+t1t'"4D) x CS	28/23		13/24						41/47	1:1	0.522
d (20"+t1"4DL) x CS	0/21				21/0				21/21	1:1	1.000
e (20"+t1t'"4D) x CS	28/16		31/41		6/0				65/57	1:1	0.469

†The ratio indicates the number of fertile/ number of sterile plants.

‡Cross e has *Ms* on the maternal t4DS, other crosses have *Ms* on the maternal complete chromosome 4D

§Crosses a, c, d and e fit the expected 1:1 ratio of fertile to sterile plant. Cross b fits a ratio of 3 fertile to 1 sterile plants.

19"+1 '"+1' and 15 fertile and 10 sterile plants with 20"+1'. These results indicated that the *Ms* gene was not located on chromosome 5A of the original euploid male-sterile plant used in this study. If *Ms* was located on chromosome 5A or was transferred to one of the 5A telocentics by recombination the expected ratios would be 3 fertile to 1 sterile plants, because the unpaired univalent chromosome with *Ms* should be transmitted through 25% of the female gametes (Sears 1954) but instead was transmitted to 50% of the progeny (Table 1).

The results from another cross of a male-sterile plant having 20"+t1t'"5A to CS confirmed the above conclusion. This cross produced 16 fertile and 21 male-sterile plants (1:1 ratio; P=0.411), including 9 fertile and 8 sterile plants with 21" and 5 fertile and 13 sterile plants with 20"+t1t'". These results indicated a more frequent occurrence of recombination between *Ms* and the centromere than was expected of *Ms3* because of close linkage to the centromere on chromosome 5A (Maan et al, 1987). Therefore, the new *Ms* gene is not located on chromosome 5A and is not the same as *Ms3*.

Next, the segregation for chromosomal constitution, fertility, and sterility was observed in progeny from a series of five crosses starting with crosses of an euploid male-sterile plant to the aneuploids of group 4 chromosomes. This was to determine if the *Ms* gene in this study is the same gene as *Ms2* located on the short-arm of chromosome 4D, 31.16 crossover units from the centromere (Liu and Deng 1986a, b; Deng and Huang 1988). The results are described below (Table 2).

A cross between a euploid male-sterile plant and nulli 4D-tetra 4A produced 12 fertile and 10 male-sterile tri 4A-mono 4D F_1's (2n=43; 19"+1'"4A+1'4D) (Table 2). A cross of a tri 4A-mono 4D male-sterile (2n=19"+1'"+1'4D) F_1 to dDt 4D produced 91 fertile plants in which maternal 4D was absent and 20 male-sterile plants in which maternal 4D was present (3:1 ratio; P=0.089). These results indicated that the new *Ms* gene was located on chromosome 4D. Of the 20 male-sterile plants, 15 had 20"+t1t'" and 5 had 19"+1'"+1t" or 20"+ tS'. A male-sterile plant with 20"+1t4D" was used in the cross described below.

The progeny from a cross between a male-sterile plant 20"+t1"4DL and CS produced 21 male sterile plants with 21" and 21 fertile plants with 20+t1"4DL (Table 2). The absence of fertile recombinant(s) with 21" and sterile recombinant(s) with 20+t1" indicated that the *Ms* gene was located on the short-arm of chromosome 4D.

A cross between a male-sterile plant with 20"+t1t'"4D carrying *Ms* on the whole chromosome 4D and CS produced a ratio of 1 fertile recombinant to 1 sterile non-recombinant with 21" ($P < 0.50$) and 1 sterile recombinants to 1 fertile non-recombinants with 20"+t1t'" ($P< 0.50$), indicating that the *Ms* gene segregated independently of the centromere on 4DS. Similarly, a male-sterile plant with 20"+t1t'"4D (carrying *Ms* on 4DS) from a cross with CS produced 16 sterile recombinants and 28 fertile non-recombinants with 21" ($P<0.05$) and 31 fertile recombinants and 41 sterile non-recombinants with 20"+t1t'" ($P< 0.25$), indicating that the female gametes carrying maternal telosome 4DS with *Ms* had a functional advantage over those carrying *Ms* on whole chromosome 4D. In conclusion, the new *Ms* gene (now designated *Ms4*) is located in the distal portion of 4DS, where *Ms2* is located 31.16 crossover units from the centromere.

The *Ms* genes producing dominantly inherited male sterility are rare in the higher plant species (Kaul 1988). The dominant or recessive mutants producing male sterility can be used to enhance outcrossing under natural conditions in a self-pollinating crop species such as common wheat (Sorrells and Fritz 1982).

Molecular biology techniques can be used to determine whether a *Ms* mutation inactivated a diplodized fertility gene or activated a silent gene from the diploid progenitor in the polyploid wheat. Also, the dosage effects and inheritance patterns of the *Ms* and other mutants indicate the degree to which certain genes from the diploid progenitors have been diplodized by natural selection after the formation of common wheat (Sears 1972).

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Research information

'Thatcher'-avirulent leaf rust pathotypes from India

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Bread wheat cultivar Thatcher is documented to carry a single gene (*Lr22b*) for adult plant resistance to leaf rust (McIntosh et al. 1995), while its seedling is susceptible to wheat leaf rust. Therefore, it has been used as the genetic background to develop the near-isogenic lines for resistance to leaf rust. However, leaf rust pathotypes avirulent to Thatcher seedlings have been known to occur in Ethiopia and Morocco until now (Huerta-Espino and Roelfs 1992). The present communication reports three Indian leaf rust pathotypes carrying avirulence to Thatcher seedlings. These three pathotypes of leaf rust, 0R8, 0R8-1, and 0R9, belonging to standard leaf rust races 11, 63, and 106 are maintained at the DWR-Shimla. These pathotypes were also avirulent to seedlings of the 14 near-isogenic lines with Thatcher background carrying a resistance gene to leaf rust, *Lr1, 2a, 2c, 3a, 10, 13, 14a, 15, 17, 18, 19, 23, 24* or *26*. They showed virulence to a bread wheat Agra Local, to which the Ethiopian and Moroccan isolates were avirulent (Mishra 1996). These three pathotypes produced the low infection types on Thatcher seedlings with minor, but consistent differences (Table 1). These pathotypes were tested with 20 lines each of bread and durum wheats (Table 1), since the Thatcher-avirulent leaf rust isolates from Ethiopia and Morocco tended to be avirulent to bread wheats but virulent to durum wheats (Huerta-Espino and Roelfs 1992). Leaf rust pathotype 121R63-1 (77-5), carrying virulence to Thatcher and many of the known *Lr* genes, was also included for comparison. Seedlings of the test lines including suitable checks were evaluated at 18-27°C (temperatures mostly ranging between 20-25°C) using standard glasshouse procedures (Stakman et al. 1962). While the three Thatcher-avirulent pathotypes were generally avirulent to bread wheats, they displayed differential interaction with durum wheats (Table 1). In contrast, pathotype 121R63-1 (77-5) was virulent to all the bread wheat lines, but avirulent to most of the durum wheats (Table 1). Similar differences in the seedling response of durum wheat and bread wheat to the leaf rust races 77 and 106 have earlier been reported from India (Pandey and Rao 1984). These findings emphasize the need for separate protocols with regard to the choice of leaf rust pathotypes for evaluating leaf rust resistance in bread and durum wheats.

One wheat cultivar Kanred, a parental line of Thatcher, was speculated as the source of seedling resistance to the Thatcher-avirulent Ethiopian leaf rust isolates based on their comparison of the infection types (Mishra and Roelfs 1995). The source of seedling resistance in Thatcher to the three Thatcher-avirulent Indian leaf rust pathotypes is not known, and further studies are being conducted to explain it.

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Table 1. Seedling response of durum wheat lines and bread wheat lines to the three Thatcher-avirulent and one of the Thatcher-virulent leaf rust pathotypes from India

Wheat lines	Leaf rust pathotypes			
	Thatcher-avirulent			Thatcher-virulent
	0R8 (11)	0R8-1 (63)	0R9 (106)	121R63-1 (77-5)
Bread wheats				
Thatcher	0;	0;1	0;-	34
Agra Local‡	34	34	34	4
C 306	0;	0;	0;	4
DL 803-3	0;	0;-	0;	34
GW 173	0;	0;	0;	34
GW 273	0;	0;-	0;-	34
HD 2009	;1	;2	;2	34
HD 2135	;2	0;	;2	33+†
HD 2189	0;	0;-	0;	34
HI 1077	0;	0;-	0;	34
Jupateco 73 'S'	;1	0;-	0;	34
Kalyan Sona	0;	;1	0;	34
Lok 1	0;	0;	0;	34
Mukta	34	;2	34	34
Nainari 60	0;	0;	0;	34
Narmada 4	;1	0;	;1	34
NP 4	34	X+	X+	34
Pavon 76	0;	0;	0;	34
Sonalika	0;	0;	0;	34
Sujata	;1	0;	;1	4
WH 147	;2	4	X	34
Durum wheats				
Malvi Local§	33+	34	34	34
A 9-30-1	33+	0;	;1	X
A 206	33+	4	34	34
Bijaga Yellow	33+	X	34	23
GW 1139	X	;1	;2	;1
HD 4672	;2	0;	;1	;1
HG 110	33+	;1	34	;2
HI 8381	33+	;1	4	;1
HI 8498	;2	0;	;2	;1
ID 1128	34	X	34	X
ID 1169	34	X+	34	X+
Jairaj	;1	0;	;1	;2
Karnataka Local	33+	34	X, 34	34
Line 1172	23	;1	4	X
Malavraj	;1	0;	0;	;1
Meghdoot	33+	0;	34	X-
Motia	33+	34	34	34
NP 404	;1	0;	;2	;2
Raj 1555	X	0;	;1	;1
Sarangpur Local	;1	0;	;2	;2

†Infection types 33+, 34 and 4 indicate virulence of the pathotypes and susceptibility of the corresponding wheat lines, others indicate avirulence of the pathotypes and resistance of wheat lines (Stakman et al 1962)
‡Agra Local: Susceptible bread wheat check §Malvi local: Susceptible durum wheat check

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Research information

New germplasm of durum wheat with stem rust resistance

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Lack of stable resistance to stem rust is the major limiting factor to develop durum wheat cultivars with high yielding potential in India. One of the reasons for this instability seems to be resulted from a narrow genetic base for stem rust resistance in CIMMYT germplasm (Singh et al. 1992) used for durum wheat improvement in India. There are relatively few reports on stem rust resistance in durum wheats outside the CIMMYT germplasm (Mishra et al. 1989a, b; Pandey and Rao 1989; Hare 1997). Hence, it is important to identify new germplasm for breeding of durums with resistance to stem rust. The present communication

Table 1. Characteristics of the new germplasm of durum wheat with stem rust resistance

Genetic stock	Parentage / Source	Phenotypic traits
ED 2398 A	Ethiopian local variety from the germplasm collection at IARI-Indore	Tall in height (>110 cm), late in flowering, long ears with glabrous glumes, purple pigmentation on stem and auricle
HG 110	Sarangpur Local/HI 8185 (Sarangpur Local - local variety of durum wheat, HI 8185 – an advanced generation of durum wheat developed at IARI-Indore)	Medium tall (<110 cm), medium early flowering, long ears with pubescent glumes, purple pigmentation on auricle
B 662	PBW 34*2/Chuanmai #18 (PBW 34 – a durum wheat cultivar released in India, Chuanmai # 18 - a Chinese accession of *Triticum aestivum* carrying *Rht8* gene for dwarfism)	Triple dwarf (Height <50 cm), medium late flowering, long ears with pubescent glumes
IWP 5019	HD 4519*2/NP 200 (HD 4519 – an advanced generation of durum wheat developed at IARI, New Delhi, NP 200 – a *T. dicoccum* cultivar released in India	Double dwarf (Height 80-85 cm), very early flowering, glabrous glumes, grains with high protein content and high SDS value
Line 1172	MACS 9*2/*T. militinae* (MACS 9 – a durum wheat cultivar released in India, *T. militinae* – a free-thresing mutant of *T. timopheevii*)	Tall in height (>110 cm), medium late flowering, glabrous glumes, grains very bold

B 662, IWP 5019 and Line 1172 were developed at IARI, New Delhi through interspecific hybridization

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reports five new genetic stocks of durum wheat with stem rust resistance; ED 2398-A, HG 110, B 662, IWP 5019 and Line 1172 (Table 1). These lines showed high levels of field resistance to all of the important Indian stem rust pathotypes at Indore during 1997-2000 with artificial inoculation (Table 2). They also exhibit good levels of leaf rust resistance as well, excepting Line 1172 (Table 2).

Seedling response to 16 important Indian stem rust pathotypes were also evaluated along with suitable checks at 18-27°C (temperatures mostly ranging between 20-25°C) using standard glasshouse procedures (Stakman et al. 1962). While B 662 was resistant to all the test pathotypes, others showed differential interaction (Table 3). Thus, the resistance of B 662 is effective throughout the plant life, whereas

Table 2. Field response† of durum wheat lines to stem and leaf rusts during 1997-2000

Durum wheat line	Crop season 1997-1998		Crop season 1998-1999		Crop season 1999-2000	
	Stem rust	Leaf rust	Stem rust	Leaf rust	Stem rust	Leaf rust
ED 2398-A	Free	Free	Free	Free	Free	Free
HG 110	TMR	20MR	TMR	5MR	TMR	5MR
B 662	Free	5MR-TS	5MR	10MR	TR	10MR
IWP 5019	5MR-TS	TMR	5MR-TS	TR	10MR-TMS	TMR
Line 1172	Free-TS	TS-80S	TMR	TS	5MR	TS-40S
Motia‡	80S	100S	80S	80S	80S	100S
Agra Local§	80S	100S	80S	100S	80S	100S

†Field response was recorded combining disease severity i.e. percentage of host tissue infected as per the modified Cobb's scale (Peterson et al. 1948) with the reaction (Free : free from any visible signs of infection, R : resistant, MR : moderately resistant, MS : moderately susceptible, and S : susceptible)
‡Motia : Susceptible durum wheat check §Agra Local : Susceptible bread wheat check

Table 3. Seedling response of durum wheat lines to various stem rust pathotypes

Pathotype	Durum wheat lines						
	ED 2398 A	HG 110	B 662	IWP 5019	Line 1172	Motia‡	Agra Local§
11	12	;2	;1	12	;2	X+	4†
11A	12	;2	;1	X	;2	4	4
21-1	4	12	;1	12	23	4	4
24 A	;2	;2	;1	X	23	X	4
34-1	0;	;1	;1	;2	;1	X	4
40	X	;1	;1	X	;1	4	4
40 A	;1	;1	;1	;1	12	4	4
40-1	0;	0;	0;	0;	0;	4	4
42 B	;2	;1	;1	;3	;1	4	4
117-1	4	;2	;1	4	;2	4	4
117-3	;2	;2	0;	X	;2	4	4
117-5	12	;1	;1	4	;1	4	4
117-6	23	4	;1	4	4	4	4
117 A-1	;2	4	;1	4	4	4	4
122	;3	;1	;1	;3	;1	4	4
295	23	12	;1	X	12	4	4

†Infection type '4' indicates susceptibility, other infection types indicate resistance (after Stakman et al. 1962)
‡Motia : Susceptible durum wheat check §Agra Local : Susceptible bread wheat check

Table 4. Differences in seedling response of durum wheat lines to the 16 test pathotypes of stem rust

Durum wheat line	Spectrum of resistance to the 16 test pathotypes† of stem rust
ED 2398 A	Resistant to all the pathotypes except 21-1 and 117-1
HG 110‡	Resistant to all the pathotypes except 117-6 and 117A-1
B 662	Resistant to all the pathotypes
IWP 5019	Resistant to all the pathotypes except 117-1, 117-5, 117-6 and 117A-1
Line 1172‡	Resistant to all the pathotypes except 117-6 and 117A-1

†The 16 test pathotypes of stem rust are the same as listed in Table 3
‡HG 110 showed infection types (ITs) '12' and ';2', respectively, to the pathotypes 21-1 and 24A, while Line 1172 showed IT '23' to these pathotypes (Table 3)

part of the resistance component of the other lines is expressed only in adult plants (Tables 2 and 3). The stem rust resistance base of Indian durum wheats comprises mostly of *Sr9e* and few other genes like *Sr11* or *Sr7b* (Directorate of Wheat Research 1999). Presence of additional genes in the germplasm under report is apparent from their resistance to stem rust pathotypes 40A and 40-1, to which all the three aforesaid *Sr* genes are susceptible. Furthermore, these pathotypes are generally virulent to bread wheat cultivars too. Although nothing is known of the occurrence of any of the designated *Sr* genes in these lines, differences in their seedling response to various stem rust pathotypes indicate that they carry different genes for stem rust resistance (Table 4). Genetic studies are in progress to determine the number and allelic relationship of these genes.

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Research information

Appropriate pathotypes of stem rust and leaf rust for evaluating resistance in durum wheat and bread wheat

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Differential response of durum and bread wheats to selected cultures of leaf rust has been reported from India (Pandey and Rao 1984; Sharma et al. 1996). Hence, a study was conducted to assess the suitability of different leaf rust pathotypes for evaluating resistance in durum and bread wheats, and to find out whether the two wheat species respond differently to various cultures of stem rust also. The present communication reports identification of appropriate pathotypes of leaf rust and stem rust for evaluating resistance in durum and bread wheats, based on the above study involving seedling tests of a number of host genotypes with different rust cultures.

A total of 52 bread wheat and 50 durum wheat genotypes including recent and past cultivars released in India, genetic stocks and local varieties, were tested with 11 and 12 pathotypes of leaf rust and stem rust, respectively. These wheat genotypes and rust cultures were selected carefully to represent a cross section of the diversity existing in the wheat crop and the variability occurring in the rust populations in India. Seedlings of the test lines were evaluated at 18-27°C (temperatures mostly ranging between 20-25°C) using standard glasshouse procedures (Stakman et al. 1962). Bread wheat cultivar Agra Local, susceptible to all the Indian pathotypes of stem rust and leaf rust, served as check. Infection types 3, 3+ and 4 produced by a pathotype on a host line indicated virulence of the respective pathotype to that host line, whereas infection types 0; 1, 2 and X indicated avirulence. Percent virulence values were rounded off to the nearest whole number.

The currently prevalent pathotypes of leaf rust races 77 and 104 were more virulent to bread wheat lines, compared to durums, while other leaf rust pathotypes were relatively more virulent to durum wheats (Table 1). The leaf rust pathotype 77-5 was highly virulent to bread wheats (Table 1). In fact, only three bread wheat varieties, HI 1454, HP 1633 and HUW 468, showed resistance to this pathotype, and to all the other leaf rust pathotypes tested. However, the pathotype 77-5 was avirulent to most of the durum wheat lines. In contrast, the leaf rust races 106 and 108, presumed to be weak races due to their low levels of virulence to known leaf rust resistance

Table 1. Percent virulence of leaf rust pathotypes to bread wheat and durum wheat

Pathotype	Percent (%) virulence to	
	Bread wheat	Durum wheat
12-2	46†	58†
12-4	21	34
77-1	57	14
77-5	94	12
77-7	82	24
104-2	67	36
104B	24	20
106	02	25
107	10	14
108	16	30
162	18	25

†Percentages (% virulence values) rounded off to the nearest whole number.

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Table 2. Percent virulence of stem rust pathotypes to bread wheat and durum wheat†

Pathotype	Percent (%) virulence to	
	Bread wheat	Durum wheat
21-1	07	12
21A-2	04	06
34-1	02	04
40A	39	32
42B	14	30
117-3	17	45
117-4	17	42
117-5	24	57
117-6	16	85
117A-1	08	70
122	22	31
295	24	24

†Footnotes same as given in Table 1

genes, were more virulent to durums, compared to bread wheats (Table 1). These findings confirm the earlier observations on the differences in leaf rust resistance between durum and bread wheats. While durum wheats showed high levels of resistance to the leaf rust race 77-pathotypes (Honrao and Rao 1996, Nayar et al. 1996, Sharma et al. 1996, Pandey and Rao, 1984), the bread wheats were generally susceptible to them, particularly to the pathotype 77-5 (Sharma et al. 1996, Mishra unpubl). Thus, in addition to leaf rust pathotypes 12-2 and 104-2 which showed considerable virulence to both durum and bread wheats, the pathotypes 77-1, 77-5 and 77-7 should be used for evaluating resistance in bread wheat, and the pathotypes 12-4, 106, 108 and 162 need to be included for evaluating leaf rust resistance in durum wheats, based on the virulence frequencies of these pathotypes to the tested host lines (Table 1).

All the stem rust pathotypes, except 40A, showed greater degree of virulence to durums, compared to bread wheats. The race 117- pathotypes, particularly 117-6 and 117 A-1, were highly virulent to durum wheats and hence, are essential for evaluating stem rust resistance in durum wheat (Table 2). In fact, only four durum lines, AKDW 3347, B 662, GW 1139 and P 6046 showed resistance to all of the race 117-pathotypes, and to the other stem rust pathotypes as well. The pathotypes 40A, 117-5, 122 and 295 are appropriate for evaluating stem rust resistance in bread wheat, based on their virulence frequencies to the test lines (Table 2).

Further studies are in progress to develop definite protocols for systematic evaluation of resistance to stem and leaf rusts in durum and bread wheats.

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Wheat Information Service
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Gene symbol

CATALOGUE OF GENE SYMBOLS FOR WHEAT: 2001 Supplement

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10. Laboratory Designators for DNA markers

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Morphological, Physiological, Molecular and DNA Traits

To reference {0066} given in the first paragraph in the 2000 Supplement add {0109}.

Gross Morphology : Spike characteristics
1. Squarehead/spelt

Q. **ma :** Complete linkage with cDNA clone PtAq22 {0127}.

q. At end of section add : 'The speltoid phenotype of at least some spelts may be caused by genes at other loci {0140}.'.

2. Club/Compact spike
QTL: Six QTLs for spike compactness were detected in Courtot/Chinese Spring {0114} but only 4 on chromosome arms 1AL, 2BS, 2DS and 4AS were consistent for at least two years.

Aluminium Tolerance
Alt2. **v :** BH1146 {1213,0115}; IAC-24 }0115}; IAC-60 {0115}; 13 induced mutants of Anahuac {0115}.

Brittle Rachis

Br2 {0130}.	3A {0130}.	**Su:tv :**	LDN(DIC 3A) {0130}.
Br3 {0130}.	3B {0130}.	**Su:tv :**	LTN(DIC 3B) {0130}.

Evidence for a homoeologous series extending to many related species is discussed in {0130}.

Crossability with Rye, *Hordeum* and *Aegilops* spp.
1. Common wheat

QTL : 65% of the variability in a Courtot/CS population was associated with *Xfba367-5*A (5BS), *Xwg583-5B* (5BL) and *Xtam51-7A* {0134}. Only the second QTL appears to coincide with known locations of *Kr* genes.

DNA Markers
Group 1S
Amendments:

Xgwm273-1B. Add '(3A,B).' in the last column.
Xgwm550-1B. Add '(7A).' in the last column.
Xgwm582-1B. Add '(2A, 6B).' in the last column.

Add:

Xgdm33-1A {0173}.		DMS F33/DMS R33.	(1D).
Xgdm33-1D {0173}.		DMS F33/DMS R33.	(1A).
Xgdm60-1D {0173}.		DMS F60/DMS R60.	
Xgwm359-1B [{0170}].	[*Xgwm359b-1B* {0170}].	WMS F359/WMS R359.	(2A).
Xgwm911-1B {0171}.		WMS F911/WMS R911.	
Xmwg2245-1D {0135}.		MWG2245.	
Xwmc333-1A {0165}.		WMC 333F/WMC333R {0166}.	

Group 1L
Amendments:

Xfbb255-1A,B. Add '(5A,B).' in the last column.
Xgwm274-1B. Revise last column to '(5B, 7B). '.
Xgwm403-1B. Add '(2B).' in the last column.
Xgwm497-1A. Revise the last column to '(2A, 3A,D, 5B). '.

Add:

Xgdm19-1D {0173}.		DMS F19/DMS R19.	(2D).
Xgdm111-1D {0173}.		DMS F111/DMS R111.	
Xgdm126-1D {0173}.		DMS F126/DMS R126.	
Xgwm633-1A {0171}.		WMS F633/WMS R633.	

Group 1
Amendments:

Xabc155-1D. Add '(5A,B ,D).' in the last column.

Add:

Xgdm28-1B {0173}.		DMS F28/DMS R28.	(6A).
Xgwm376-1A [{0171}].	[*Xgwm376d-1A*i {0171}].	WMS F376/WMS R376.	(3B, 7A).
Xgwm408-1B [{0170}].	[*Xgwm408b-1B* {0170}].	WMS F408/WMS R408.	(5B).

Xgwm558-1A [{0171}].	[*Xgwm558c,d-1A* {0171}].	WMS F558/WMS R558.	(2A).
Xgwm903-1B {0171}.		WMS F903/WMS R903.	
Xgwm934-1B {0171}.		WMS F934/WMS R934.	
Xwmc44-1B {0153}.		WMC 44F/WMC 44R {0154}.	
Xwmc120-1A {0153}.		WMC 120F/WMC 120R {0155}.	

Group 2S
Amendments:

Xgwm55-2B. Revise the last column to '(3B, 6D). '.
Xgwm359-2A. Add '(1B).' in the last column.
Xgwm374-2B. Add '(3A).' in the last column.
Xgwm497-2A. Revise the last column to '(1A, 3A,D, 5B). '.
Xgwm630-2B. Add '(2A).' in the last column.
Xpsr131-2A,B,D. Revise the last column to '(5A,D).'.
Xpsr801(Rbcs)-2A,B,D. Add 'pW4.3 {139}' to the third column and add the following note : 'The development of probes specific to two of the three gene subfamilies on chromosome arms 2S and to the subfamily on chromosome arm 5L have been reported in {0149}.'.
Xrz395-2A. Revise the last column to '(5A,B ,D).'.

Add:

Xgdm5-2A {0173}.		DMS F5/DMS R5.	(2D).
Xgdm5-2D {0173}.		DMS F5/DMS R5.	(2A).
Xgdm19-2D {0173}.		DMS F19/DMS R19.	(1D).
Xgdm35-2D {0173}.		DMS F35/DMS R35.	
Xgdm107-2D {0173}.		DMS F107/DMS R107.	
Xgwm68-2A [{0171}].	[*Xgwm68a-2A* {0171}].	WMS F68/WMS R68.	(5B, 7B).
Xgwm297-2A [{0171}].	[*Xgwm297b-2A* {0171}].	WMS F297/WMS R297.	(4A, 7B).
Xgwm630-2A [{0171}].	[*Xgwm630c-2A* {0171}].	WMS F630/WMS R630.	(2B).

Group 2L
Amendments:

Xbcd266-2D. Revise the first column to ' *Xbcd266-2B* {0164}, *2D* {864}.'.
Xgwm558-2A. Add '(1A).' in the last column.

Add:

Xgdm6-2D {0173}.		DMS F6/DMS R6.	
Xgdm87-2B {0173}.		DMS F87/DMS R87.	(2D).
Xgdm87-2D {0173}.		DMS F87/DMS R87.	(2B).
Xgdm93-2A {0173}.		DMS F93/DMS R93.	(2D, 4B).
Xgdm93-2D {0173}.		DMS F93/DMS R93.	(2A, 4B).
Xgdm114-2B {0173}.		DMS F114/DMS R114.	
Xgwm88-2B [{0171}].	[*Xgwm88b-2B* {0171}].	WMS F88/WMS R88.	(6B).

Group 2
Add:

Xgdm29-2D {0173}.		DMS F29/DMS R29.	
Xgdm77-2D {0173}.		DMS F77/DMS R77.	
Xgdm86-2B {0173}.		DMS F86/DMS R86.	(7D).
Xgdm124-2B {0173}.		DMS F124/DMS R124.	
Xgdm148-2D {0173}.		DMS F148/DMS R148.	
Xgwm403-2B [{0171}].	[*Xgwm403b-2B* {0171}].	WMS F403/WMS R403.	(1B).
Xgwm582-2A [{0171}].	[*Xgwm582-2A* {0171}].	WMS F582/WMS R582.	(1B, 6B).
Xwmc24-2A {0153}.		WMC 24F/WMC 24R {0162}.	
Xwmc25-2D {0153}.		WMC 25F/WMC 25R {0162}.	
Xwmc149-2B {0153}.		WMC 149F/WMC 149R {0156}.	

Xwmc167-2D {0153}.		WMC 167F/WMC 167R {0157}.	
Xwmc170-2A {0153}.		WMC 170F/WMC 170R {0157}.	
Xwmc245-2D {0153}.		WMC 245F/ WMC 245R {0159}.	

Group 3S
Amendments:

Xfbb166-3B. Add '(5B).' in the last column.
Xgwm376-3B. Add '(1A, 7A).' in the last column.
XksuF34-3D. Revise the first column to ' *XksuF34-3B* {0152}[2], *-3D* [{448}][4], {233}[1].'

Add:

Xgdm72-3D {0173}.		DMS F72/DMS R72.	
Xgwm107-3B [{0171}].	[*Xgwm107a-3B* {0171}].	WMS F107/WMS R107.	(4B, 6B).
Xgwm374-3A [{0171}].	[*Xgwm374b-3A* {0171}].	WMS F374/WMS R374.	(2B).
XksuD30-3B {0152}.		pTtksuD30.	(5A,B,D).

Group 3L
Amendments:

Xabc172-3A.1,.2. Revise the first column to ' *Xabc172-3A.1,.2* {1061}*,3D* {0173}.'.
Xfbb237-3A,D. Revise the last column to '(5B,D).'.
Xgwm108-3B. Add '(6B).' in the last column.
Xgwm247-3B. Add '(3A).' in the last column.
Xgwm340-3B. Add '(3A).' in the last column.

Add:

Xgdm8-3D {0173}.		DMS F8/DMS R8.	
Xgdm38-3D {0173}.		DMS F38/DMS R38.	
Xgdm128-3D {0173}.		DMS F128/DMS R128.	
Xgdm134-3A {0173}.		DMS F134/DMS R134.	
Xgwm55-3B [{0171}].	[*Xgwm55c-3B* {0171}].	WMS F55/WMS R55.	(2B, 6D).
Xgwm113-3B [{0171}].	[*Xgwm113b-3B* {0171}].	WMS F113/WMS R113.	(4B).
Xgwm247-3A [{0171}].	[*Xgwm247b-3A* {0171}].	WMS F247/WMS R247.	(3B).
Xgwm273-3B [{0171}].	[*Xgwm273c-3B* {0171}].	WMS F273/WMS R273.	(1B, 3A).
Xgwm340-3A [{0171}].	[*Xgwm340b-3A* {0171}].	WMS F340/WMS R340.	(3B).
Xgwm497-3A [{0171}].	[*Xgwm497c-3A* {0171}].	WMS F497/WMS R497.	(1A, 2A, 3D, 5B).

Group 3
Amendments:

Xgwm497-3D. Revise the last column to '(1A, 2A, 3B, 5B). '.

Add:

Xgdm62-3D {0173}.		DMS F62/DMS R62.	
Xgdm64-3B {0173}.		DMS F64/DMS R64.	
Xgdm120-3B {0173}.		DMS F120/DMS R120.	
Xgwm273-3A [{0171}].	[*Xgwm273b-3A* {0171}].	WMS F273/WMS R273.	(1B, 3B).
Xwmc169-3A {0153}.		WMC 169F/ WMC 169R {0157}.	

Group 4S (4AL :4BS:4DS)
Add:

Xgdm129-4D {0173}.	DMS F129/DMS R129.

Group 4L (4AS :4BL:4DL)
Amendments:

Xgwm251-4B. Add '(7A).' in the last column.
Xgwm601-4A. Add '(7A).' in the last column.

Add:

Xgdm93-4B {0173}.	DMS F93/DMS R93.	(2A,D).
Xgdm125-4D {0173}.	DMS F125/DMS R125.	

It is not clear whether *Xgdm125-4D* belongs to the group 4L or to the group 5AL :5BL:4DL.

Xgwm297-4A [{0171}]. [*Xgwm297c-4A* {0171}].	WMS F297/WMS R297.	(2A, 7B).
Xgwm663-4A {0171}.	WMS F663/WMS R663.	

Group 5AL:4BL:4DL
Amendments:

Xcdo1333-4B,D. Revise the first column to ' *Xcdo1333-5A* {255,282}[3], {0148}[1], *4B,D* {1008}.' and remove '(5A).' from the last column.
Xfbb255-4B. Add '(5A,B).' in the last column.
Xgwm126-5A. Add '(6B).' in the last column.
Xkvl920(OxoLP)-4D. Revise the first column to '*Xkvl920(OxoLP)-5A* [{0148}], *4D* [{0091}].' and revise the second column to '[*Oxo1-5A* {0148}, *4D* {0091}].'.

Add:

Xycu518-5A,4B,4D {0186}.	pTaQ18.
Xycu524-5A,4B,4D {0186}.	pTaQ24.

Group 4
Amendments:

Xgwm107-4B. Add '(3B, 6B).' in the last column.
Xgwm113-4B. Add '(3B).' in the last column.

Add:

Xgdm34-4D {0173}.	DMS F34/DMS R34.	
Xgdm40-4D {0173}.	DMS F40/DMS R40.	
Xgdm61-4D {0173}.	DMS F61/DMS R61.	
Xgdm88-4A {0173}.	DMS F88/DMS R88.	
Xgdm133-4D {0173}.	DMS F133/DMS R133.	(5B).
Xgdm145-4A {0173}.	DMS F145/DMS R145.	
Xwmc35-4B {0153}.	WMC 35F/WMC 35R {0162}.	
Xwmc254-4B {0153}.	WMC 254F/WMC 254R.	

Group 5S
Amendments:

Xgwm293-5A. Add '(5B).' in the last column.
Xmgb191-5A. Add '(5AL, 5BL, 5DL).' in the last column.
Xmgb341-5A. Add '(5BL,5DL).' in the last column.

Add:

Xgdm109-5A {0173}.	DMS F109/DMS R109.
Xgwm415-5A {9929, 0178}.	WMS415F/WMS415R.
Xutv711-5A {0152}.	UTV711.
Xutv1441-5A {0152}.	UTV1441.

Group 5L
Amendments:

Xabg391-5A,D. Revise the first column to ' *Xabg391-5A* {1059}[1], *5B* {0148}[1], *5D* {9926}[4], {0148}[1].'
Xabg473-5A,B. Revise the first column to ' *Xabg473-5A* {9933}[1,3], *5B* {1059}[1], *5D* {0148}[1].'.

Xbcd9-5A,B. Revise the first column to ' *Xbcd9-5A* {0282}[3], *5A.1,.2* [{0148}][1], *5B* {1059}[1], *5D* {0148}[1].' and add '[*Xbcd9a,b-5A* {0148}].' in the second column.
Xbcd21-5A. Revise the first column to ' *Xbcd21-5A* {9933}, *5D* {0148}.'.
Xbcd183-5A. Revise the first column to ' *Xbcd183-5A* {1059}, *5B,D* {0148}.'.
Xbcd508-5A,B,D. Revise the first column to ' *Xbcd508-5A* {255,282}[3], {0148}[1], *5B* {1059}[1], *5D.1,.2* {446}.'.
Xbcd1030-5B. Revise the first column to ' *Xbcd1030-5A* {0148}, *5B* {1059,0148}, *5D* {0148}.'.
Xbcd1235-5A. Revise the first column to ' *Xbcd1235-5A.1,.2* {1059}, *5B, 5D.1,.2* {0148}.'.
Xcdo388-5A,D. Add as a note: 'Two loci were detected for *Xcdo388-5A* and *Xcdo388-5D* in {0148}.
Xcdo457-5A. Revise the first column to ' *Xcdo457-5A* {1059}, *5B,D* {0148}.'.
Xcdo465-5A. Revise the first column to ' *Xcdo465-5A* {282}[3], {0148}[1], *5B,D* {0148}[1].'.
Xcdo548-5A. Revise the first column to ' *Xcdo548-5A* {9933}[1,3], *5A.1,.2* [{0148}][1], *5B* {0148}[1], *5D.1, .2* [{0148}][1].' and add '[*Xcdo548a,b-5A,D* {0148}].' in the second column.
Xcdo584-5A,B. Revise the first column to ' *Xcdo584-5A* {0068}, *5B* {1059}, *5D* {0148}.'.
Xcdo1312-5A,B,D. Revise the first column to ' *Xcdo1312-5A* {255}[3], {0148}[1], *4B,D* {1059}[{028}][1].
Xcdo1326-5A,B. Revise the first column to ' *Xcdo1326-5A,B* {1059}, *5D* {0148}.'.
Xcdo1333-5A. Delete (moved to 5AL:4BL:4DL).
Xfba68-5A. Revise the first column to ' *Xfba68-5A* {1059}, *5B,D* {0148}.'.
Xfba351-5A,B. Revise the first column to ' *Xfba351-5A,B* {1059}, *5D* {0148}.'.
Xfbb255-5A. Revise the first column to ' *Xfbb255-5A* {1059}, *5A.1, .2* [{0148}], *5D* {0148}.', add '[*Xfbb255a,b-5A* {0148}].' in the second column, and add '(1A,B, 4B, 6A).' in the last column.
Xfbb237-5B. Revise the first column to ' *Xfbb237-5B* {1059}, *5D* {0148}.'.
Xgwm68-5B. Revise the last column to '(2A, 7B). '.
Xgwm408-5B. Add '(1B).' in the last column.
Xksu919(Lpx)-5A,B. Revise the first column to ' *Xksu919(Lpx)-5A,B* [{0091}], *5D* [{0148}].' and revise the second column to '[*Lpx-5A,B* {0091}, *5D* {0148}].'.
Xksu923(Pr1)-5D. Revise the first column to '*Xksu923(Pr1)-5A,B* [{0148}], *5D* [{0091}].' and revise the second column to '[*Pr1-5A,B* {0148}, *5D* {0091}].'.
XksuD30-5A,B,D. Add '(3B).' in the last column.
XksuG14-5A,B,D. Revise the first column to ' *XksuG14-5A* {282}[3], {0148}[1], *5B,D* {446}[1].'.
XksuH1-5A. Revise the first column to ' *XksuH1-5A* {860}, *5D* {0148}.' and remove '(5D)' from the last column.
Xmgb63-5A. Revise the first column to ' *Xmgb63-5A* {9959}[2], {0148}[1], *B,D* {0148}[1].'.
Xmwg522-5A,B,D. Revise the first column to ' *Xmwg522-5A* {1059}[1], *5B* {446}[1], *5D* {9926}[4], {0148}[1].'.
Xmwg602-5A,D. Revise the first column to ' *Xmwg602-5A* {446}, *5B* {0148}, *5D* {446}.'.
Xmwg900-5D. Revise the first column to ' *Xmwg900-5A,B* {0148}, *5D* {1059}.'.
Xmwg922-5D. Revise the first column to ' *Xmwg922-5A,B* {0148}, *5D* {1059}.'.
Xpsr801(Rbcs)-5A,B,D. Add 'pW4.3 {139}' to the third column and add the following note : 'The development of probes specific to two of the three gene subfamilies on chromosome arms 2S and to the subfamily on chromosome arm 5L have been reported in {0149}.'.
Xrz395-5A,D. Revise the first column to ' *Xrz395-5A* {1059}, *5B* {0148}, *5D* {1059}.'.
Xwg889-5A,B,D. Revise the first column to ' *Xwg889-5A* {255,282}[3], {0148}[1],*5B* {1059}[1], *5D* {446}[1].'.
Xwg908-5A,B,D. Revise the first column to ' *Xwg908-5A* {255,282}[3], {0148}[1], *5B.1,.2*, *5D* {446}[1].'.

Add:

Xabc155-5A,B,D {0148}.		ABC155	(1D).
Xabc168-5A,B,D {0148}.		ABC168.	
Xbcd21-5A,D {0148}.		BCD21.	
Xbcd307-5B.1,.2 [{0148}].	[*Xbcd307a,b-5B* {0148}].	BCD307.	
Xbcd881-5A,B,D {0148}.		BCD881.	
Xbcd1427-5A,B,D {0148}.		BCD1427.	
Xbcd1734-5A,B,D {0148}.		BCD1734.	
Xcdo87-5A,B,D {0148}.		CDO87.	
Xcdo385-5A,B,D {0148}.		CDO385.	
Xcdo1475-5A,B {0148}.		CDO1475.	
Xfbb166-5B {0148}.		FBB166.	(3B, 6A).
Xgdm3-5D {0173}.		DMS F3/DMS R3.	
Xgdm43-5D {0173}.		DMS F43/DMS R43.	
Xgdm63-5D {0173}.		DMS F63/DMS R63.	

It is not clear whether *Xgdm63-5D* belongs to the group 5L or to the group 4AL:5BL:5DL.

Xgdm68-5D {0173}.		DMS F68/DMS R68.	(5A,B).

Xgdm99-5D {0173}.		DMS F99/DMS R99.	
Xgdm116-5D {0173}.		DMS F116/DMS R116.	
Xgdm138-5D {0173}.		DMS F138/DMS R138.	
Xgdm153-5D {0173}.		DMS F153/DMS R153.	
Xgwm497-5B [{0171}].	[*Xgwm497d-5B* {0171}].	WMS F497/WMS R497.	(1A, 2A, 3A,D).
XksuG7-5A {446}[1], *5D.1,.2* [{448}][4], {0148}[1].	[*XksuG7(A),(B)-5D* {448}].	pTtksuG7.	(7A,B,D).
XksuG57-5D {0148}.		pTtksuG57.	(2D).
XksuP6-5A {0148}.		pTtksuP6.	
XksuP10-5A {0148}.		pTtksuP10.	
XksuP18-5A,B,D {0148}.		pTtksuP18.	
XksuP20-5A,B,D {0148}.		pTtksuP20.	
XksuP21-5A {0148}.		pTtksuP21.	
XksuP23-5A,D {0148}.		pTtksuP23.	
XksuP50-5A,B,D {0148}.		pTtksuP50.	
XksuP64-5A,B,D {0148}.		pTtksuP64.	
XksuQ10-5A,B,D {0148}.		pTtksuQ10.	
XksuQ11-5A,B,D {0148}.		pTtksuQ11.	
XksuQ13-5B,D {0148}.		pTtksuQ13.	
XksuQ16-5A,B,D {0148}.		pTtksuQ16.	
XksuQ24-5A,D {0148}.		pTtksuQ24.	
XksuQ32-5A,B,D {0148}.		pTtksuQ32.	
XksuQ34-5A,B,D {0148}.		pTtksuQ34.	
XksuQ35-5A,B,D {0148}.		pTtksuQ35.	
XksuQ45-5A.1,.2, B.1,.2, D [{0148}].	[*XksuQ45a,b-5A,B* {0148}].	pTtksuQ45.	
XksuQ58-5A,B,D {0148}.		pTtksuQ58.	
XksuQ59-5A,B,D {0148}.		pTtksuQ59.	
XksuQ60-5A,B,D {0148}.		pTtksuQ60.	
XksuQ61-5A,B,D {0148}.		pTtksuQ61.	
XksuQ62-5A {0148}.		pTtksuQ62.	
XksuQ63-5B,D {0148}.		pTtksuQ63.	
XksuQ64-5B,D {0148}.		pTtksuQ64.	
XksuQ65-5B,D {0148}.		pTtksuQ65.	
XksuQ66-5B {0148}.		pTtksuQ66.	
XksuQ67-5B {0148}.		pTtksuQ67.	
Xkvl930(Pr1)-5A,D [{0148}].	[*Pr1b-5A,D* {0148}].	HvPr1b {00104}.	(7B,D).
Xmgb1-5A,D {0148}.		MGB1.	
Xmgb8-5A {0148}.		MGB8.	
Xmgb10-5B {0148}.		MGB10.	
Xmgb174-5A,B,D {0148}.		MGB174.	
Xmgb191-5A,B,D {0148}.		MGB191.	(5AS).
Xmgb301-5A,B,D {0148}.		MGB301.	
Xmgb341-5B,D {0148}.		MGB341.	(5AS).
Xmwg72-5A,B {0148}.		MWG72.	
Xmwg76-5A,B,D {0148}.		MWG76.	
Xmwg514-5A,B,D {0148}.		MWG514.	(6A,D).
Xmwg549-5A,D {0148}.		MWG549.	(4A, 6D).
Xmwg550-5A,B,D {0148}.		MWG550.	
Xmwg516-5A,B,D {0148}.		MWG516.	
Xmwg604-5A,B,D {0148}.		MWG604.	
Xmwg716-5A,B,D {0148}.		MWG716.	(6D).
Xmwg740-5A,B,D {0148}.		MWG740.	
Xmwg805-5A,B,D {0148}.		MWG805.	

Xmwg862-5B,D {0148}.		MWG862.	
Xmwg933-5B {0148}.		MWG933.	
Xpsr131-5A {0148}[1], *5D* {9926}[4], {0148}[1].		PSR131.	(2A,B,D).
Xrz328-5A,B,D {0148}.		RZ328.	
Xrz575-5A,B,D {0148}.		RZ575.	
Xrz589-5A,B,D {0148}.		RZ589.	
Xrz744-5A,B,D {0148}.		RZ744.	
Xubp25-5B,D {0148}.		UBP25.	

4AL:5BL:5DL
Amendments:

Xmwg549-4A. Add '(5A,D).' in the last column.

Add:

Xgdm118-5D {0173}.		DMS F118/DMS R118.	
It is not clear whether *Xgdm118-5D* belongs to the group 4AL :5AL:5DL or to the group 7BS:5BL:5DL.			
Xgwm577-4A [{0177}].	[*Xgwm577b-4A* {0171}].	WMS F577/ WMS R577.	(7B).
It is not clear whether *Xgwm577-4A* belongs to the group 4AL :5AL:5BL or to the group 7AS:4AL:7DS.			

7BS:5BL:5DL
Amendments:

XksuG7-5A, D.1,.2. Delete (moved to 5L).

Group 5
Amendments:

XksuG57-5D.1,.2,.3 . Revise the last column to '(2D, 5DL).
XksuH1-5D. Revise the last column to '(5A,D)'.
Xpsr131-5D. Delete (moved to 5L).

Add:

XgbxG103-5D[{0146}].	[*XgbxG103b-5D* {0146}].	gbxG103.	
Xgdm68-5A {0173}.		DMS F68/DMS R68.	(5B,D).
Xgdm68-5B {{0173}.		DMS F68/DMS R68.	(5A,D).
Xgdm101-5B {0173}.		DMS F101/DMS R101.	
Xgdm109-5A {0173}.		DMS F109/DMS R109.	
Xgdm115-5B {0173}.		DMS F115/DMS R115.	(5D).
Xgdm115-5D {0173}.		DMS F115/DMS R115.	(5B).
Xgdm133-5B {0173}.		DMS F133/DMS R133.	(4D).
Xgdm136-5D {0173}.		DMS F136/DMS R136.	
Xgdm146-5B {0173}.		DMS F146/DMS R146.	
Xgdm149-5B {0173}.		DMS F149/DMS R149.	
Xgwm274-5B [{0171}].	[*Xgwm274b-5B* {0171}].	WMS F274/WMS R274.	(1B, 7B).
Xgwm293-5B [{0171}].	[*Xgwm293b-5B* {0171}].	WMS F293/WMS R293.	(5A).
Xgwm494-5D [{0146}].	[*Xgwm494a-5D* {0146}].	WMS F494/WMS R494.	(6A).
Xwmc267-5A {0153}.		WMC 267F/WMC 267R {0160}.	

Group 6S
Amendments:

Xbcd21-6A,B,D. Revise the last column to '(5A,D).'.
Xfbb166-6A. Add '(5B).' in the last column.
Xfbb255-6A. Add '(5A,B).' to the last column.
Xgwm88-6B. Add '(2B).' in the last column.
Xgwm361-6B. Add '(7BS, 7BL).' in the last column.
Xmwg549-6D. Add '(5A,D).' to the last column.

Add:

Xgdm14-6D {0173}.		DMS F14/DMS R14.	
Xgdm36-6D {0173}.		DMS F36/DMS R36.	
Xgdm108-6D {0173}.		DMS F108/DMS R108.	
Xgdm113-6B {0173}.		DMS F113/DMS R113.	
Xgdm127-6D {0173}.		DMS F127/DMS R127.	
Xgdm132-6D {0173}.		DMS F132/DMS R132.	
Xgdm141-6D {0173}.		DMS F141/DMS R141.	
Xgwm107-6B [{0171}].	[*Xgwm107c-6B* {0171}].	WMS F107/WMS R107.	(3B, 4B).

Group 6L
Amendments:

Xabg473-6B. Revise the last column to '(5A,B ,D)'.
Xfbb364-6A,B,D. Revise the first column to *'Xfbb364-6B* {900}, *6A,6D* {0081}.'.
Xmwg514-6A,D. Add '(5A,B,D).' in the last column.
Xcmwg716-6D. Add '(5A,D).' in the last column.

Add:

Xcmwg664-6B,D {0081}.		cMWG664.	(6A).
Xfbb95-6A,B,D {0081}.		FBB95.	
Xgdm28-6A {0173}.		DMS F28/DMS R28.	(1B).
Xgdm98-6D {0173}.		DMS F98/DMS R98.	
Xgdm147-6B {0173}.		DMS F147/DMS R147.	
Xgwm108-6B [{0171}].	[*Xgwm108a-6B* {0171}].	WMS F108/WMS R108.	(3B).
Xgwm126-6B [{0171}].	[*Xgwm126b-6B* {0171}].	WMS F126/WMS R126.	(5A).
Xgwm494-6A.2 [{0146}].	[*Xgwm494b-6A* {0146}].	WMS F494/WMS R494.	(5D, 6A).
Xgwm582-6B [{0171}].	[*Xgwm582a-6B* {0171}].	WMS F582/WMS R582.	(1B, 2A).

Group 6
Amendments:

Xbcd9-6D. Revise the last column to '(5A,B ,D).'.
Xcmwg664-6A. Add '(6B,D)'. in the last column.
Xgwm55-6D. Revise the last column to '(2B, 3B). '.
Xgwm494-6A. Revise the first column to ' *Xgwm494-6A.1* [{9929}].', add '[*Xgwm494-6A* {9929}].' in the second column, and add '(5D, 6AL).' in the last column.

Add:

Xgdm153-6B {0173}.		DMS F153/DMS R153.	
Xwmc76-6B {0153}.		WMC 76F/WMC 76R {0161}.	
Xwmc256-6A {0153}.		WMC 256F/WMC 256R.	(6D).
Xwmc256-6D {0153}.		WMC 256F/WMC 256R.	(6A).

Group 7S
Amendments:

Xgwm68-7B. Revise the last column to '(2A, 5B). '.
Xgwm297-7B. Add '(2A, 4A).' in the last column.
Xkvl930(Pr1)-7B,D. Add '(5A,D).' in the last column.

Add:

Xgwm251-7A [{0171}].	[*Xgwm251b-7A* {0171}].	WMS F251/WMS R251.	(4B).
Xgwm361-7B [{0171}].	[*Xgwm361b-7B* {0171}].	WMS F361/WMS R361.	(6B, 7BL).
Xgwm631-7A. {0178}.		WMS F631/WMS R631.	

7AS:4AL:7DS
Amendments:

Xksu919(Lpx)-4A. Revise the last column to '(5A,B ,D).'.

Add:

Xgdm86-7D {0173}.		DMS F86/DMS R86.	(2B).
Xgdm130-7D {0173}.		DMS F130/DMS R130.	

Group 7L
Amendments:

XksuG7-7A,B,D. Remove '5B' from the last column.
Xgwm274-7B. Revise last column to '(1B, 5B). '.
Xgwm577-7B. Add '(4A).' in the last column.

Add:

Xgdm46-7D {0173}.		DMS F46/DMS R46.	
Xgdm67-7D {0173}.		DMS F67/DMS R67.	
Xgdm84-7D {0173}.		DMS F84/DMS R84.	
Xgdm150-7D {0173}.		DMS F150/DMS R150.	
Xgwm361-7B [{0171}].	[*Xgwm361a-7B* {0171}].	WMS F361/WMS R361.	(6B, 7BS).
Xgwm550-7A [{0171}].	[*Xgwm550b-7A* {0171}].	WMS F550/WMS R550.	(1B).
XksuH1-7B {0152}.		pTtksuH1.	(5A,D).

Group 7
Add:

Xgdm142-7D {0173}.		DMS F142/DMS R142.	
Xgdm152-7A {0173}.		DMS F152/DMS R152.	
Xgwm376-7A [{0171}].	[*Xgwm376c-7A* {0171}].	WMS F376/WMS R376.	(1A, 3B).
Xgwm601-7A [{0171}].	[*Xgwm601b-7A* {0171}].	WMS F601/WMS R601.	(4A).
Xwmc47-7A {0153}.		WMC 47F/WMC 47R {0154}.	
Xwmc83-7A {0153}.		WMC 83F/WMC 83R {0161}.	
Xwmc216-7B {0153}.		WMC 216F/WMC216R {0158}.	

Dormancy (seed)
Add at the end of the section:
'Several QTL for falling number and _-amylase activity, two indicators for pre-harvest sprouting resistance, were identified in {0169}. The most significant were associated with *Xglk699-2A* and *Xsfr4(NBS)-2A* , *Xglk80-3A* and *Xpsr1054-3A*, *Xpsr1194-5A* and *Xpsr918-5A*, *Xpsr644-5A* and *Xpsr945-5A*, *Xpsr8(Cxp3)-6A* and *Xpsr563-6A*, and *Xpsr350-7B* and *Xbzh232(Tha)-7B* [{0169}]. '

Earliness per se
EpsWi. Replace the current v: listing with: ' **su:** Cheyenne*7/ Wichita 3A {0025}'. **ma:** Linked to QTLs for plant height, kernel number per spike, and 1,000-kernel weight in RSLs derived from CNN/CNN(WI3A) {0025}.
QTL : Analysis in Courtot/CS {0132}.

Gametocidal Genes
1. Gametocidal activity

Gc1-C1	{0188}.	2CL {0189}.	**ad:**	CS/2C {0189}.
			su:	CS2C(2A), CS2C(2B), CS2C(2D) {0189}.

Insert after the first paragraph:
'Gametocidal genes in chromosomes in the same homoeologous group have the same gametocidal action {0190}. In monosomic additions of chromosomes with gametocidal effects, chromosome deletions and translocations are produced in gametes not having the gametocidal genes. This feature has been exploited to isolate genetic stocks suitable for physical mapping of wheat {0191} chromosomes, and of rye {0192} and barley {0193,0194,0195} chromosomes in a wheat background.'.

Glaucousness
Add at the end of the section:

'A gene for spike glaucousness, *Ws*, was mapped distally on the short arm of chromosome 1B in the cross *T. durum* cv. Langdon x *T. dicoccoides* acc. Hermon H52 {0171}.'

Epistatic inhibitors of glaucousness
Insert prior to last sentence : 'A non-glaucous spike phenotype present in line L-592, a 7S(7A) substition line, is described in {0113}.'.

Grain Hardness / Endosperm Texture
To be inserted in a reorganised section :
'QTL : In a DH population of Courtot/CS a major locus in chromosome 5DS coincided with *Ha* ; minor QTLs mapped in chromosomes 1A (associated with *Xfba92*) and 6D (associated with *Xgwm55*) {0141}.'.

Grain Quality Parameters
1. Sedimentation value

2. Flour colour
Transfer from previous location following DNA section (1999 Supplement) and replace section with:
'QTL: A QTL was detected on chromosome 7A {9936}. Cultivar Schomburgk contributed the yellow colour allele in a cross Schomburgk/Yarralinka {9936}. Markers *Xcdo347-7A* and *Xwg232-7A* accounted for 60% of the genetic variation {9936}. A Sequence Tagged Site PCR marker is available {0180}. Other references to flour colour are given under *Lr19* and *Sr25.*'.

3. Amylose content
Replace previous section : Amylose content has a significant effect on industrial quality ; for example, reduced amylose wheats have better performance in some types of noodles. The waxy protein genes have an important influence, but other genes are also involved.
QTL : *QAmc-ocs.4A.1*was located in a *Xbcd1738 – Xcdo1387* segment in chromosome 4AS of Kanto 107 relative to CS {0047}.

4. Milling yield
QTL: A QTL was detected on chromosome 3A {0181}. Cultivar Schomburgk contributed the higher milling yield allele in a cross Schomburgk/Yarralinka {0181}. RFLP markers *Xbcd115* and *Xpsr754* were associated with this QTL at LOD>3 {0181.}

5. Alveograph dough strength W
QTL: QTLs for W were detected on chromosome arms 5DS (associated with *Xmta10*), 1AS (associated with *Xfba92*), and 3B (associated with *XksuE3*) in a cross Courtot/Chinese Spring {0141}. The first two QTLs coincided with those for hardness.

Grain Weight
QTL : Variation at locus *QGw1.ccsu-1A*, associated with *Xwmc333-1A*, accounted for 15% of the variation in a RIL population from RS111/CS {0143}.

Height
QTL: Two QTLs for plant height were assigned to chromosome 3A in RSLs from Cheyenne*7/Wichita 3A substitution line {0025}.
Seven QTLs on chromosomes 1A, 1D, 2B, 2D, & 4B affected plant height among RILs of CS/*T. spelta duhamelianum*. Effects linked with the CS alleles of *Xbcd1160-1A, Xksu127-1D,* and *XksuF11-2D* increased height whereas those CS alleles associated with*Xpsr131-2B, Xpsr135-2B, Xpsr934-2D* and *Xcs22.2-4B* reduced it {0196}.

Hybrid Weakness
1. Hybrid necrosis
Add to genotype list : {0112}.

Meiotic Characters
2. Pairing homoeologous

Ph1b. **ma :** A PCR-based detection system for *ph1bph1b* individuals is described in {9965}.

Proteins
1. Grain Protein Content

QTLs for grain protein content were detected on chromosome arms 6AS (associated AFLP marker, *XE38M60$_{200}$*) and 1BL (associated RFLP marker, *Xcdo1188*) in Courtot/Chinese Spring {0141}.

QGpc.ndsu-6Bb **v:** Glupro {0179}. **ma :** Flanking microsatellite markers and PCR-specific markers are available {0179}.

3. Endosperm Storage Proteins

3.1 Glutenins

Add below *Glu-A1-1c* in the *Glu-A1-1* section:
'A PCR marker specific for the *Glu-A1-1c* (Ax2*) allele was developed in {0147}.'

Add below *Glu-B1-1a* in the *Glu-B1-1* section:
'A PCR marker (2373 bp) for the *Glu-D1-1a* (Bx7) allele was developed in {0145}.'

Add below *Glu-D1-1d* in the *Glu-D1-1* section:
'PCR markers specific for the *Glu-D1-1d* (Dx5) allele were developed in {0145} and {0147}.'

Add below *Glu-D1-2a* in the *Glu-D1-2* section:
'A PCR marker (612 bp) for the *Glu-D1-2a* (Dy12) allele was developed in {0145}.'

Add below *Glu-D1-2b* in the *Glu-D1-2* section:
'PCR markers (576 bp and 2176) for the *Glu-D1-2b* (Dy10) allele were developed in {0145} and {0147}, respectively.'

Between the *Glu-A1* allelic list and the text beginning 'There is a possibility that *Glu-A1* alleles *i*, *j* {1527} and *k* {478} correspondto alleles...', insert the paragraph:
'The importance of the HMW glutenin subunits for bread-making quality was first noted from observations in wheat cultivars of related pedigree on the effects of the presence of subunit 1 encoded by *Glu-A1a* {0197}, effects that have repeatedlybeen confirmedsince (for example {0198,0199,01100}).'

After the end of the paragraph that closes the *Glu-3* (LMW glutenin) section ('The *Glu-3* loci can be recognised with pTag544 {49} and pTdUCD1 {167} and by specific microsatellite primers {252}.'), addthe paragraph:
'PCR amplification of genomic DNA has been used to isolate three LMW glutenin genes in cultivar Chinese Spring, named LMWG-MB1, LMWG-MB2 and LMWG-MB3 {01101}. The deduced amino-acid sequences showed a high similarity between these ORFs and with those of other LMW glutenin genes. The authors state that the study provided direct evidence that insertions and/or deletions provide a mechanistic explanation for the allelic variation, and hence the resultant evolution, of prolamin genes, and comment on relationships with γ-secalins and β-hordein families. Single-base substitutions at identical sites generate premature stop codons in both LMWG-MB2 and LMWG-MB3, indicating that these clones are pseudogenes.'

3.2. Gliadins

Before the final paragraph of the preamble (which now reads, after an amendment included in the 1999 Supplement: 'The *Gli-1* loci may be recognised by probes pcP387 {372} and pTag1436 {065}, and by specific microsatellite primers {252}. Furthermore, it has been shown that probe pTag1436 differentiates gliadin alleles rather well; using this probe, families of gliadin alleles and some of their relationships have been described {9988}.') insert the paragraph:
'Based upon morphological observation and RFLP analysis, it has been proposed that the cultivar 'Chinese Spring' is a strain of the landrace 'Chengdu-guangtou' from the Chengdu Plain, Sichuan Province; this proposal is supported by the observation that the cultivar and landrace share the same alleles at all nine *Gli-1*, *Gli-2* and *Glu-1* loci {see 01102}.'

After the final paragraphof the preamble (as given above), addthe paragraphs:
'PCR primers GAG5 and GAG6 were applied to 35 cultivars of closely related spelt and hexaploid wheat, and to eight cultivars of durum wheat, to yield products originating from two γ-gliadin genes mapped to chromosomes 1B (termed GAG56B) and 1D (termed GAG56D) {01103}. Two alleles for GAG56D (differing in a 9 bp deletion/duplication and single nucleotide polymorphism) were found, one a new allele and the other previously published {01104}. Meanwhile two alleles found for GAG56B among the durum wheats correlated with the presence of gluten quality markers γ-gliadins 42 or 45.

1B and 1D sulphur-poor ω-gliadins in cultivar Butte 86 were characterised by RP-HPLC, SDS-PAGE, two-dimensional PAGE, amino acid composition determination and sequencing, matrix assisted laser desorption ionisation – time of flight mass spectrometry and circular dichroism spectroscopy to reveal the detailed nature of the peptides belonging to the two groups, and showing that the complexity of mixture of the peptides of the 1B group was greater than that of the 1D group {01105}. Although circular dichroism spectra were similar for the two groups of peptides, and suggested a mainly flexible random structure, there was evidence for a significant amount of left-handed polyproline II helical conformation in the case of the 1D components. The authors placed some of the results in the context of the possible ancestor of the B-genome and relationships with the barley C-hordeins and rye ω-secalins'

4. Enzyme inhibitors (previously, protease inhibitors)
4.4. Inhibitors (dimeric) of heterologous _-amylases
Chromosome 3BS has duplicated loci controlling two dimeric inhibitors of exogenous _-amylases, one known as 0.53 or Inh I {1260}, and the other as WDAI-3 {1260}. Chromosome 3DS has a homoeologous locus controlling a dimeric inhibitor of exogenous _-amylases, known as 0.19 or Inh III {1260,0124}, that is closely related to 0.53/Inh I. Intervarietal polymorphism for the WDAI-3 protein was identified by isoelectric focussing of water-soluble endosperm proteins {0124}. This was mapped on 3BS using both a DH population of Cranbrook/Halberd, and a set of RILs of Opata 85/W-7984 (ITMI population) {0125}.

Iha-B1.1 {1260}.	3BS {1260}.	**v :**	CS {1260}.
Iha-B1.2 {0124}.	3BS {0124}.	**v :**	CS {0124}.
Iha-B1.2a {0124}.		**v :**	CS {0124,0125}.
Iha-B1.2b {0125}.	Null allele.	**v :**	Cadoux {0125}; Cranbrook {0125}; Tasman {0125}.
Iha-D1 {1260}.	3DS {1260}.	**v :**	CS {1260}.

5. Other proteins
5.6 Waxy proteins

Wx-A1. At end of section add : Variation in the microsatellite gene *Xsun1-7A* provides a co-dominant marker for this locus {0116}.

Wx-A1b.		**tv :**	Asrodur {0111}.
Wx-B1b.		**tv :**	Blaquetta (BG-13701) {0111}.
Wx-B1f {0111}.		**tv :**	BG-12413 {0111}; BG-12415 {0111}.
Wx-D1b {0116}.	At end of section add :	**ma :**	Microsatellite marker *Xsun1-7D* is absent in wheats with this allele {1116}; *Xsun4 (Wx)-7D* is a perfect marker {0118}.
Wx-D1d {0118}.		**v :**	K107wx1 {0118}; EMS mutants {0118}.
Wx-D1e {0117}.	Null allele {0117}.	**v :**	NP150 {0117}.

Add {0144} to genotype list.

Add at the bottom of the section:
'Isolation of a wheat cDNA encoding *Wx-A1* and *Wx-D1* was reported in {0123} and {0167}, respectively. Isolation of genomic sequences for the genes encoding granule-bound starch synthase (*GBSSI* or *Wx*) in *T. monococcum*, *Ae. speltoides* and *T. tauschii* was reported in {0168}. Cloning of a second set of *GBSS* or *waxy* genes, *GBSSII*, which were shown to be located on chromosomes 2AL, 2B and 2D, was reported in {0167}.

5.7 Starch granule proteins
At end of section for *Sgp-1* add : 'A triple null stock (SGP-1 null wheat) is reported in {0137}.'.

5.8 Puroindolines
To sentence : 'Present only in some hard wheats' in 2000 Supplement add : '*Pina-D1b* is associated with harder texture than *Pinb-D1b* {0177}.'.

Red Grain Colour
Add at end of section : 'See also Variegated Red Grain Colour'.

Response to Photoperiod

QTL : A QTL was detected in chromosome 4BS in Courtot/CS {0132}.

Response to Vernalization

QTL : Analysis in Courtot/CS {0132}.

Variegated Red grain Colour
Add at end of section : 'Variegated red pericarp was also studied in crosses of cv. Supreme. In this case, two red colour genes were present {0136}.

Yield Components
1000-grain weight
QGw1.ccsu-1A 1AS {0165}. **v:** RS111/CS mapping population {0165}.
{0165}.
ma: Associated with *Xwmc333-1A* {0165}.
QTL: Two QTLs for 1,000-kernel weight were assigned to chromosome 3A in RSLs from Cheyenne*7/Wichita 3A {0025}.

Spike number per square metre
QTL: A QTL for spike number per square meter was assigned to chromosome 3A in RSLs from Cheyenne*7/Wichita 3A {0025}.

Kernel number per spike
QTL: Three QTLs for kernel number per spike were assigned to chromosome 3A in RSLs from Cheyenne*7/Wichita {0025}.

Spike length
QTL: Five QTLs for spike length were detected in Courtot/Chinese Spring {0114} but only one on chromosome arm 5AL was consistent for at least two years.

Spikelet number/ear
QTL: Three QTLs for spike length were detected in Courtot/Chinese Spring {0114} but only two on chromosome arms 2AS and 2BS were consistent for at least two years.

Pathogenic Disease/Pest Reaction

Reaction to Barley Yellow Dwarf Virus.

Bdv2. 7DL= T7DS.7DL-7Ai#1L {0182}. **tr:** H960642 {0182}.
ma: Distal 10% of 7DL, translocation point between RFLP markers *Xpsr680* and *Xpsr965* {0182}.

Reaction to *Diuraphis noxia*

Dn5 **ma:** A SCAR marker developed from the RAPD fragment OPF14 $_{1083}$ mapped 5.5 cM proximal to *Dn5* {0172}.

Reaction to *Erysiphe graminis*

Pm6. **i :** 6 NILs based on Prins {0139}.
ma : *Pm6* was mapped to the interval *Xbcd135-2B – Xpsr934-2B* {0139}. However, the fact that Timgalen and a 'CI12632/Cc' line lacked the critical *T. timopheevii* markers {0139} is cause for concern.

Pm13. **ma :** STS marker *Xutv13* {0036}; several other markers located in introgressed segments {0036}.

Pm24. Add : 'Although *Pm24* had previously been located to chromosome 6D {571},*Pm24* was mapped on chromosome arm 1DS in the cross Chinese Spring (susceptible) x Chiyacao (resistant){0150}.

Pm29 **v :** Pova {0129}.
{0129}.

ma : Location confirmed with molecular markers {0129}.

Pm30 5BS {0163}. **v:** 87-1/C20//2*8866 selections {0163}.
{0163}.

tv: *T. dicoccoides* accession C20 {0163}.

MlRE. 6AL {0142}.
Add at the end of the sentence '*Mlre* showed a residual effect on the quantitative expression of APR in the presence of *E. graminis* pathotypes considered virulent for *Mlre* in standard seedling tests {0016}.':
'In addition to the *Mlre* gene on chromosome arm 6AL, a QTL for resistance effective at the seedling stage was identified on chromosome 5D in {0146}. The QTL was associated with microsatellite marker *Xgwm174-5D.*'.

Reaction to *Fusarium graminearum*
Replace previous section:

QFhs.ndsu-2A {9925,0175}. 2AL {9925}. **v:** Stoa {0081}.
ma: Associated with *XksuH16-2A* (LOD>3) {0175}.
QFhs.ndsu-3B {9925,0175}. 3BS {9925}. **v:** Sumai 3 {9925,0175}.
ma: Associated with *Xbcd907-3B.2* (LOD>3) {9925} andmicrosatellite markers *Xgwm533* and *Xgwm493* {0175}.
Add at the bottom of the section :
'Two major genes with additive effects were reported in crosses between Sumai 3 (resistant) and two susceptible cultivars {0174}. One of the genes was assigned to 5AL based on linkage to the dominant awn suppressor B1 (RF 15.1 – 21.4 %).'.

Reaction to *Heterodera avenae*

Cre2 {238}. **v:** Replace H93-8 {238} with 'H-93-8 *Cre6* {238}.'.
Cre5 {0107}. Derived from *Aegilops ventricosa* {0107,0009}. *CreX* {0009,0183}. 2AS{0107}= 2A-2N^{v}-6N^{v}.
v: VPM1 {0107}. Many VPM1 derivatives {0107}. Notable exceptions of lines with *Lr37, Sr38* and *Yr17,* but lacking *Cre5* include Trident and Line L22 {0107}.
su : Moisson 6N^{v}(6D) {0183}.
dv : *Ae. ventricosa* 10 {0183}.
Two resistance gene analogues similar to the candidate gene *Cre3* were isolated from the *Ae. ventricosa* segment carrying *Cre5* {0183}.
Cre6 {0138}. Derived from *Aegilops ventricosa* {0138}. 5N^{v} {0138}.
ad : Moisson + 5N^{v} {0138}. **v :** H-93-35 {0138}; H-93-8 *Cre2* {0138} .
Cre7 {0104}. *CreAet*{0105}. Derived from *Aegilops triuncialis* {0105}.
v : TR353 derivatives {0105}.

CreR {0133}. 6RL {0133}. **su :** CS + 6R(6D) {0133} **al:** Rye accession T701-4-6 {0133}.
ma : Cent.....*XksuF37* – 3.7cM – *CreR* {0133}.

Reaction to *Meloidogyne* spp.

Rkn-mn1 Derived from *Aegilops variabilis* {1621}.
{1621}.
v : X8 = CS/ *Ae variabilis* No 1//Rescler/38*Lutin {1620} ; X35 {1620,1621}.
ma : Co-segregation with RAPD *OpY161065* and close linkage with several markers including *Est-B5* {0103}.

Reaction to *Mycosphaerella graminicola*

Stb5 {0186}. Identified using *M. graminicola* culture IPO94269 {0186}.
Derived from *Aegilops tauschii* accession 37-1 {0186}.

7DS {0186}. **su:** CS*8/(Syn7D) {0186}. **v:** Sear's Synthetic {0186}.

Stb6 {0187}. Confers resistance to *M. graminicola* isolate IPO323 but not to isolate IPO94269 {0187}. 3AS {0187}. **v:** Flame {0187}.

Reaction to *Pratylenchus* spp.
Disease : Root lesion nematode; prats

1. Reaction to *Pratylenchus neglectus*

Rlnn1 {0121}. 7AL {0121}. **v :** Excalibur {0121}; Krickauff {0121}.

2. Reaction to *Pratylenchus thornei*
QTLs were located on chromosomes 2BS and 6DS {0122}.

Reaction to *Puccinia graminis*
Following SrZdar, to genotype list add : '0102'.

Reaction to *Puccinia recondita*

Lr19. Add to ma : ' The following gene order for the *Thinopyrum* segment is given in {0101} : Cent – *Sd1* – *Xpsr165 - Xpsr105 – Xpsr129 – XcsIH81-1 – Xwg380 – Xmwg2062 – Lr19 – Wsp-D1 – Sr25/Y.*'.

Lr37. A resistance gene analog containing an NBS-LRR R gene sequence was isolated from the*Ae ventricosa* segment carrying *Lr37* {0183}.

Lr46. 1BL {0119}. **v :** Pavon F76 *Lr1 Lr10 Lr13* {0119}. *Lr46* is completely linked with *Yr29* {0119}.

Lr47. Modify and add to earlier sentence : 'Complete linkage with several RFLP {9901} and PCR specific markers {0126}.'.

To genotype list, for Czeckoslovakian cultivars add reference{0102} to {855}, i.e. '{855,0102}'.

Reaction to *Puccinia striiformis*

Yr15. **ma :** *Xgwm33-1B* – 4.5cM – *Yr15* – 5.6cM – *UBC199200* – 5.6cM – *Nor-B1* {0110}.

Yr28 **ma :** Close association witk *Xmwg634-4DS* {1377}.

Yr29 {0119}. Adult plant resistance {0119}. 1BL {0119}.
v : Pavon F76 *Yr6 Yr7 Yr30* {0119}. *Yr29* is completely linked with *Lr46* {0119}.

Yr30 {0120}. Adult plant resistance {0120}. 3BS {0120}.
v : Opata 85 {0120} ; Parula {0120}. Inia 66 *YrA* {0120}. Pavon F76 *Yr6 Yr7 Yr29* {0120}. *Yr30* is closely linked with *Sr2* and *Lr27* {0120}.

YrH52. **ma :** *Xgmw273a* – 2.7cM – *YrH52* – 1.3cM – *Xgwm413/Nor1*.....centromere {0108}.

Reaction to *Tilletia caries* (D.C.) Tul., *T. foetida* (Wallr.) Liro, *T. controversa*

Bt10. **v :** Add to Others : '{0128}'. **ma :** Add at the end : 'The RAPD fragment was sequenced and converted to a diagnostic PCR marker for*Bt10* in {0151}.'.

Reaction to Wheat Spindle Streak Mosaic Bymovirus (WSSMV)

QTL : . 79% of the variation between Geneva (resistant) and Augusta (susceptible) was associated with markers *Xbcd1095-2D* and *Xcdo373-2D* located 12.4cM apart in chromosome 2DL {0131}.

Genetic Linkages

Much of the information listed in this section comes from work carried out prior to the use of DNA markers. More recent information from molecular markers is provided under 'ma:' within sections describing individual genes. There, the linkage values are usually limited to 10cM. Further integrated mapping information can be found in:

tv: {0185,0184}.

In the following section, unless otherwise indicated.....(as in 1998 Catalogue).

Chromosome 1D

1DS

Pm22	-	*Pm24*	I {0150}.

Chromosome 3A

Br2	-	*R-A1b*	44.2cM {0130}
Br3	-	*R-B1b*	47.0cM {0130}

Chromosome 6B

Amp-B2	-	*B2*	0.9% {0176}
Amp-B2	-	*B2*	2.1% {0176}

Summary Tables

Additions to Summary Table 1

Amc	Amylose content
Gw	Grain weight
Iha	Inhibitor (dimeric) of heterologous -amylase
Plnn	Reaction to *Pratylenchus neglectus*
Plnt	Reaction to *Pratylenchus thornei*

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Amendments.

1377. Replace: Singh RP, Nelson JC & Sorrells ME 2000 Mapping *Yr28* and other genes for resistance to stripe rust in wheat. Crop Science 40: 1148-1155.
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0001. Euphytica 115: 121-126.
0002. Genome 43: 377-381.
0022. Peusha H, Enno T & Priilinn O 2000 Hereditas 132: 29-34.
0029. Wheat Information Service 91: 33-70
0030. Theoretical & Applied Genetics 100: 686-689.
0034. Genome 43: 191-198.
0050. Theoretical and Applied Genetics 100: 419-431
0081. Theoretical & Applied Genetics 100: 519-527

Deletion

0078. Delete, identical with {9925}.

New.

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Editorial remarks

WIS No.93 contains 7 research and 3 information articles, in addition to 21 pages of Gene Catalogue (2001 supplement). The decision to have a wheat gene catalogue was made at the Third International Wheat Genetics Symposium held in Canberra in 1968. At the symposium Dr. R. A. McIntosh was appointed as a coordinator, and the first catalogue was publicized at the Fourth Symposium at Columbia Mo., USA in 1973, which included 328 citations. Since then, various wheat researches have been achieved, and numbers of references cited in the present supplement reaches 1882. Recently more than 120 articles have been added annually. Dr. McIntosh have mentioned in the preface of the 1998 issue "If the Catalogue is to continue it is essential to link it to a database capable of rapid updating". The present Catalogue was prepared in a way that should expedite transfer to such a system. The Catalogue is never complete. Information can always be added – the problem is what information, and what form. Yes indeed, and WIS is willing to support his idea and proposal by continuing publication of supplement issues every year. At the same time, a Japanese database group KOMUGI (http://www.shigen.nig.ac.jp/wheat.html) has started to construct a database of the gene catalogue through a web interface. Already GrainGenes (http://wheat.pw.usda.gov) and KOMUGI have referred this gene catalogue on their cites, but not yet linked to map information nor genetic stocks. Send us about ideas for construction of a wheat gene catalogue database.

WIS will keep functioning for exchange of information on wheat genetics and breeding. At the present time, we have constantly received contribution papers, and the average acceptance for publication is about 60%. The category of Research Information is on the base of non-reviewing system and would like to be published more frankly. We appreciate deeply your donation. In the year of 2001, we have received it from 72 persons. On TV news, frequently the landscapes of Afghanistan appear. Some wheat researchers may worry about the wild habitat of *Aegilops* species. We hope peace and safe in the world.

December, 2001
The Editors of WIS

Kihara Memorial
Yokohama Foundation for the Advancement of Life Sciences

The Kihara Memorial Foundation (KMF) was established in 1985 in memory of the late Dr. Hitoshi Kihara, a world famous geneticist and evolutionary scientist. The activities of the KMF are promotion of life science by supporting symposia, workshops, and technical courses for researchers, enlightenment of scientific information to citizens, awarding of 'KMF Prize' and 'Child Scientist Prize', and publication of journals such as 'Wheat Information Service'.

The 21st century is the century of life sciences. KMF intends to continue contribution for a better future of the earth to solve many problems facing us such about health, food, resources and environment.

The recent economic condition in Japan is limiting our support of these KMF activities. KMF is, therefore, taking up subscriptions from colleagues who approve of the activities of KMF. We would appreciate receiving from you inquiries about this matter, thank you.

Kihara Memorial Foundation

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