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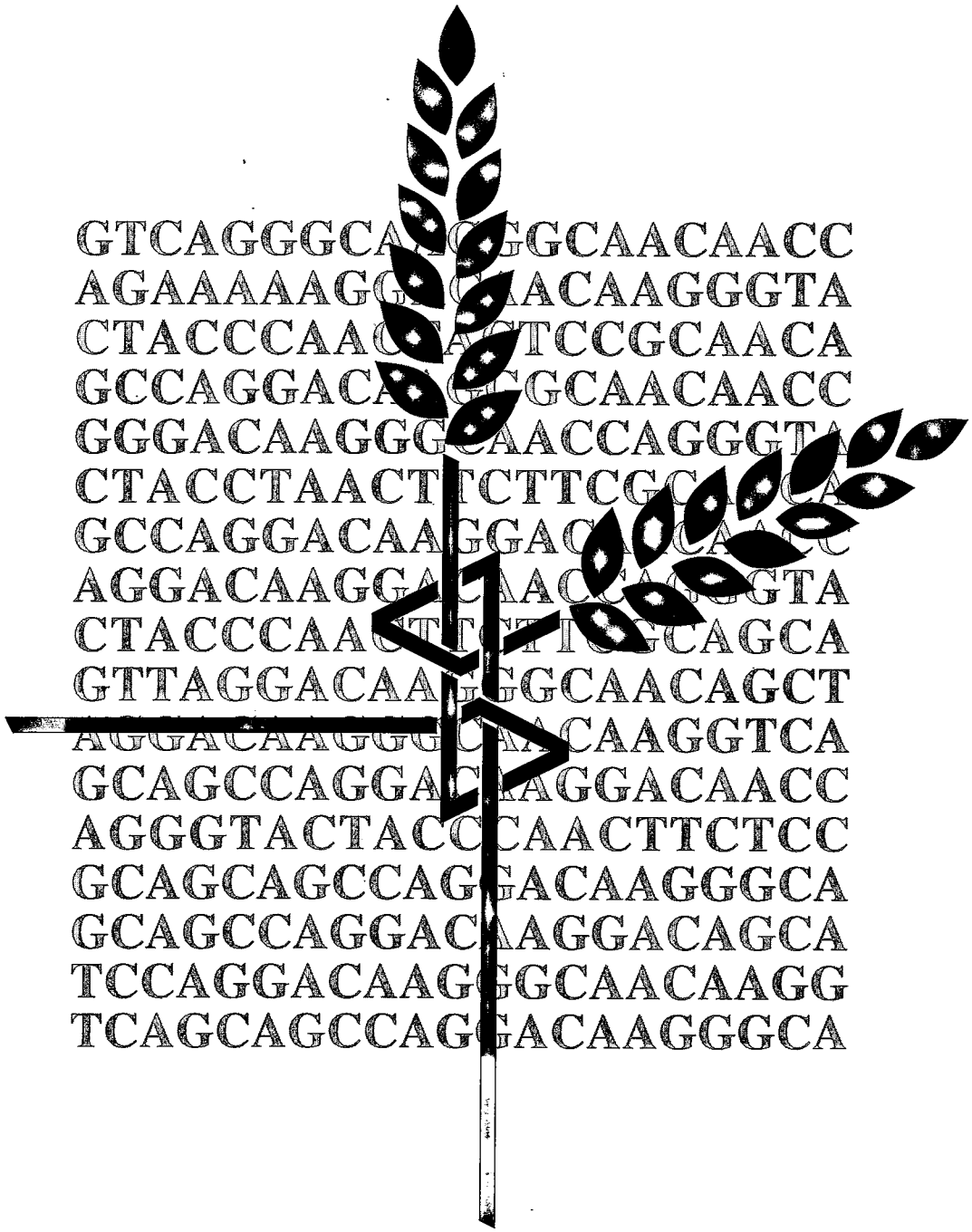

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Research article

Assimilate transportation efficiency in diverse wheat accessions in the absence of leaf photosynthesis

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Summary

In order to elucidate assimilate transportation efficiency, diverse wheat accessions were subjected to defoliation and potassium iodide induced leaf senescence. Grain development in response to these treatments was compared with that under late sown condition, which exposed the crop to high temperatures. It was observed that KI (0.25%) sprayed at the time of anthesis severely affected grain growth by interfering with both the assimilate supply and other processes associated with grain formation. On the contrary, defoliation reduced only the grain weight but not the grain number/spike. Defoliation could also partly explain the response of late sown crop to terminal heat stress. It is concluded that KI (0.25%) can not be used to screen wheat accessions for assimilate transportation efficiency. It is suggested that lower concentration of KI or any other desiccant with no effect on processes in the developing grain needs to be tested and used for screening large number of accessions.

Introduction

Late sown wheat is invariably exposed to temperatures greater than 30 °C during grain development in North Western Plains Zone of India (Nagarajan and Rane 1998). Hence, improvement of heat tolerance in wheat is one of the major objectives of breeding programs in this region. However, the limited progress made so far is mainly due to the lack of suitable techniques for screening large number of accessions. Widely proposed screening methods like canopy temperature depression (Reynold et al. 1994) and membrane thermostability (Fokar et al. 1998) are rarely used by breeders because of complexity involved in handling large number of accessions.

Translocation of assimilate accumulated in stem during grain growth under stress environment is one of the promising traits for selection of stress tolerant genotypes. Translocation of the assimilate responds to drought stress, removal of leaves and senescing agents such as potassium iodide (KI) particularly during grain growth in wheat (Nicolas and Turner 1993), pearl millet (Mahalakshmi et al. 1994) and triticale (Royo and Blanco 1998). KI when sprayed at appropriate time bring about gradual decrease in chlorophyll content and photosynthesis. As a result, developing grains are forced to depend on the amount of assimilate stored in stem and its translocation. Reduction in stem biomass of wheat plants in response to elevated temperatures has also indicated increased utilisation of stem reserves under heat stress conditions (Stone et al. 1995).

The present study was conducted with two objectives viz., evaluation of large number of diverse wheat accessions for the ability of translocating the assimilate accumulated in stem and to explore the possibility of using potassium iodide for screening for this trait. The ultimate aim was to develop breeder friendly, efficient and rapid technique to screen early generation breeding material for heat tolerance.

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Material and methods

Experiments were conducted under natural environment in a small piece of land (clay loam soil) with negligible soil-heterogeneity. Ten seeds each of 90 diverse wheat (*Triticum aestivum* L.) accessions were planted in separate rows with 10cm and 23cm distance between seeds and rows respectively. Two sets of seeds were planted in time and one set was planted late (60 days after first sowing). Genotypes with poor germination or less than 10 plants were rejected and ultimately 69 entries were used for further analysis. Three plants with uniform morphology and flowering time, from each row in the first set were subjected to manual defoliation 90 days after sowing and remaining plants were maintained as a control. Simultaneously, all the genotypes in the second set were sprayed with 0.25% of KI solution. This concentration was chosen on the basis of earlier reports (Mahalakshmi et al 1994; Royo and Blanco 1998) and also by our preliminary observations. Spraying was carried out until whole plant including tillers was fully drenched. Days to anthesis and physiological maturity was noted in all the four treatments viz., manual defoliation, KI spray, late sowing and control. Matured main shoot-spike of three plants of each accession from all the four treatments was sampled for studying grain development. Spikes were threshed manually and each grain was separated with maximum care. Grain weight and grain number in each spike was recorded. Standard deviation expressed as a percent of mean (coefficient of variation) was used to explain the variability in response of accessions to different treatment.

Results and discussion

There was remarkable reduction in grain growth period (from 41 days to 23 days) when genotypes were planted late mainly due to continuous rise in temperature. Potassium iodide (KI) brought about gradual decrease in chlorophyll content in leaves as reported in earlier studies (Mahalakshmi et al. 1994; Royo and Blanco 1998). However, in defoliated plants spike remained green for longer time.

Significant difference in flowering time of accessions was recorded with days to anthesis and maturity ranging from 77 to 97 days and 129 to 137 days after sowing, respectively. As a result, defoliation or KI spray carried out on the same day exposed genotypes to these treatments 0 to 20 days after ear emergence (Table 1). Breeders dealing with segregating material often come across such constraints, wherein comparison between response of accessions becomes difficult. However, to have a realistic interpretation of data, accessions were categorized into four groups on the basis of stage of crop at the time of treatment. The accessions in the first two groups were exposed to defoliation or KI treatment before or at the time of anthesis. Accessions in the other two groups were exposed to these treatments one week or two weeks after anthesis. Early stages of grain development were more prone to damage caused by defoliation, KI treatment or high temperatures.

There was considerable variability in grain weight /spike recorded in 69 wheat genotypes which ranged from 1.4 to 3.3 g with mean 2.1 g and coefficient of variation (26 %) (Table 2). However, in response to defoliation, variability reduced to 18.4 % indicating that the differences in grain weight/spike might be partly due to variation in current photosynthesis. This was also evident in genotypes subjected to delayed sowing, wherein CV for grain weight/spike was as high as 23.9 %. These results are on par with observations in which contribution of current photosynthesis plays a major role both under normal and stress condition (Setter et al. 1998). The pooled analysis revealed that manual defoliation

Table 1. Effect of different treatments on grain weight and grain number in different groups.

Group	Time of treatment*	Frequency of genotypes	% reduction in grain weight			% reduction in grain number		
			Defoliation	KI-treatment	Late sowing	Defoliation	KI-treatment	Late sowing
I	3	10	29.68	71.42	56.35	9.61	49.41	24.66
II	8	22	26.79	66.25	52.40	13.09	46.88	22.39
III	13	24	19.51	64.44	38.95	10.95	44.43	19.81
IV	18	13	19.74	61.33	42.37	7.63	32.28	23.20

* Days after spike emergence (± 2 days)

Table 2. Effect of different treatments and variability among wheat genotypes

Treatment	Grain weight/spike (g)				Grain number/spike				Single grain weight (mg)			
	Range	CV (%)	Mean	DMRT	Range	CV (%)	Mean	DMRT	Range	CV (%)	Mean	DMRT
Control	1.4-3.3	26.1	2.1	A	34.7-79.7	18.4	53.6	A	22.3-55.7	38.6	38.6	A
Manual defoliation	1.0-2.6	18.4	1.7(19)	B	35.7-74.3	16.4	53.3(1)	A	22.3-51.0	31.3	31.3(19)	B
KI-spray	0.3-1.9	49.8	0.7(67)	D	14.3-52.0	27.8	31.4(41)	C	13.7-58.6	22.7	22.5(42)	D
Late sowing	0.6-1.8	23.9	1.1(48)	C	26.7-60.3	18.3	43.9(18)	B	15.3-42.3	25.6	25.6(34)	C
Cd at 0.05			0.07				1.72				1.24	

Figures in braces indicate percent over control, CV: Coefficient of variation (standard deviation/mean) x 100, DMRT: Duncan's multiple range test

significantly reduced the grain weight/spike but not the grain number. In contrast to this, remarkable reduction in both of these traits was observed in response to KI spray and delayed sowing. Significant correlation coefficient between reductions in grain weight/spike and grain numbe/spike in response to KI spray ($r=0.73$, $p=0.01$) indicated that the reduction in grain weight/spike was largely due to reduction in grain number rather than reduction in assimilate availability. Further, there was significant correlation ($r=0.83$, $p=0.01$) between number of grains in untreated control and those in KI-treated plants. Presumably, KI might have selectively and uniformly inhibited initial post anthesis processes associated with grain development. It might be due to toxic effect of KI on development of grains at disadvantageous location within the spikes or spikelets of all the accessions. Reduced availability of assimilate for florets located in the distal part of the spike and spikelets has been reported when source was a limiting factor (Slafer et al. 1996). Reduction in grain weight/spike in response to defoliation could not be explained by reduction in grain number. Instead, it was obvious from reduction in individual grain weight that there was reduction in assimilate supply to the developing grains. This was further supported by significantly high correlation ($r=0.63$, $p=0.01$) between grain weight/spike in defoliated and those in control plants irrespective of variation in flowering time. Hence, it was inferred that response to defoliation could explain the potential of the accessions to mobilize stem reserves in a better way as compared to KI treatment.

No perceptible relation was observed between response to KI spray and defoliation. This could be attributed to difference in effect of these two treatments on grain number/spike. Response of accessions to defoliation could partly explain their performance under late sown condition as there was significant correlation between grain weights in response to these two treatments ($r=0.54$, $R^2=0.3$). However, failure of perfect simulation as reflected by low R^2 value was mainly because of the fact that the reduction in grain weight/spike was largely due to reduced grain growth period under late sown condition. Under such conditions, both the amount and rate of supply of assimilate must have played a significant role.

It is concluded that KI (0.25%) used to stop leaf photosynthesis also affected processes other than assimilate supply during grain growth. Hence, it can not be used to determine potential of wheat genotypes to use the carbohydrate accumulated in stem for grain development. However, lesser concentration or novel chemicals with no effect on biological activities in the developing grain may be used to explain the translocation of assimilate for grain development and yield potential of wheat genotypes under heat stress environments.

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Wheat Information Service
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Research article

Transfer of resistance to wheat pathogens from *Aegilops triuncialis* into bread wheat

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Summary

An interspecific cross was made to transfer leaf rust, Karnal bunt, powdery mildew and cereal cyst nematode resistance from a non-progenitor tetraploid species, *Aegilops triuncialis* (UUCC), into bread wheat. Sterile F_1 of the cross between susceptible *Triticum aestivum* cv. WL 711 and a resistant accession of *Ae. triuncialis* (Acc.3549) was backcrossed to the cultivated parent. Two sets of resistant derivatives were selected from selfed progenies of BC_2/BC_3 plants. One group of derivatives with 42 chromosomes had spelta type head and possessed resistance to cereal cyst nematode and powdery mildew in addition to moderate resistance to leaf rust. Giemsa C-banding of mitotic metaphase chromosomes showed that these derivatives possess a substitution of 5U chromosome of *Ae. triuncialis* for 5A of bread wheat. The second set of derivatives (2n=44) with disomic addition of an acrocentric *Ae. triuncialis* chromosome possessed leaf rust, Karnal bunt and powdery mildew resistance. Genomic *in situ* hybridization showed that this set of derivatives also possess a pair of translocated chromosomes involving break point in the centromere and short arm of *Ae. triuncialis* chromosome.

Introduction

Wild relatives of wheat have proven to be useful sources of novel genes for resistance to various diseases (Sharma and Gill 1983; Gale and Miller 1987; Jauhar 1993; Jiang et al. 1994; Friebe et al. 1996). A number of genes for resistance to various diseases and pests have been transferred from closely related as well as distantly related species (McIntosh et al. 1998). However, many of the alien disease resistance genes transferred into wheat cultivars have been overcome, thereby necessitating the search for new sources of resistance.

Evaluation of different accessions of wild *Triticum* and *Aegilops* species maintained at the Punjab Agricultural University, Ludhiana, led to the identification of a number of new sources of resistance to wheat diseases including leaf rust, stripe rust, powdery mildew, Karnal bunt, loose smut and cereal cyst nematode (Dhaliwal et al. 1993; Gill et al. 1995; Dhaliwal and Harjit-Singh 1997; Harjit-Singh et al. 1998). The studies showed that among the less closely related species, *Aegilops* species with the C, U and M genomes are excellent sources of resistance to leaf rust, stripe rust, powdery mildew, Karnal bunt and cereal cyst nematode (Dhaliwal et al. 1991, 1993; Pannu et al. 1994; Harjit-Singh et al. 1998). Keeping this in view a wide hybridization programme was initiated to transfer the disease resistance from tetraploid *Aegilops* species carrying these less related genomes (Harjit-Singh et al. 1993). In the present paper, we describe the transfer of disease and cereal cyst nematode resistance genes from *Ae. triuncialis* into bread wheat.

Materials and methods

Triticum aestivum cv. WL 711 (a widely adapted and agronomically superior Indian spring wheat cultivar)

Table 1. Reactions to leaf rust, powdery mildew, Karnal bunt and Cereal cyst nematode of parents

Parent	Reaction										
	Leaf rust								Powdery mildew (seedling reaction)***	Karnal bunt (percent incidence)	Cereal cyst nematode (% change in cyst population)
	Seedling reaction to pathotype*							Adult plant reaction			
	77	77A-1	77-1	77-2	77-4	77-5	104-1				
T. aestivum cv. WL 711	3+4-	3+4-	33+	4	4	34	–	90S-100S**	4	22.5-99.0	+218.2
Ae. triuncialis Acc.3549	0;2	0;	0;	0	00;	0	0	F	0	0	-68.2

* Resistant: 0 to 2, Susceptible: 3 to 4 (Seedling reactions recorded on 0; to 4 scale. The range of infection types produced on different plants by a given race is presented without spaces or dashes between the figures).

** S: Susceptible, F:Free

*** Avirulence/virulence attributes of pathotype (Keylong isolate) used for seedling tests: *Pm1, Pm2, Pm3b, Pm4a, Pm4b, Pm8/Pm3a, Pm3c, Pm5, Pm6, Pm7* (Data recorded on 0-4 scale).

was crossed as female with a leaf rust, Karnal bunt, powdery mildew and cereal cyst nematode resistant accession (Table 1) of *Ae. triuncialis* (Acc.3549). WL 711 is susceptible to these diseases/pests. The sterile F_1 was backcrossed to *T. aestivum* cv. WL 711. The desirable progenies were selfed after two to three backcrosses. Data on chromosome number and meiotic chromosome pairing were recorded in F_1 and the subsequent backcross/selfed progenies. The observations on field reaction to leaf rust were recorded at adult plant stage in each generation by using modified Cobb's scale (Paterson et al. 1949). However, seedling response to an individual pathotype of leaf rust was recorded in early backcross generations (up to BC_2) and a part of advanced progenies. The standard procedure for inoculation of seedlings (Nagarajan et al. 1986) was followed and seedling response to rust was recorded on 0-4 scale (Knott 1989). The addition/substitution/translocation of the alien chromosome(s) or chromosome part (s) were investigated through Giemsa C-banding (Friebe et al. 1992) and/or genomic *in situ* hybridization (Mukai and Gill 1991). Advanced progenies were scored for reaction to leaf rust, Karnal bunt, powdery mildew (one Indian isolate from Keylong and a Japanese isolate) and cereal cyst nematode. To record reaction to Karnal bunt, 10-15 plants from each progeny were inoculated at boot stage (3-5 tillers per plant) using artificial inoculation method of Aujla et al. (1982) and percent incidence of disease was recorded on tiller basis at maturity. Reaction to powdery mildew was recorded on first leaf of 7-10 days old seedlings. Seedlings were inoculated by dusting conidia and inoculated seedlings were incubated in a growth chamber at 20 ± 2 °C, 70-80% relative humidity and 14 hours day light. The infection types were recorded on 0-4 scale (Smith and Blair 1950) after 8-14 days of inoculation. Reaction to cereal cyst nematode, *Heterodera avenae*, was recorded under artificial inoculation conditions following the method used by Singh et al. (1991).

Results and discussion

A set of derivatives from the cross *T. aestivum* cv. WL 711 x *Ae. triuncialis* Acc.3549 had spelta type head, a characteristic associated with monosomy for 5A. C-

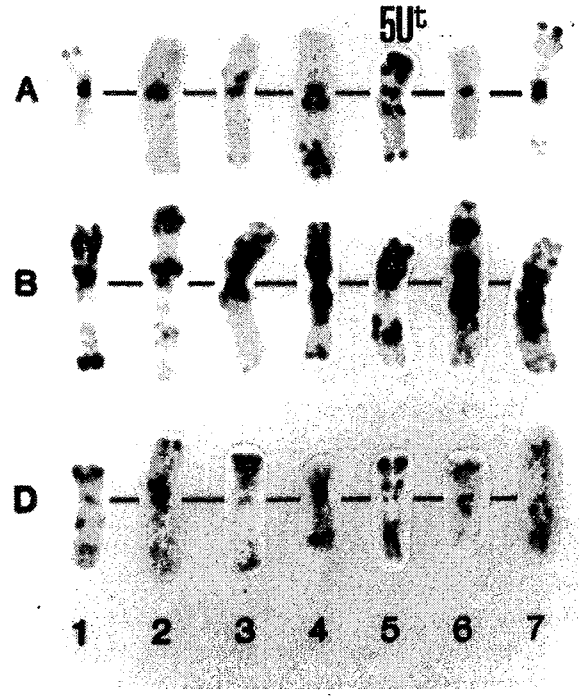

Fig.1. Giemsa C-banding of mitotic metaphase chromosomes of an interspecific derivative showing substitution of 5U of *Ae. triuncialis* for 5A of bread wheat.

banding of mitotic metaphase chromosomes showed that in these derivatives chromosome 5U was substituted for 5A (Fig. 1). Genomic *in situ* hybridization further confirmed the presence of this satellited chromosome (Fig. 2a). These derivatives with 42 chromosomes had 0 to 4 univalents and occasionally a quadrivalent. The occurrence of a quadrivalent suggested the homoeology of 5U with one of the chromosomes of group 5 of wheat (5B or 5D).

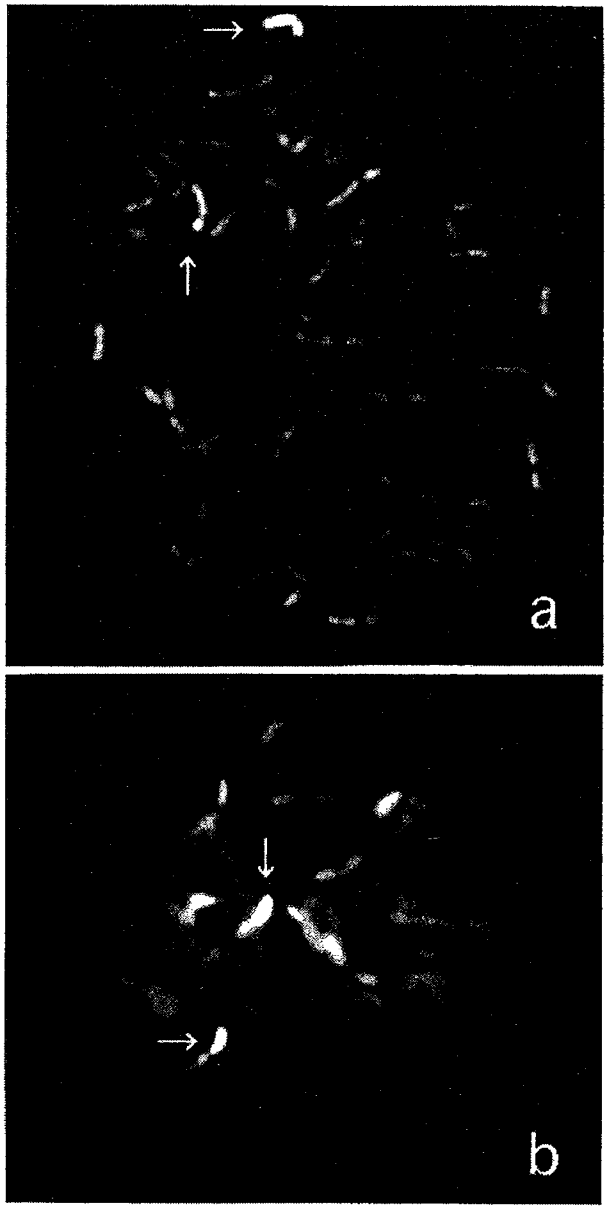

Fig.2. Genomic *in situ* hybridization of mitotic chromosomes of interspecific derivatives. a:showing substitution of a satellited chromosome from *Ae. triuncialis* (arrow) b: showing a pair of translocated chromosomes with short arm of *Aegilops triuncialis* chromosome (arrow)

The progenies gave a high reduction in cyst population of *Heterodera avenae* as compared to the initial cyst population under artificial screening (Table 2). The significant reduction in the cyst population in *Ae. triuncialis* and increase in the susceptible recurrent parent (WL 711) suggested that the nematode resistance of these derivatives has been derived from chromosome 5U of *Ae. triuncialis*. These derivatives also exhibited moderate resistance to leaf rust under field conditions as compared to highly susceptible recurrent parent (90S to 100S). One of the progenies (L98.99-1180) that segregated for leaf rust resistance also segregated for spelta/normal head. The plants with spelta head possessed moderate resistance (20X) and those with normal head were susceptible (80S). This suggested that the 5U chromosome of *Ae. triuncialis* in these derivatives is also carrying the gene(s) for moderate leaf rust resistance. However, this leaf rust resistance may be of adult plant type resistance as the BC_2 plant from which this set of progenies were derived (BC_2 plant No.20) exhibited 3+ seedling reaction to pathotype 77A-1 (Avirulence/virulence formula: *Lr 9, Lr20, Lr23, Lr26/Lr1, Lr3, Lr10, Lr13, Lr15*). Also, two progenies, L98.99-1183 and L98.99-1184 gave 1+ to 3 and 2 to X reactions, respectively, to pathotype 77-2 (Avirulence/virulence formula: *Lr9, Lr26/Lr1, Lr3, Lr10, Lr13, Lr15, Lr20, Lr23*) whereas *T. aestivum* cv. WL 711 exhibited 3+ to 4 (susceptible) reaction and *Ae. triuncialis* was resistant (reaction=0) in this test. These two progenies exhibited 3+ to 4 and 3 to 3+ reactions, respectively, to pathotype 77-5 (Avirulence/virulence formula *Lr9/Lr1, Lr3, Lr10, Lr13, Lr15, Lr20, Lr23, Lr26*). *T. aestivum* cv. WL 711 was fully susceptible (reaction = 4) and *Ae. triuncialis* was resistant (reaction = 0 to 0;) to pathotype 77-5. The intermediate seedling responses of these progenies to pathotypes 77-2 and 77-5 further supported the presence of low adult plant resistance which is presumably due to the rust resistance gene(s) on 5U. These derivatives also carried moderate to high seedling resistance to an Indian isolate (Keylong isolate) of powdery mildew. One of the progenies that segregated for resistance to powdery mildew (L98.99-1184) also segregated for resistance to leaf rust.

Another set of derivatives (Table 3) possessing resistance to leaf rust from *Ae. triuncialis* were obtained among the backcross progenies. Genomic *in situ* hybridization showed that these progenies possess a pair of translocated chromosomes where the break point is on the centromere and the short arm of the translocated chromosome is alien (Fig. 2b). These progenies possess 44 chromosomes as there is disomic

Table 2. Pedigree and disease reactions of interspecific derivatives carrying 5U substitution for 5A from the cross *T. aestivum* cv. WL 711 x *Ae. triuncialis* Acc.3549

Derivative no.	Pedigree	Reaction to leaf rust*		Reaction to *H.avenae*		Reaction to powdery mildew	
		1997-98	1998-99	1997-98***	1998-99	Indian isolate	Japanese isolate
L98.99-1167	$BC_220/4/WL711$-5-2-5-16 x	10S**	5S-40S	–	–	–	–
L98.99-1168	$BC_220/4/WL711$-5-2-5-24 x	10S	5S-10S	-100.0	-92.42	R#	R
L98.99-1169	$BC_220/4/WL711$-5-2-5-25 x	10S	5MR	-100.0	-62.12	R	R
L98.99-1172	$BC_220/4/WL711$-5-2-13-11 x	40S	R/5MR-40S	-100.0	-92.42	MR	R
L98.99-1174	$BC_220/4/WL711$-5-2-13-38 x	20S	5S-10S/5MR	-100.0	-72.27	MR	R
L98.99-1176	$BC_220/4/WL711$-5-2-4-19 x	40S	5X-10X	-81.8	-92.42	–	–
L98.99-1180	$BC_220/4/WL711$-5-2-6-16 x	10S	20X/80S@	-100.0	-100.00	–	–
L98.99-1183	$BC_220/4/WL711$-5-8-2-21 x	20S	5X-10X	-100.0	-84.84	MR	–
L98.99-1184	$BC_220/4/WL711$-5-8-2-34 x	10S	5S-20S/60S	-100.0	-92.42	MR/S	–
Parents	*T. aestivum* cv. WL 711	90S	90S	+29.3	+218.2	S	S
	Ae. triuncialis Acc.3549	F	F	-74.1	-68.2	R	R

* Adult plant reaction of single plant in 1997-98 and reaction of the progeny from same plant in 1998-99.

** F: Free from rust, R: resistant flecks, MR: moderately resistant, S: Susceptible.

*** Reaction of the progeny from which single plant was picked up to produce the progeny in 1998-99.

@ Spelta type plants (presumably carrying 5U) had 20X reaction whereas non-spelta type plant had 80S reaction.

For reaction to powdery mildew, R: 0 to 1 on 0 to 4 scale, MR: 2 to 3, S: 4.

Table 3. Pedigree and disease reactions of interspecific derivatives from the cross *T. aestivum* cv. WL 711 x *Ae. triuncialis* carrying a homozygous translocation with short alien arm and a disomic addition of an acrocentric chromosome.

Derivative no.	Pedigree	Reaction to leaf rust		Reaction to Karnal bunt (% incidence)	Reaction to powdery mildew (seedling response)
		1997-98	1998-99		
L98.99-1268	BC_218-13-2-13-11 x	0	0-5MR*	0	MR**
L98.99-1213	BC_218-13-2-13-12 x	0	0-5MR	0	MR
L98.99-1214	BC_218-13-2-13-13 x	0	0	–	MR
L98.99-1269	BC_218-13-2-13-33 x	0	0-tR	0-4.5	MR
L97.98-51-34	BC_218-13-2-13-34 x	0	–	–	R
L97.98-51-37	BC_218-13-2-13-37 x	0	–	–	R
L98.99-1218	BC_218-13-2-13-42 x	0	0	–	MR
Parents	*T. aestivum* cv. WL711	90S-100S	90S-100S	22.5-99.0	S
	Ae. triuncialis Acc.3549	0	tR	0	R

* For reaction to leaf rust, R: resistant, MR: moderately resistant, S: susceptible, tR: resistant flecks in traces.

** For reaction to powdery mildew, R: 0 to 1 on 0 to 4 scale, MR: 2 to 3, S: 4

addition of an acrocentric chromosome (Fig. 3). Occasionally, a trivalent (mean frequency from 0.04 to 0.13) or a quadrivalent (mean range of 0.02 to 0.16) was observed. Also 0 to 4 univalents (mean ranging from 0.20 to 1.12) were observed in these derivatives.

The derivatives carrying the homozygous translocation and disomic addition of acrocentric chromosome were derived from the BC_2 plant No.18 that had exhibited 1 to 2- seedling response to leaf rust pathotype 77A-1. The selfed progeny of the plant No.BC_218-13-2-13 from which the derivatives presented in Table 3 were derived, also showed

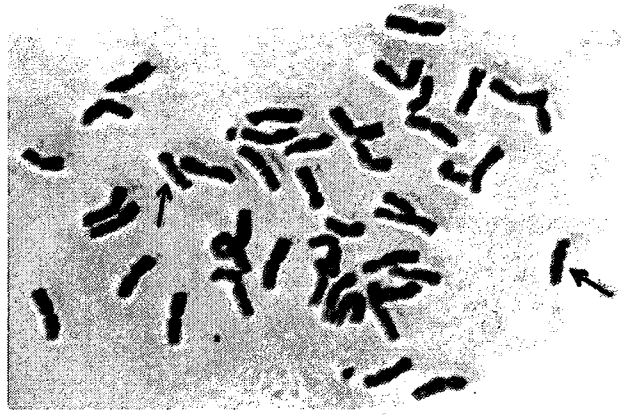

Fig.3. Mitotic metaphase of an interspecific derivative showing disomic addition of an acrocentric chromosome (arrow)

seedling resistance (Table 4) to pathotypes 77-2 as well as 77-4 (Avirulence/virulence formula: *Lr9, Lr20, Lr26/Lr1, Lr10, Lr13, Lr15, Lr23*). Progenies of two plants from this progeny exhibited uniform seedling resistance to pathotype 77-5 as well. So this set of derivatives possesses seedling resistance to leaf rust that is effective at adult plant stage. The slight shift in adult plant response to leaf rust (from 0 to tR/5MR) may be attributed to some shift in racial structure of pathogen population (Nayar et al. pers comm).

The recurrent parent is highly susceptible to Karnal bunt. It had disease incidence ranging from 22.5 to 99.0 percent whereas *Ae. triuncialis* remained free from Karnal bunt under artificial conditions. Out of the three interspecific derivatives (Table 3) tested, two remained completely free from Karnal bunt. In the third progeny, a few plants had disease incidence up to 4.5 percent. In case of Karnal bunt, entries having less than 5 percent disease incidence are classified as resistant (Fuentes-Davila 1996). So this progeny also falls into resistant category. This set of derivatives also exhibited moderate to high seedling resistance to the Keylong (Indian) isolate of powdery mildew.

These observations suggested that the small alien arm translocated to the wheat chromosome or the alien acrocentric chromosome is carrying gene(s) for resistance to the three diseases viz., leaf rust, Karnal bunt and powdery mildew. There is equal possibility that gene(s) for resistance to one or two diseases are located on the alien translocated arm and the gene(s) for resistance to the rest of the disease(s) are located on the acrocentric chromosome. Further investigations are needed to clarify this point.

The observations presented here show that the wild tetraploid non-progenitor species, *Ae. triuncialis* (UUCC), could be a good source of resistance to wheat pathogens. This is the first report of transfer of useful resistance genes from this non-progenitor species though similar transfers have been made from other non-progenitor tetraploid species like *Ae.triaristata* (Bai et al. 1994) and *Ae. ovata* (Harjit-Singh and Dhaliwal 1996). The lack of transfer of useful genes from *Ae. triuncialis* may be due to the presence of chromosome with gametocidal genes reported in this species which is preferentially transmitted and kills the gametes without it (Tsujimoto and Tsunewaki 1985). The work to use these alien substitution/ translocation and addition lines for precise transfer of the alien resistance genes to wheat chromosomes through induced homoeologous pairing and use of

Table 4. Seedling reactions to individual pathotypes of leaf rust in the progeny from the cross *T. aestivum* cv. WL 711 carrying a homozygous translocation with short alien arm and a disomic addition of an alien chromosome

Tests	Pedigree	Seedling reaction to individual pathotype*		
		77-2	77-4	77-5
Test I	$BC_2$18-13-2-13 x	0	00N	–
	T. aestivum cv. WL 711	4	4	–
	Ae. triuncialis Acc.3549	0	00;	–
Test II	$BC_2$18-13-2-13-34	–	–	0
	$BC_2$18-13-2-13-37	–	–	0
	T. aestivum cv. WL 711	–	–	34
	Ae. triuncialis Acc.3549	–	–	0

* Seedling reactions recorded on 0 to 4 scale. The range of infection types produced on different plants by a given pathotype is presented without spaces or dashes between the figures.

molecular markers is in progress.

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Research article

Genetic variability and inheritance of grain dormancy in three white-grain wheats

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Summary

Breeding for high grain dormancy is the main approach for preventing preharvest sprouting in white-grain wheat (*Triticum aestivum* L.). Several white-grain sources express dormancy including Brevor (BV), Clark's Cream (CC) and Losprout (LS). This study examined the genetic expression and relatedness of dormancy among these sources. The $F_{2:4}$ and $F_{2:5}$ progenies of five populations were scored for dormancy in 1989 and 1993, respectively. The populations included crosses between BV, CC, and LS with Greer (GR), a nondormant variety, plus crosses of LS with BV and with CC. Germinability at 30 °C was determined to assess dormancy using a definitive germination percent (G%). Parent-offspring heritabilities included low to moderately high estimates varying from 0.17 to 0.63. The G% means of $F_{2:5}$ progeny of all crosses were not normally distributed. LS had higher dormancy than CC and BV while CC was more dormant than BV. LS has some dormancy genes in common with BV and CC and may have others which they lack. Using LS as the primary source of dormancy is justified. Testing large populations of several hundred F_2-derived individuals should facilitate recovering lines with dormancy similar to LS.

Key words: preharvest sprouting, *Triticum aestivum* L.

Introduction

Preharvest sprouting (PHS) of wheat (*Triticum aestivum* L.) is a serious problem in many major wheat production areas especially where white-grain varieties are grown. Several morphological and physiological traits influence the expression of PHS but grain dormancy is considered to be the most important process (Hong 1979; Li and Foley 1997). Wheats with white grain are considered to be more predisposed to PHS than red-grain varieties; yet a number of white grain wheat genotypes have appreciable grain dormancy and resistance to PHS (McCrate et al. 1982; Walker-Simmons 1987; Paterson and Sorrells 1990; DePauw et al. 1993; Mares 1993). The inheritance of dormancy among white-grain wheats has received considerable attention (Upadhyay and Paulsen 1988; Paterson and Sorrells 1990; Allan 1993; Anderson et al. 1993). Heritability estimates of dormancy have ranged from low (Paterson and Sorrells 1990) to medium (Upadhyay and Paulsen 1988; Allan 1993). Resistance has been reported to be normally distributed and quantitatively inherited (Paterson and Sorrells 1990; Allan 1993; Sorrells and Anderson 1996). Anderson et al. (1993) identified eight genomic regions controlling resistance to PHS in two populations. In contrast Mares (1996) reported that two independent recessive genes located on chromosome 3D controlled the high dormancy phenotype of AUS1408. The objective of this study was to examine the genetic expression of dormancy in three white-grain wheat sources and to determine whether they had similar or different genetic mechanisms controlling dormancy.

Materials and methods

Crosses were made among four white-grain varieties. Brevor (BV), Clark's Cream (CC) and Losprout (LS)

express grain dormancy. Greer (GR) expresses low dormancy (Walker-Simmons 1987; Hagemann et al. 1988). Progeny and parents were grown near Pullman, WA. In 1989, 180 to 200 $F_{2:3}$ lines of crosses CC/GR, BV/GR, LS/GR, LS/BV and LS/CC were grown in single row plots of 0.7m², about 50 seeds were sown per plot. Each of the four parents was included 3 to 4 times among the progeny of each population set. When harvest-ripe, the $F_{2:3}$ lines and parental plots of each population set were harvested and immediately threshed using a thresher having a rubber cylinder to minimize mechanical damage to the grain. The F_4 grain was directly stored at -5 °C to preserve dormancy. In 1993, $F_{2:4}$ progenies and their parents of the five populations were grown in a similar manner as the $F_{2:3}$ progenies; the F_5 grain was harvested, threshed and stored in the freezer. Between anthesis and harvest in 1989 and 1993 mean daily temperatures were 17.3 and 16.4 °C, respectively. Precipitation received between anthesis and harvest was 123 and 103 mm, in 1989 and 1993, respectively.

Grain germinability was measured at 30 °C, as the optimum temperature for assessing grain dormancy according to George (1967). For all progeny and parents, 50 grains were placed on blotting paper in a petri dish containing distilled water. Progeny and parents were replicated with 2 to 4 sub-samples of grains taken from each F_4, F_5 line and parental sample. The petri dishes were incubated in the dark. Once plates of GR commenced germination, four consecutive daily counts were made.

Two measures of dormancy were made. Germination % (G%) was the value attained by the fourth count. Rate of germination was estimated by the germination index (GI) using a method similar to that described by Hagemann and Ciha (1984). After count four, dishes were incubated at 16 °C for 7 days and samples having fewer than 40 viable seeds were not included in the results.

Data were analyzed using SAS procedure. The 5% LSD for each population set was used to determine the number of progeny having similar or different G% and GI values to the parental means of each population. Frequency distributions for G% and GI were made for the F_5 progeny of each cross. The F_5 generation was used because it was more homozygous than the F_4 and the 1993 season produced greater dormancy than the 1989 season. The progeny distribution patterns were tested for normality by chi-square test (Snedecor and Cochran 1967). Narrow sense parent-offspring heritability (h^2) estimates were calculated by the regression method (Falconer 1981) and by the standard unit method (Frey and Horner 1957).

Results and discussion

Although GI was measured only G% data are presented here. The two measurements were closely associated. Correlation coefficients between G% and GI ranged from 0.85 to 0.98 ($P<0.001$) among F_4 progeny in 1989 and 0.77 to 0.99 ($P<0.001$) among F_5 progeny in 1993. Hence using GI was unwarranted.

The four parents showed distinctly different ($P<0.05$) levels of dormancy when their G% values were combined over the five 1993 population sets. The overall G% means of LS, CC, BV and Gr were 11, 23, 39 and 90%, respectively. Among the five population sets, LS had G% means lower ($P<0.05$) than CC in all but one set where they were equal ($P>0.05$); LS had lower G% means than BV in all five sets. The G% values of CC were lower than BV in four sets and equal to BV in one set.

Heritability estimates for G% were moderate (0.30 to 0.54) based on the standard unit method and low to moderately high (0.17 to 0.63) based on the regression method (Table 1). Among crosses of dormant parents with nondormant GR, h^2 estimates were generally higher for crosses with LS and CC than for BV. Among crosses between dormant parents, the LS/CC cross had low heritability especially based on the regression method. These grain dormancy h^2 values are similar to those obtained by Upadhyay and Paulsen (1988), and by Allan (1993). Paterson and Sorrells (1990) obtained low regression h^2 estimates in a cross between a nondormant parent and CC.

The G% distribution patterns of F_5 progeny of crosses between the dormant parents with GR bore some similarities. None followed normal distribution

Table 1. Narrow sense heritability estimates of germination percent for six wheat populations

Population	Heritability estimates	
	Standard unit	Regression
Losprout/Greer*	0.49	0.58
Clark's Cream/Greer*	0.40	0.63
Brevor/Greer*	0.38	0.31
Losprout/Clark's Cream*	0.41	0.17
Losprout/Brevor*	0.54	0.60
Brevor/Clark's Cream**	0.30	0.31

*Based on F_4 (1989 season) and F_5 (1993 season) progeny G% means.

**Based on F_3 (1986 season) and F_4 (1987 season) progeny G% means.

(P<0.001). Rather they were skewed toward nondormancy. Very few progeny had G% means similar to their dormant parents. Among progenies of BV/GR, CC/GR, and LS/GR populations, 3, 1 and 2% had G% means similar to their respective dormant parent (P>0.05) versus 77, 73, and 55% of the progenies with G% means comparable to GR (Table 2).

The G% values of progenies of crosses between dormant by dormant parents also were not normally distributed (P<0.01). Progenies with high G% values were not recovered in the LS/CC populations. About 12% of the progeny had G% values higher than LS while 98% of the progeny had G% values similar (P>0.05) to CC (Table 2). Apparently the dormancy traits of LS and CC have some genetic similarities. A few progeny (4%) of the LS/BV population had higher G% means than BV (P<0.05). Yet no progeny of LS/BV and LS/CC populations had high G% values similar to GR suggesting that the three dormant parents had some genes in common controlling grain dormancy.

Table 2. Germination percent (G%) distributions of progeny expressed as percent of the total population for the six populations and G% means of parents and their respective populations

Germination percent	Population*					
	BV/GR	CC/GR	LS/GR	LS/BV	LS/CC	CC/BV**
	<——————————% of population——————————>					
0				2	4	
4				8	32	
8				15	30	
12				14	15	
16				13	7	
20			1	11	6	
24			0	6	4	
28			1	5	2	
32		1	1	4		2
36		2	0	4		0
40		4	1	3		2
44		2	1	4		2
48		1	3	4		3
52		3	1	2		4
56	1	8	1	1		8
60	2	6	5	3		4
64	1	5	7	1		5
68	1	5	9			7
72	4	11	4			10
76	3	12	10			10
80	11	11	11			9
84	8	11	11			11
88	18	11	14			11
92	20	6	10			9
96	26	1	6			2
100	5		3			1
Parent 1 $\bar{x}$	50	13	20	3	2	73
Parent 2 $\bar{x}$	97	82	96	37	12	71
Population $\bar{x}$	88	71	76	20	8	72
LSD (0.05)	14	20	18	14	14	11

*BV: Brevor, CC: Clark's Cream, LS: Losprout, GR: Greer. Parent 1 and 2 before and after /, respectively.

**Based on F_5 (1993 season) progeny G% means for all populations except CC/BV which was based on F_4 (1987 season) progeny G% means.

Earlier BV and CC were reported to have dissimilar genetic mechanisms controlling dormancy (Allan 1993). In that study GI was used to assess dormancy of F_3 and F_4 lines of a BV/CC population. The same conclusion was reached based on the distribution of G% values of F_4 progeny of this cross. Over 20% of the progeny had G% values lower than BV and CC while 12% of the progeny had G% values greater than both parents (Table 2).

Although it is likely that CC and BV differ genetically for grain dormancy, they may not have additional dormancy genes to those occurring in LS. Anderson et al. (1993) also did not recover segregants that transgressed both parents for PHS resistance in a cross between CC and a selection with moderate PHS resistance.

Among the three sources of dormancy, LS offers the most breeding potential because it expressed the highest level of phenotypic grain dormancy. Progeny numbers greater than studied here should be screened to recover an adequate proportion of selections with the LS dormancy phenotype.

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Research article

Morphological characterisation and evaluation of the subdivision of *Aegilops tauschii* Coss.

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Summary

In a study of plant morphology involving 54 accessions of *Aegilops tauschii* Coss. held at the John Innes Centre, Norwich, UK the subspecies *strangulata* and the ssp. *tauschii* varieties *typica, meyeri* and *anathera* were identified. Linear discriminant analysis, based on sixteen morphological characters, found that ssp. *strangulata* was morphologically distinct, being taller with a greater seed weight, prominent stem curvature and a rounded seed shape. The variety *anathera* was clearly separated on awn type and was, on average, shorter than the other accessions but var. *typica* and var. *meyeri* proved difficult to identify and were not easily distinguished by multivariate analysis. Consequently, the subdivision of *Ae. tauschii* on the basis of plant morphology appears to be reasonably valid. Seven of the accessions showed promising resistance to stripe rust infection, one was found to have a tough rachis and there was significant variation overall for yield related characters such as spikelet number, seed weight, leaf length and plant height.

Key words: *Aegilops tauschii*, intraspecific classification, genetic resources.

Introduction

Aegilops tauschii Coss. (syn. *Ae. squarrosa* L.) is a species of goat grass with a centre of distribution in the south Caspian area, spreading westwards to Turkey and eastwards to Afghanistan and China. Considerable morphological variation exists within the species and this allowed Eig (1929) to describe different subspecies and varieties of *Ae. squarrosa*. He divided the species into two subspecies, *eusquarrosa* and *strangulata*, and described three varieties within *eusquarrosa* — *typica, meyeri* and *anathera*. Hammer (1980), using the correct name *Ae. tauschii*, renamed ssp. *eusquarrosa* as ssp. *tauschii*. He also divided ssp. *tauschii* into varieties, with Eig's var. *typica* becoming var. *tauschii* and retaining var. *meyeri* and var. *anathera*. He also added var. *paleidenticulata*. However, these divisions were not formally recognised by van Slageren (1994) in his monograph of *Aegilops*. This was partly because of the existence of intermediate and hybrid forms (Kihara et al. 1965) but was also influenced by various molecular studies in which morphological variation failed to match or predict the genetic variation (Kim et al. 1992; Tsunewaki et al. 1991; Lubbers et al. 1991).

Ae. tauschii is the D genome donor of bread wheat (McFadden and Sears 1946) and is known to provide bread wheat with many qualities including bread making quality (Orth and Bushuk 1973), cold hardiness (Limin and Fowler 1981) and salt tolerance (Schachtman et al. 1992). Genetic variability within the D genome of wheat is much lower than it is within *Ae. tauschii* (Appels and Lagudah 1990; Lagudah et al. 1991) so the species offers great potential for wheat improvement. Utilisation of the species for wheat improvement is further aided by the ability of the chromosomes of *Ae. tauschii* and the D genome chromosomes of wheat to recombine naturally.

The species has been well collected in the past and many collections are now being evaluated for useful agronomic characters such as pest and disease

resistance (Murphy et al. 1997; Mujeeb-Kazi et al. 1996; Cox et al. 1995; Yildirim et al. 1995; Appels and Lagudah 1990).

The aim of this study was to characterise 54 accessions of *Ae. tauschii* held at the John Innes Centre, Norwich, UK and to determine whether Eig's divisions of the species were valid.

Materials and methods

The plant material studied represents 54 accessions of *Aegilops tauschii* held at the John Innes Centre, Norwich, UK. Four pots of each accession were sown in mid-December with five seeds in each pot. The plants were grown in an unheated glass-house until mid-April when they were planted out in experimental plots at the John Innes Centre. The four pots of each accession were planted intact to give a close group of up to 20 plants, with a one metre space between accessions.

An initial, visual assessment of variability within the collection identified 21 characters that were easy to score, appeared to vary between the accessions and made an obvious contribution to the plant phenotype. These were habit, height, stem curvature, peduncle length, leaf length, leaf width, glume colour, glume hairs, glume beak, upper glume apex, awn length, awn colour, awn type, brittle rachis, spike length, spikelet number, spike density, number of seeds per spikelet, seed weight, seed shape and seed colour. Susceptibility to stripe rust, *Puccinia striiformis*, was also scored. Two further characters, rachis segment length and spike yield, were derived from these characters. Well-defined descriptors were produced for each character. Many of these were based on standard descriptors recommended by IPGRI (IBPGR 1981) but for stem curvature, peduncle length, leaf width, glume beak, upper glume apex, awn length, awn type, seed shape, distance between spikelets and spike yield, new descriptor states had to be defined. A full descriptor list is given by Knaggs (1999). Each character was scored over a few days to allow a direct comparison of the character between accessions. Eight replicate measurements were taken for each measured character and a mean score calculated.

The descriptions of each subspecies and variety given by Eig (1929) were used to identify each accession. Linear discriminant analysis was then used to test the classifications given by the intraspecific identifications. The test works by calculating the smallest squared distance (Mahalanobis distance) to the group mean and then classifies the accession within that group. There is no need to make any assumptions about the underlying distribution of the data but the test does assume equal covariance matrices for each group. Sixteen characters were used for the analysis. Eight characters were discounted for various reasons: rust susceptibility is not a morphological character; leaf width, awn length, distance between spikelets and spike yield were too highly correlated with other characters; glume hairs and seed colour did not actually vary between the accessions and brittle rachis did not vary enough. The analysis was carried out using the Minitab computer program.

Results and discussion

The accessions proved to be highly variable for many of the characters. However, all accessions had red seeds and short, hook-like glume hairs. Other characters for which there was little variation included awn colour, brittle rachis, spike density, seed shape and the upper glume apex. All accessions had purple awns except for one with yellow awns and all but one had a brittle rachis. Three of the characters displayed a bimodal distribution in which a small number of accessions were clearly distinct. Stripe rust infection was severe on all but seven of the accessions and stem curvature was prominent on only four accessions. The calculated rachis segment length was derived from spike length divided by spikelet number. Most of the accessions gave lengths below 9.5mm but six accessions stood out with lengths of 10mm or more. Results for all the measured characters are summarised below in Table 1.

The descriptions given by Eig (1929) were used to identify different subspecies and varieties within the collection. The subspecies *strangulata* proved easy to identify; the glumes being only as long as they are broad, in contrast to the more elongated glumes of ssp. *eusquarrosa*. Dividing ssp. *eusquarrosa* into three varieties proved to be more difficult. The variety *anathera* is easily distinguished by the lack of all but terminal awns on the spike. The other two varieties are divided on dimensions of the spike with var. *typica* having thick spikes over 3.5mm wide and var. *meyeri* having slender spikes less than 3mm wide. Several accessions had spikes that were 3mm to 3.5mm thick, making it difficult to positively identify them at this level but, despite these problems, identifications were made so that the results could be compared by multivariate analysis of the morphological data. Five ssp. *strangulata*, fourteen ssp. *eusquarrosa* var. *anathera*, 27 ssp. *eusquarrosa* var. *typica* and eight ssp. *eusquarrosa* var. *meyeri* accessions were

Table 1. Summary of data for observed and derived characters from a study of 54 accessions of *Ae. tauschii.*

Character	Mean	Standard deviation	Minimum	Maximum
Plant height (cm)	40.74	6.22	29	59
Spike length (mm)	67.7	6.45	54	83
Spikelet number	8.38	0.86	6.4	10.3
Rachis segment length (mm)	8.15	1.01	6.25	10.44
Leaf length (mm)	67.11	16.78	37	104
Leaf width (mm)	5.69	0.79	4	7.3
Awn length (mm)	32.81	10.14	5	51
Peduncle length (cm)	18.81	2.29	14	24
Seed weight*(g)	0.17	0.04	0.11	0.27
Spike yield (g)	0.4	0.12	0.19	0.74

All values calculated from accession means, *Seed weight : weight of 10 seeds.

Table 2. Classifications of intraspecific divisions according to discriminant analysis of 54 accessions of *Ae. tauschii* based on 16 characters.

Group	Division after Eig (1929)			
	var. *anathera*	var.*meyeri*	ssp. *strangulata*	var. *typica*
Division by discriminant analysis				
var. *anathera*	12	0	0	1
var. *meyeri*	1	7	0	6
ssp. *strangulata*	0	0	5	0
var. *typica*	1	1	0	20
Total number	14	8	5	27
Number correct	12	7	5	20
Proportion correct	0.857	0.875	1.000	0.741

identified within the collection.

Discriminant analysis was carried out to see if the different subspecies and varieties of *Ae. tauschii* were well defined and to explore the relationships between them. Results of the first test (Table 2) demonstrate how well the subspecies and varieties describe morphological variability within the species. The results show that 44 out of the 54 accessions can be correctly identified according to the characters used in the analysis. This suggests that the subspecies and varieties can be used to describe morphological variability between the accessions fairly well but not completely accurately. The separation of ssp. *strangulata* seems to be the most well defined division; none of them were found to be misclassified by the analysis and no other accessions were reclassified as ssp. *strangulata*. The divisions within ssp. *eusquarrosa* were less well defined.

Relationships between the different subspecies and varieties can be discerned from the squared distances between them (Table 3). All of the squared distances between ssp. *strangulata* and the ssp. *eusquarrosa* divisions were over 35 suggesting that ssp. *strangulata* is clearly distinct. In contrast, the

Table 3. Squared distances between different subspecies and varieties of *Ae. tauschii* according to discriminant analysis of 54 accessions.

Group	var. *meyeri*	ssp. *strangulata*	var. *typica*
var. *anathera*	9.4587	51.9053	11.1923
var. *meyeri*		35.0564	2.1888
var. *strangulata*			35.3547

Table 4. Comparison of five selected characters between different subspecies and varieties of *Ae. tauschii* from a study of 54 accessions.

Division	Plant height (cm)*	Stem curvature†	Awn type†	Seed weight (g)*	Seed shape†
var. *typica*	42	straight	half-awned	0.17	oval
var. *meyeri*	42	straight	half-awned	0.16	oval
var. *anathera*	35	straight	tip-awned	0.16	oval
ssp. *strangulata*	49	prominent	half-awned	0.23	round

*Mean, †Mode

squared distance between varieties *meyeri* and *typica* is only 2.19 suggesting that these varieties are morphologically very close. Many of the accessions that stood out for stripe rust resistance, stem curvature and distance between spikelets proved to be ssp. *strangulata*. Other characters that could help to define each subspecies and variety were discerned by comparing character scores for accessions within each division. Some of these are contrasted in Table 4 and suggest that plant height, stem curvature, awn type, seed weight and seed shape are the most useful characters for distinguishing the four divisions. Differences in plant height and seed weight were confirmed as significant by a Kruskal-Wallis test (height $p<0.001$, seed weight $p = 0.012$).

Many species exhibit intraspecific variation and it can often be important to express this in some way (Stace 1989). The descriptions given by Eig (1929) have been widely used to identify the subspecies *strangulata* and the varieties *typica, meyeri* and *anathera* but how well do these describe morphological variation within *Ae. tauschii*? This study utilized a relatively small number of accessions but both subspecific and varietal divisions were evident, affording an opportunity to assess how robust these divisions are.

Results of the discriminant analysis suggest that ssp. *strangulata* forms a very distinct division within the species. The *strangulata* accessions were taller with heavier seeds, prominent stem curvature and a rounded seed shape. The analyses also suggest that within ssp. *eusquarrosa*, var. *anathera* is fairly distinct from var. *typica* and var. *meyeri*. The var. *anathera* accessions were clearly distinguished on awn type and also tended to be shorter than the other accessions. The varieties *typica* and *meyeri* were difficult to separate and also proved to be difficult to distinguish by multivariate analysis. Kihara et al. (1965) found intermediate and hybrid forms between var. *typica* and var. *anathera* and between ssp. *eusquarrosa* and ssp. *strangulata* and this may explain some of the problems of identifying the var. *typica* and var. *meyeri* accessions. The occurrence of intermediate forms could explain the difficulty in distinguishing the var. *typica* and var. *meyeri* accessions.

Ideally, effective divisions should be distinct both morphologically and genetically. The decision by van Slageren (1994) not to formally recognise any intraspecific classification within *Ae. tauschii* was heavily influenced by the results of Kim et al. (1992). In their study, based on a highly conserved region of ribosomal-DNA, they were unable to find consistent polymorphism that distinguished ssp. *strangulata* accessions. RFLP studies by Tsunewaki et al. (1991) and Lubbers et al. (1991) found close similarities between ssp. *eusquarrosa* var. *meyeri* and ssp. *strangulata*; and a recent molecular study by Dvořák et al. (1998) found evidence of gene migration between the different divisions in accessions from the south-west Caspian area of Iran. It is evident from these studies that morphological variation within *Ae. tauschii* can not always be used to predict genetic variation at the molecular level because phenotypic divisions become blurred due to hybridisation and hence the occurrence of intermediate forms (Kihara et al. 1965). There is, however, evidence of genetic distinction at the molecular level between the different subspecies in Transcaucasia (Dvořák et al. 1998), yet the same study also failed to find any between var. *typica* and var. *meyeri*. This would appear to be consistent with the lack of a clear morphological distinction between them in this study.

The intraspecific classification for *Ae. tauschii* based on plant morphology appears to be of value. In particular, the distinction between the subspecies *strangulata* and *eusquarrosa* is an easy one to make; the ssp. *strangulata* accessions were easy to identify and were well separated by multivariate analysis. There is also some molecular evidence to support the separation (Dvořák et al. 1998). This conclusion is different to the one by Kim et al. (1992) but it is worth pointing out that both studies have involved a limited

number of ssp. *strangulata* accessions. The different varieties within ssp. *eusquarrosa* were less easy to distinguish, especially varieties *meyeri* and *typica*. The lack of a difference between these two suggests that this division is unsound to a certain degree and this is supported by some molecular evidence (Dvořák et al. 1998). The variety *anathera* may, however, be distinct enough to retain.

Promoting greater utilisation of material is now a major task for genebanks. Other collections of *Ae. tauschii* have been extensively evaluated over the past ten years and the species is being increasingly used for wheat improvement (Murphy et al. 1997; Mujeeb-Kazi et al. 1996; Cox et al. 1995; Yildirim et al. 1995; Appels and Lagudah 1990). Assessing the breeding potential of the material was obviously beyond the scope of this study but characters of interest such as height, spikelet number and seed weight have been scored. In addition a preliminary evaluation of resistance to stripe rust revealed that seven of the accessions, including all five of the ssp. *strangulata* accessions, showed resistance to the prevailing natural population of stripe rust infection. This result is consistent with other studies in which ssp. *strangulata* accessions showed greater disease resistance (Yildirim et al. 1995; Cox et al. 1995; Appels and Lagudah 1990). One accession was found to have a tough rachis and there was significant variation within the collection for yield related characters such as spikelet number, seed weight, leaf length and plant height. Further evaluation is essential to reveal the range and potential of other agronomically important characters.

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Identification of amphiploid between *Triticum durum* cv. Ailanmai native to Sichuan, China and *Secale africanum*

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Summary

An amphiploid between *Triticum durum* native to Sichuan, China, and *Secale africanum* was evaluated by cytological observation, seed storage protein electrophoresis analysis and disease resistance surveys. Feulgen staining and Giemsa-C banding of somatic metaphases indicated that the nucleoli from *S. africanum* were frequently suppressed in the amphiploid. APAGE and SDS-PAGE showed that most gliadin and glutenin of both parents were observed in the endosperm of the amphiploid with codominant expression. Inoculated by the stripe rust and powdery mildew isolates, the amphiploid totally expressed the resistance from *S. africanum*. It is concluded that the amphiploid can be used to triticale and wheat breeding for quality and diseases resistances.

Key words: *T. durum*, *S. africanum*, amphiploid, giemsa C-banding, seed storage protein

Introduction

Wheat cultivar improvement is dependent on a continued supply of genetic variability. The tribe Triticeae offers a vast genepool in which most agronomically interesting traits including some not existed in wheat are available. Genus *Secale* consists of cultivated rye (*S. cereale*) and five annual or perennial wild species (Love 1984). Above all, *S. cereale* had provided many desirable genes, such as those for resistance to many biotic and abiotic stresses, to world wheat breeding. Studies on gene transfer involving the wild species of *Secale* were mainly carried out on *S. montanum* and the substitution, translocation lines between wheat and *S. montanum* were obtained (Miller 1973; Montero et al. 1986; Cuadrado and Jouve 1995). But reports on other wild *Secale* species, such as *S. africanum* were rather limited (Sharma and Gill, 1983).

The production of amphiploid is an important step for successful gene introgression, and the amphiploid also allows more 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 used. A tetraploid wheat line, *Triticum durum* cv. Ailanmai, native to Jianyang of Sichuan, China, was reported to have high crossability genes with alien species (Jiang et al. 1988; Pen et al. 1998; Liu et al. 1999). The amphiploid between Ailanmai and *Triticum tauschii* was obtained for successfully transferring the novel tolerances to preharvest sprouting from *T. tauschii* to wheat (Lan et al. 1997). In order to utilize the novel gene from *S. africanum* (2n=14; genome R^aR^a), amphiploid between Ailanmai and *S. africanum* was developed through colchicine treated hybrid F_1, and the morphological observation of the amphiploids were also carried out (Jiang et al. 1992). In present paper, we attempted to identify amphiploid (2n=42, $AABBR^aR^a$) and describe the gene expression of *S. africanum* in the amphiploid involving nucleolus, seed storage protein

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and disease resistance. Moreover, the breeding value of the amphiploid to wheat and triticale was also discussed.

Materials and methods

T. durum cv. Ailanmai, a tetraploid wheat landrace of Jianyang, Sichuan, was collected and maintained in Triticeae Research Institute, Sichuan Agricultural University, China. The accession of *S. africanum* was obtained from Missouri Botanical Garden, USA. The fertile amphiploid between *T. durum* cv. Ailanmai and *S. africanum* were kindly provided by Prof. Jiang H. R. of Sichuan Agricultural University (Jiang et al. 1992). Wheat line Moulin with subunit 17+18 encoded by *Glu-B1* was obtained from Prof. P. I. Payne of Plant Breeding Institute, Cambridge, U.K..

Chromosome counts were performed after the Feulgen squash method and the silver-staining procedure was according to the method of Lacadena et al. (1984). Giemsa-C banding technique was described by Ren and Zhang (1995).

Acid polyacylamide gel electrophoresis (APAGE) separation of endosperm gliadin was conducted following the method reported by Cook (1987). According to the procedure of Ng and Bushuk (1987), glutenin subunits were separated by sodium dodecyl sulphate-polyacrylamide gel electrophoresis (SDS-PAGE). The determination of HMW-glutenin subunits was described by Payne and Lawrence (1983) with the bread wheat lines Chinese Spring and Moulin as references.

The amphiploid with 2n=42 were evaluated on their seedling resistance to powdery mildew and adults-plant to stripe rust with reference to its parents. The plants are grown at the field in Dujiangyan, Sichuan, where has a favorable environment for stripe rust and powdery mildew epidemics. The adult plants were inoculated by new physiological strains CYR-30 and CYR-31 of wheat stripe rust in China, provided by Plant Protection Institute, Sichuan Academy of Agricultural Sciences. The powdery mildew isolates collected from Pingshan, Sichuan, were applied to inoculate the seedling. Stripe rust and powdery mildew response observation referred to Ma et al. (1995) and Zeller et al. (1993), respectively.

Results

Feulgen staining indicated that the euploid of the amphiploid with 2n=42 chromosomes contained only two pairs of chromosomes with nucleolus organizer

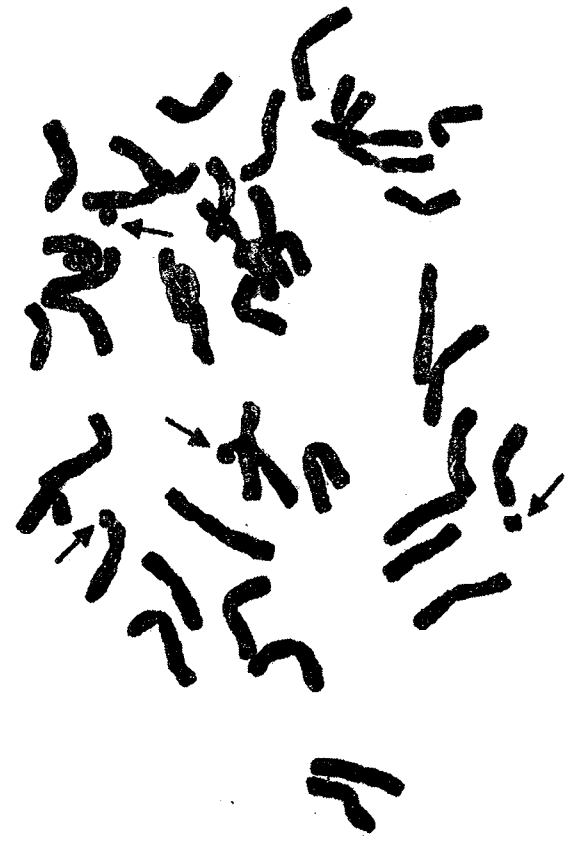

Fig. 1 Feulgen stained karyotype of amphiploid (2n=42) with four nucleolus organizer regions (arrow).

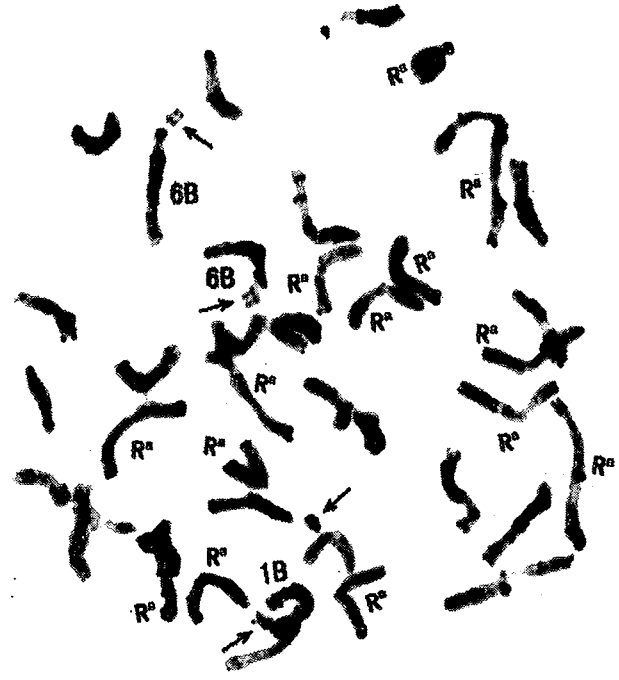

Fig. 2 C-banded karyotype of amphiploid (2n=42). Arrows showed the chromosomes with nucleolus organizer region and R^a marked the 14 *S. africanum* chromosomes.

region (NOR) (Fig. 1). It was also supported by the results of silver-staining, which indicated that 4 stained nucleolus organizer regions (Ag-NORs) were observed in the metaphase cells analyzed (data not shown). Theoretically, the parents of the amphiploid would carry three pairs of chromosomes with NOR, in which two pairs from *T. durum* cv. Ailanmai, one pair from *S. africanum*. Furthermore, Giemsa-C banding clearly indicated that four NOR existed in the chromosomes 1B and 6B from *T. durum* (Fig. 2), when compared with the standard C-banding karyotypes (Gill et al. 1991). Thus, the nucleoli from *S. africanum* chromosomes did not express in the amphiploid.

The composition of glutenin was analyzed by SDS-PAGE (Fig. 3). The high molecular weight glutenin subunits (HMW-GS) of Ailanmai contained subunit 2* of *Glu-A1*, and two close bands existed between subunits 7 and 8 referred by *Glu-B1* of Chinese Spring. The slow-moving band is stronger which would be the x-type of the subunit, and the fast-moving band had the same mobility as subunit 18 contained in the wheat line Moulin. In comparison with the description of HMW-GS in *T. durum*, it is concluded that two bands belong to *Glu-B1IV*, which were named as subunits 23 + 18 by Branlard et al. (1989). Glutenin band slightly faster than subunit 2 in *S. africanum* with the same mobility as strong bands encoded by *Sec-3* of *S. cereale* (Shewry and Miller 1983), was quite weak. By observing the band pattern of the amphiploid, it is easily concluded that both HMW and low molecular weight (LMW) glutenin in the amphiploid overlapped those of its parents.

APAGE of seed gliadin revealed that the strong bands of *S. africanum* concentrated on aggregated zone and ω-secalin zone (Shewry and Miller 1983), and the bands in γ, β and α zone are quite weak (Fig. 4). Most of the bands in amphiploid overlap in the corresponding zones from that in *T. durum* and *S.*

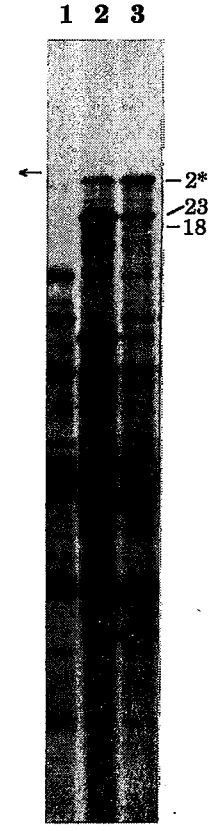

Fig. 3 Seed glutenin of *S. africanum* (lane 1), *T. durum* (lane 2) and amphiploid (lane 3) separated by SDS-PAGE. Arrow indicates the bands from *S. africanum*.

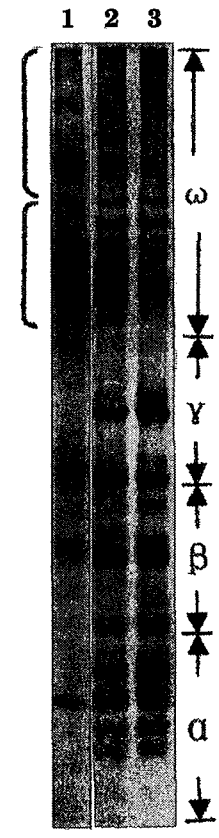

Fig. 4 Seed gliadin of *S. africanum* (lane 1), amphiploid (lane 2) and *T. durum* (lane 3) separated by APAGE. Left brackets indicated the aggregated secalin (up) and ω-secalin (down) of *S. africanum*.

Table 1. Disease reaction of *T. durum* - *S. africanum* amphiploid and its parents when inoculated with stripe rust and powdery mildew isolates.

Line	Field response of stripe rust		Seedling response of powdery mildew	
	FDR[a]	AUDPC[b]	Infection grade[c]	Resistance reaction
S. africanum	0R	<1	0	R
Ailanmai	10M	8.0	9	S
Amphiploid	0R	<1	0	R

[a]Final adult-plant disease rating.
[b]Area under the disease progress curve (AUDPC) with references to susceptible check line 'Mingxian169' of China.
[c]Infection grade based on 0–9, where 0 = no visible disease sympton and 9 = 50-100% leaf area covered with mycelium.

africanum. But the aggregated secalin zone from *S. africanum* and weak gliadin bands in γ and β zones from both parents were not observed in those of amphiploid.

Resistance investigation of *T. durum-S. africanum* amphiploid were conducted with references to its parents when inoculated by powdery mildew isolates and stripe rust races (Table 1). *S. africanum* showed high resistance to these tested isolates in seedling and adult plants, respectively, *T. durum* showed high susceptible to powdery mildew isolates in seedling, but show intermediate to stripe rust races in adult plants. But the amphiploid with 2n=42 displayed high resistance to both diseases. These results indicated that the disease resistance from *S. africanum* was totally expressed in the amphiploid background.

Discussion

A stable amphiploid is a permanent resource to combine the genetic variations of donor species. The amphiploid between *T. durum* cv. Ailanmai and *S. africanum* contained the valuable genes from AABB genome from *T. durum* and R^aR^a genome of *S. africanum*. It was a new type of hexaploid triticale and can serve as a novel germplasm for triticale improvement. By crossing of the amphiploid with wheat, it can also provide desirable genes to wheat breeding.

To transfer the available genes from alien species, the characterization of alien chromatin in wheat background was vital. Giemsa-C banding techniques made chromosome identification fast, reliable and economical (Gill et al. 1991; Jiang et al. 1994). C-banded mitotic metaphase cell allows distinguishing the chromosomes from *T. durum* and *S. africanum* in the amphiploid (Fig. 2). Above all, five pairs of chromosomes with strong telomeric heterchromatins derived from *S. africanum* (Bennett et al. 1977) were easily observed, and the other two pairs of chromosomes with their characterized C-bands also were longer than the chromosomes of *T. durum* (Fig. 2). Therefore, in the process of gene transfer, the band pattern of *S. africanum* can be used to identify the introgression of *S. africanum* chromatin in wheat background.

The endosperm storage protein have been considered as useful genetic marker and utilized for gene pool evaluations, cultivar identification and chromosome markers for directed genetic manipulation (Konarev et al. 1979). In the amphiploid or F_1 hybrids, the electrophoresis patterns were often additive, with bands from both parents. The present study showed that most gliadin and glutenin from *T.durum* and *S. africanum* were expressed in the endosperm of the amphiploid and these band patterns also confirmed the genealogy of the amphiploid. The gliadin patterns of *S. cereale* were used to trace the rye chromosome 1RS in wheat background (Sozinow et al. 1987). Therefore, the similar gliadin band pattern of *S. africanum* can also used as genetic marker to detect the corresponding chromosome in gene transfers to wheat.

The variation in HMW glutenin subunits of wheat accounted for most of the variation in bread making qualities (Payne et al. 1987). Determination of HMW glutenin subunits was of importance to evaluate its quality contribution. Recently, we transfer the subunit 23+18 of *Glu-B1* from a hexaploid triticale to Sichuan wheat. The result indicated that the advanced lines with this subunits appear relatively higher protein contents and SDS sedimentation volume than recipient wheat with subunit 7+8 by *Glu-B1*. It is likely that the subunits 23+18 has a good influence for bread-making quality of wheat. The amphiploid expressed the subunits 2* of *Glu-A1* and 23+18 of *Glu-B1* from its Ailanmai parents. It is thus to note that the amphiploid can be used as a resources to exploit the desirable glutenin subunits to wheat quality improvement.

The resistance expression of wheat-alien amphiploid is mostly dependent on genotype of wheat. The rust and powdery mildew resistance from *S. cereale* were quite easily expressed in its amphiploid with *T. durum* (Singh and Sethi 1994). The present results also showed that the stripe rust and powdery mildew resistance from *S. africanum* were expressed in this amphiploid. But the studies on another amphiploid involved different tetraploid wheat did not express the resistance from their donor *S. africanum* (Yang et al. unpublished). Therefore, on the utilization of alien resistance, the genotype of wheat should be considered in order to provide a wheat background for its resistance expression.

In addition, the different resistance genes existed in chromosomes of different rye-derived, such as gene *Pm8* in 1R of Petkus rye, but *Pm17* in that of Insave (Heun and Friebe 1990). *S. africanum* was a species different from cultivated rye. It is possible that powdery mildew or stripe rust resistance genes in *S. africanum* may be different from the genes in *S. cereale*. The amphiploid can be used as a new germplasm for improving the resistance of wheat.

The bread-making quality of Sichuan, China was very poor and the *Glu-1* quality score for the composition of HMW-GS in Sichuan cultivars was

rather low (Li and Wang 1998; Yen 1999). Moreover, stripe rust and powdery mildew resistance provided by 1RS/1BL translocation chromosome, widely existed in 70 percent of Sichuan wheat cultivars, was overcome (Chen and Ren 1996). Searching for novel germplasm resource was of importance for Sichuan wheat breeding. Based on the present study, the HMW-GS 2* of *Glu-A1* and 23+18 of *Glu-B1* in the amphiploid of *T. durum* cv. Ailanmai and *S. africanum* can be transferred to Sichuan wheat in order to increase the diversity for the composition of *Glu-1* subunits in Sichuan released varieties. Meanwhile, the stripe rust and powdery mildew resistance of *S. africanum* in the amphiploid can also be utilized to improve the disease resistance to new races. Therefore, the amphiploid can serve as a new source for Sichuan wheat breeding for quality and resistance.

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Research article

Genetic variation in allelopathic activity of wheat (*Triticum aestivum* L.) genotypes

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Summary

Exploiting genotypic variation of allelopathic activity of crops for controlling weeds is relatively a younger area of research. Barley, rice and sorghum are some of the crops which have been proved promising in this regard. However, there seems to be no report on genetic variation in allelopathic activity of wheat genotypes, and its possible application for weed control. Therefore, a suitable bioassay was developed and various wheat genotypes were evaluated for their allelopathic activity against *Avena ludoviciana*. Tested wheat genotypes showed a significant genetic variation ranging between +10% to -30%. Varieties like Ghods, Khazar-1 and PI 4512 caused 27.9%, 28.3% and 30.2% reduction, respectively, in dry weight of the test weed. Interestingly, cultivars Bezostaya-1, Naveed and Niknejad expressed positive allelopathy, and promoted the dry weight of Avena by 6.6%, 10.9% and 10.4%, respectively. An increase in the wheat seed density improved the allelopathic inhibition of the test weed but did not demonstrate any autotoxicity. There was no correlation between the growth and allelopathic activity of different wheat varieties. Bezostaya-1, a variety with maximum height (35.1 cm) caused a 6.6% promotion in Avena dry weight. Whereas cv. Inia with a comparatively lesser height (26.3 cm) inhibited the test weed by 28.3%. These results demonstrate that some of the wheat genotypes carry genes for allelopathic traits (both inhibitory and promoters) which can be used for breeding wheat varieties with allelopathic activity for controlling weeds. Genetic analysis of wheat varieties with significant allelopathic activity and studies on inheritance pattern of this trait are suggested.

Key words: *Triticum aestivum*, wheat, allelopathy, genetic variation, weed control

Introduction

One of the most prevalent interference mechanisms among plants is competition for the essential factors required for their normal growth and development. However, in recent times it has been repeatedly pointed out that release of chemical compounds from living plants and/or their residues could also be a strong way of interference. It was Molisch (1937) who coined the term 'allelopathy' to describe this kind of interference. It denotes that body of scientific knowledge which concerns any direct or indirect harmful or beneficial effect by one plant on another (including micro-organism) through production of chemical compounds (allelochemicals) that escape in to the environment (Rice 1984).

Allelopathic property of plants can be utilized for weed control as allelochemicals suppress plant growth, and regulate species diversity (like herbicides) in the habitat of the producer plant. Further, because majority of the naturally occurring allelochemicals is

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rapidly degraded into non-toxic compounds, these are considered eco-friendly as compared to the commercially used herbicides. One of the ways to use crop allelopathy for weed control is to employ residues of crop plants like rye (*Secale cereale*) and sorghum (*Sorghum bicolor*) (Putnam and DeFrank 1979; Shilling et al. 1985). Another way to use allelopathy for weed control is to use purified allelochemicals or their derivatives as weed killers. Alfa-terthienyl, a potent phototoxin, isolated from members of Asteraceae acts as a contact herbicide on broad-leaf weeds (Lambert et al. 1991). Several other allelochemicals like, ailanthone, caffeine, cineole, citronellol, mimosine and azadirachtin have been identified with weed controlling activity (Rizvi et al. 1999).

Due to environmental concerns attention has recently been focussed on a new aspect of crop allelopathy which can be used for weed control. There is variable degree of allelopathic activity by different genotypes of a particular crop species against weeds. It has been found that amongst the several accessions of cucumber (*Cucumis sativus*), one inhibited *Brassica hirta* by 87%, and 25 others by 50%. Out of the tested 100 accessions of sorghum, 25 inhibited the germination and growth of *Amaranthus retroflexus* by 82% and 85%, respectively (Rice 1995; Weston 1996). However, the most convincing work on selection of crop germplasm with allelopathic activity against weeds has been done on rice. Dilday et al. (1998) evaluated more than 16000 accessions of rice for allelopathic control of *Heteranthera limosa* and *Ammannia coccinea*. Out of these, about 3.4% accessions showed considerable activity. Cultivars like Taichung Native 1, Johna 349, Masrai, CR52-3, IR788-16-1-1-1 and S 12 DZK etc. inhibited the test weeds ranging from 60% to 90 %. So far no attempt seems to have been made to evaluate the allelopathic potential of wheat genotypes. However, allelopathic effects of wheat straw against plants including weeds have been demonstrated (Perez 1990; Alsaadawi et al. 1998). This indicates that wheat genotypes are capable of producing allelochemicals, which can regulate the growth of other plants. Therefore, possibility does exist to identify wheat varieties/ genotypes with major gene(s) for allelopathic activity against weeds. Present study was initiated to study the genetic diversity for the allelopathic trait in bread wheat genotypes.

Materials and methods

Thirty-four bread wheat genotypes with diverse growth habit and plant height out of modern improved cultivars and landraces were selected for the evaluation of their allelopathic trait against the most prevalent weed, *Avena ludoviciana*. For this, different bioassays were tried, and the 'greenhouse-box-assay' was found to be reliable, efficient and inexpensive. For the assay, plastic boxes (18 x 12 x 7 cm) filled

Table 1. Evaluation of allelopathic potential of various bread wheat genotypes in terms of their effect on *Avena ludoviciana*

Triticum aestivum		*Avena ludoviciana* (Percent effect over control)	
Genotypes	Height/plant (cm)	Dry weight / plant	Height / plant
Adla	22.5	-10.0*	-11.6*
Alamoot	26.1	-5.1	-0.9
Alburz	25.1	-18.8*	-4.3
Arvand	24.7	-11.9*	-3.5
Atila-5	23.8	-11.9*	-2.4
Atrakh	23.6	-3.9	+0.4
Azadi	25.4	-15.9*	+5.6
Baiyat	28.1	-25.8*	-11.9*
Bezostaya-1	35.1	+6.6*	-2.2
Darab-2	28.1	-13.6*	0.6
Ghods	27.8	-27.9*	-4.7
Inia	26.3	-28.3*	-29.8*
Jenab	28.8	-20.1*	-28.9*
Karaj-1	30.7	-13.2*	-16.8*
Karaj-3	26.3	-24.1*	-22.3*
Kavkaz	32.4	-25.7*	-20.7*
Kaveh	26.4	-13.8*	-21.2*
Khazar-1	30.7	-28.3*	-23.7*
Khiramand	29.2	-9.9	-22.2*
Naveed	30.8	+10.9*	+14.0*
Niknejad	33.1	+10.4*	+3.6
Qazi	26.1	-19.3*	-9.9
Safed Bofghi	32.7	-16.2*	-13.7*
Surkhu	29.9	-26.1*	-27.4*
Zarin	25.9	-7.8	-13.7*
PI -2064	25.4	-5.8	-3.2
PI -2082	27.4	-7.3	-4.5
PI -2159	27.8	-10.2*	-4.8
PI -2202	26.9	+8.3	-3.2
PI -2391	26.2	-11.9*	-9.1
PI -2474	28.8	-8.5	-7.9
PI -2578	29.9	-12.4*	-3.7
PI -2580	28.8	-4.8	+11.4*
PI -4512	26.5	-30.2*	-29.4*

* Significant at 5% level

with 1 kg greenhouse soil, a guide-plate (having 3 rows at 4 cm distance with 8 holes in each row at 2 cm interval) and a steel borer (adjusted to make 1.0 cm deep holes for seed sowing) were used. Fourteen seeds of wheat and seven of test weed were maintained in the two border rows and the central row, respectively. Twenty-four hours prior to sowing, each box was supplied with 150 ml of distilled water. Four replicates for each variety were randomised in a greenhouse maintained at 20°C and 12 hour light (2800 lux ca) / dark cycle. Each box was watered with 70 ml of distilled water/day. To see if the wheat density has any effect on its allelopathic activity, the number of wheat seeds were doubled in one of the experiments. Heights of Avena plants were recorded on every 4th day. Plant height (cm) above the ground and dry plant weight (g) of Avena, and height of wheat plants were recorded on day 26th after sowing, when the experiment was terminated. Percent inhibition (–) or promotion (+) in plant height and dry weight of Avena were calculated. Duncan's multiple range test was employed for comparing the means at 5% level. Variation in the allelopathic activity of different wheat varieties and correlation (using linear regression) with their vigour (height) was established.

Results and discussion

Evaluated wheat varieties showed significant genetic variation in their allelopathic activities ranging between +10.9% (cv. Naveed) ~ –30.2% (cv. PI 4512) in terms of their effect on increase or decrease in dry weight of Avena, and +14.0% (cv. Naveed) ~ –29.8% (cv. Inia) on its height. Nine of the evaluated varieties exhibited more than 20% inhibition of the dry weight of the test weed. However, seven genotypes/varieties e.g. Baiyat, Ghods, Inia, Kavkaz, Khazar-1, PI 4512 and Surkhu caused 25.8, 27.9, 28.3, 25.7, 28.3, 30.2 and 26.1% reduction in the dry weight, respectively, indicating the existence of gene(s) for allelochemical(s) production which in turn inhibit the growth of *A. ludoviciana*. Interestingly, Bezostaya-1, Naveed and Niknejad varieties exerted a positive allelopathy, and promoted the dry weight of Avena by 6.6, 10.9 and 10.4%, respectively (Table 1).

When the number of wheat seeds were increased in the assay boxes, the extent of inhibition was further enhanced. However, varieties, Bezostaya-1, Naveed and Niknejad that had positive effects on the growth of the test weed at low seed density, showed some inhibitory effect but there was no significant change in the height of wheat plants when grown with increased density (Table 2). This indicates that the adverse or inhibitory effect of wheat varieties on Avena is mediated through the release of chemical inhibitors (allelopathy) rather than simple competition, otherwise growth of wheat plants would have also been affected. Furthermore, none of the wheat varieties showed any autotoxicity.

It is generally presumed that plant species with high growth vigour are better competitors of weeds and are allelopathically superior. However, we could not get any correlation between the growth (height) and allelopathic activity of different wheat varieties. Bezostaya-1, a variety with maximum height (35.1 cm) caused a 6.6% promotion in Avena dry weight whereas Inia with a comparatively less height (26.3 cm) inhibited the test weed by 28.3%. Naveed and

Table 2. Effect of seed density on the allelopathic potential of wheat genotypes

Parameters	*Triticum aestivum*		*Avena ludoviciana* (Percent effect over control)			
	Height/plant (cm)		Dry weight/plant		Height/ plant	
Genotypes	Single density	Double density	Single density	Double density	Single density	Double density
Atrakh	23.6	24.9	-3.9	-41.7*	+0.4	-37.5*
Bezostaya-1	35.1	35.5	+6.6*	-42.5*	-2.2	-26.9*
Darab-2	28.1	28.5	-13.6*	-41.4*	+0.6	-23.7*
Jenab	28.8	26.1	-20.1*	-41.7*	-28.9*	-21.9*
Khazar-1	30.7	25.6	-28.3*	-45.9*	-23.7*	-35.7*
Naveed	30.8	30.1	+10.9*	-39.8*	+14.0	-23.9*
Niknejad	33.1	32.0	+10.4*	-42.3*	+3.6	-17.6*

*Significant at 5% level

Niknejad varieties had almost similar plant heights with those of Karaj-1, Karaj-3 and Khazar-1 but the earlier ones had a 10% promotory effect while the latter varieties inhibited the test weed by 13.0% to 28.3% (Fig. 1). These results indicate that in this case

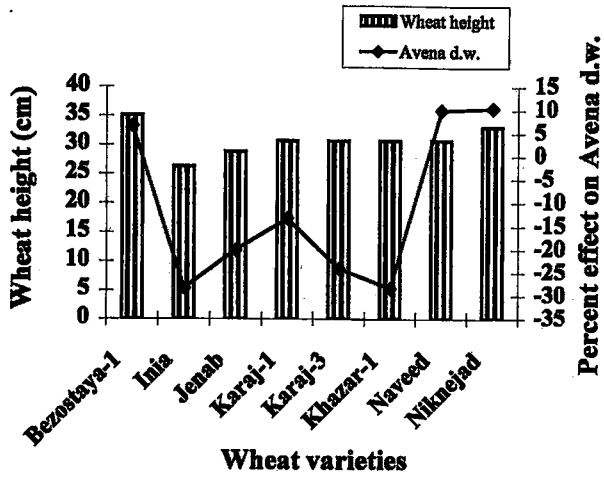

Fig. 1. Relationship between wheat growth (height) and change in dry weight of *A. ludoviciana*

the allelopathic activity is independent of the early growth vigour of wheat varieties. In order to see any possible genetic variation in allelopathic activity of wheat genotypes at different growth stages (the age of plants) their effect on dry matter and plant height of Avena was studied. All the allelopathic varieties showed a similar trend of inhibition and the allelopathic activity increased with the advancing age of wheat seedlings (Figs. 2 and 3). Out of the tested wheat genotypes, Inia, Jenab, Karaj-3, Kavkaz, Khazar-1, PI -4512 and Surkhu showed more than 60% inhibition in the growth rate of Avena during 22nd to 26th day (Table 1, Figs. 2 and 3). PI-4512 caused the maximum inhibition (74.6%). Inhibition caused by different wheat genotypes can be attributed to the presence of major gene(s) controlling the production of allelochemicals like acetic, propionic, butyric, vanillic, syringic, p-coumaric, p-hydroxybenzoic and hydroxamic (2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one) acids which are known to cause reduction in plant growth, total biomass and act as herbicide (Tang and Waiss 1978; Perez 1990; Alsaadawi et al. 1998).

These preliminary results indicating great genetic variability in bread wheat genotypes for allelochemical production can be of paramount importance in view of the success achieved with allelopathic rice (Olofsdotter 1998), in the control of weeds and reduction in the use of herbicides. These results have also revealed that it is not the early growth vigour of some of the wheat genotypes, helpful in the control or reduction of weed population in wheat field, rather it is the ability of wheat genotypes to produce certain types of allelochemicals which suppress, inhibit or kill the competing weed. Possibility of identifying strongly allelopathic wheat varieties possessing major gene(s) to control weeds under field condition lies in screening a large number of genetically diverse genotypes and their wild relatives. Putnam and Duke (1978) have suggested that wild types and progenitors of existing crops may have possessed high allelopathic activity and this character was reduced or lost as they were hybridised and selected for other useful characteristics. The facts that allelopathic activity is genetically inherited (Panchuk and Prutenskaya 1973), and is caused in synergistic manner involving a complex of chemicals

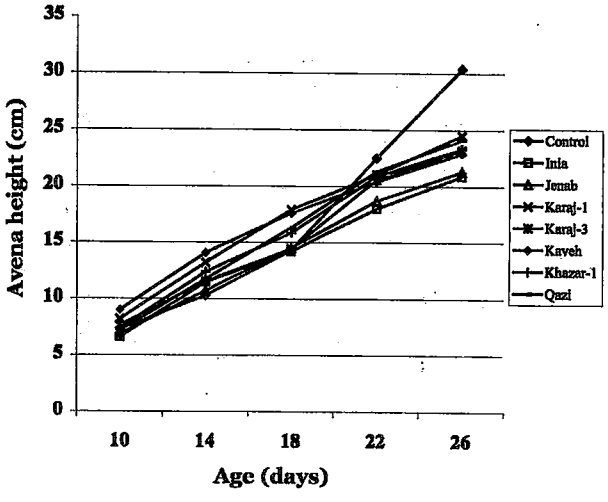

Fig. 2. Effect of wheat genotypes on growth of *A. ludoviciana*

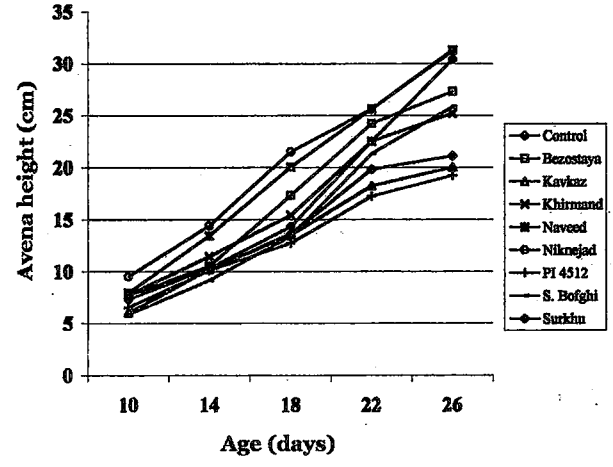

Fig. 3. Effect of wheat genotypes on growth of *A.ludoviciana*

(Rizvi et al. 1992) which is produced under the action of more than one gene (Alborn et al. 1992) make the wheat allelopathy research very attractive. However, unfortunately in wheat which is one of the most widely researched and grown crop throughout the world, the research on its allelopathic trait is negligible. These studies clearly indicate the possibilities of exploiting allelopathic trait through genetic manipulations while breeding new wheat cultivars which will help the farmers in reducing the cost of production and at the same time the approach will be environment friendly.

Allelopathic crop varieties may not only lead to the reduction in the use of herbicides, these could also be used as a 'source' of allelopathic trait for breeding crops with strong 'weed resistance'. However, evaluation of a large number of genetically diverse wheat genotypes, identification of useable gene(s) controlling allelochemicals production, inheritance, their mode of action and non-target toxicity, are pre-requisite before allelopathic wheat genotypes can be used commercially. Experiments are in progress on the above mentioned aspects.

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Research information

A synthetic zhukovskyi wheat

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A new hexaploid wheat was discovered by Menabde and Eritzjan in 1957 and named *Triticum zhukovskyi* by Jakubziner (1959). Bowden (1959) suggested that this species arose through chromosome doubling of an F_1 between *Triticum timopheevi* L. and *Triticum monococcum* L. Upadhya and Swaminathan (1963) concluded that the genomic constitution of *T. zhukovskyi* was AAAABB, based on study of karyotypes of *T. zhukovskyi*, *T. timopheevi*, *T. monococcum* and *Aegilops squarrosa*. Subsequently, Kimber and Sears (1983) suggested that *T. zhukovskyi* has the genomic constitution AAAAGG. More recently Dvorak et al. (1992) concluded that the genomic constitution of *T. zhukoskyi* is AAA^mA^mGG, based on the repeated nucleotide sequence analysis of *T. timopheevi*, *T. monococcum* and *T. urartu*. We created an amphiploid of *T. timopheevi-T. monococcum* and the morphological characteristics of the amphiploid supported the suggestion that *T. zhukovskyi* originated from a natural cross of *T. timopheevi* and *T. monococcum* through chromosome doubling.

An accession of *T. timopheevi* (PI 290518) used as a female parent, was crossed with an accession of *T. monococcum* (PI 352267). At 14 days after pollination, hybrid caryopses were harvested and sterilized in 20% bleach ($CaCl_2$), followed by washing four times in distilled water. Young embryos were isolated and plated on MS medium in darkness at 20 °C until the embryos germinated and grew roots. Then the embryos were transferred to an incubator with a 12 hour photoperiod and a temperature of 25 °C. When the seedlings were about 5 to 10 cm tall, they were planted in pots with a mix of 50% soil, 40% peat moss, and 10% sand.

At the three-tiller stage, the seedlings were removed from soil, and roots of the seedlings were washed and trimmed. The roots and crown of the seedlings were immersed in a solution of 0.1% colchicine, 10 ppm GA3, 2% dimethyl sulfoxide (DMSO) and 0.01% Tween 20 in a beaker for 5 hours to induce chromosome doubling. After the roots were washed three times in tap water, they were placed in running tap water for 24 hours before transplanting to pots.

Root tips were collected from the F_1 seedlings in the pots for cytological observation before the seedlings were treated with colchicine. For the amphiploid of *T. timopheevi-T. monococcum*, root tips were collected from germinated seeds in petri dishes. The root tips were pretreated for 4 hours in a solution of colchicine (0.05%), 8-hydroxyquinoline (0.025%) and DMSO (25 drops per 100 ml), and stained for one week in 2% carmine in 45% acetic acid. The root tips were heated to boiling prior to squashing.

The F_1 hybrids of *T. timopheevi* and *T. monococcum* had 21 chromosomes (Fig. 1), and after chromosome doubling, the amphiploid of *T. timopheevi-T.monococcum* had 42 chromosomes (Fig. 2). Morphologically, the amphiploid resembled the accession of *T. zhukovskyi*, PGR 10370 (Fig. 3). It had a red coleoptile, and the entire plant was pubescent. Spikes of the amphiploid also resembled *T. zhukovskyi*. This amphiploid had a laterally compressed spike and long awns and, was non-free-threshing as a result of glumes which tightly held the grains. The spike was tapered at the base and tip. The rachis was rather brittle, and easily disarticulated into individual spikelets at maturity. The spikelets disarticulated at a point above the junction of the

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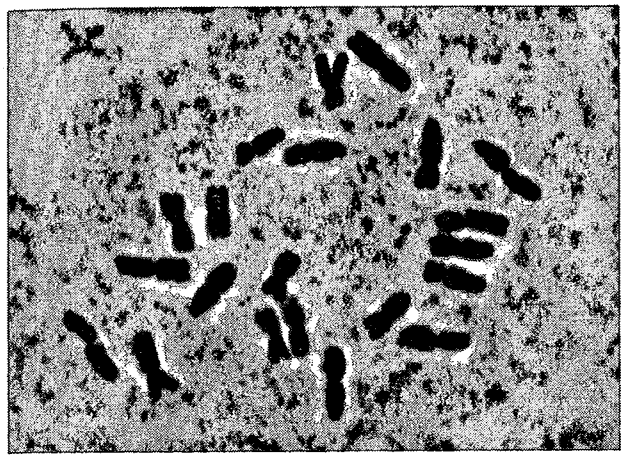

Fig. 1. Twenty-one chromosomes of the F1 hybrid of *T. timopheevi /T. monococcum*

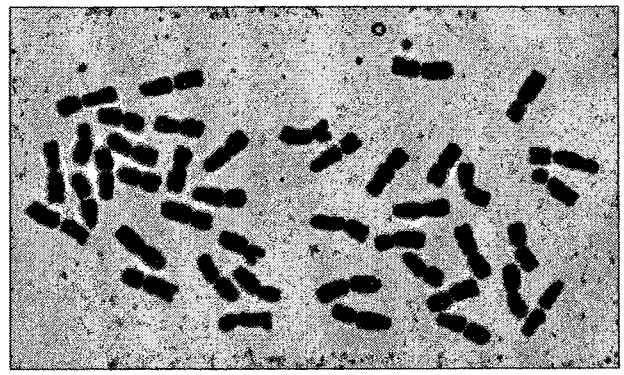

Fig. 2. Forty-two chromosomes of the amphiploid of *T. timophevi-T. monococcum*

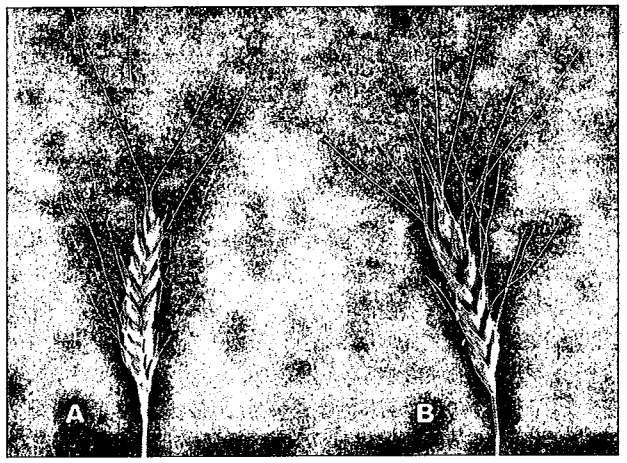

Fig. 3. Spikes of (A) *T. zhukovskyi*, (B) the amphiploid of *T. timopheevi-T. monococcum*

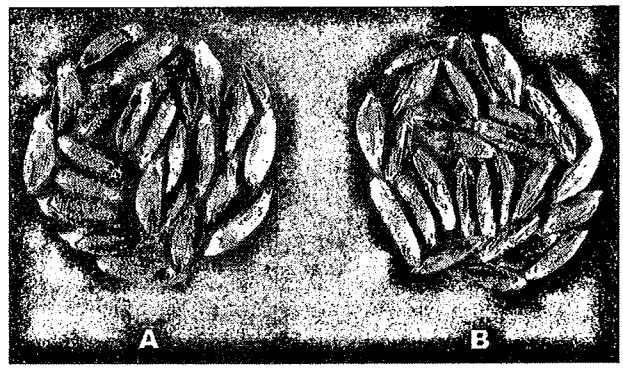

Fig. 4. Grains of (A) *T. zhukovskyi*, (B) the amphiploid of *T. timopheevi-T. monococcum*

rachis and rachilla, and each spikelet contained two grains. Both *T. zhukovskyi* and the amphiploid had long red grains (Fig. 4). *T. timopheevi* has been noted for its resistance to diseases, including rusts (Knott 1989), septoria nodorum and tan spot (Ma and Hughes 1995), and *T. monococcum* has resistance to rusts (Knott 1989). Therefore, this amphiploid derived from *T. timopheevi* and *T. monococcum* might have resistance to several diseases. Synthetic wheat with genome AAGGAA was previously developed using *T. timopheevi* and *T. monococcum* by Y. Watanabe in 1955 and T. Kawahara in 1982 (http://www.shigen.nig.ac.jp/wheat). In the current study, the accessions of *T. timopheevi* and *T. monococcum* used for the development of synthetic zhukovskyi wheat are different from those used by Y. Watanabe and T. Kawahara, and they have some resistance to fusarium head blight (data not shown).

The development of synthetic zhukovskyi wheat not only can provide useful germplasm for wheat breeding program but also can help scientist study wheat evolution. An evaluation of the amphiploid for resistance to fusarium head blight, septoria nodorum and tan spot etc. will be carried out in the greenhouse and fields. In order to confirm the evolution of *T. zhukovskyi*, a conventional cytogenetic study will be conducted by crossing the amphiploid with *T. zhukovskyi* for observation of chromosome pairing behavior of the F1, and a molecular cytogenetic study will also be conducted using *in situ* hybridization techniques.

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Gene symbol

CATALOGUE OF GENE SYMBOLS FOR WHEAT: 2000 Supplement

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The 1999 Supplement is included in 1999 Annual Wheat Newsletter, Wheat Information Service and is listed in the Graingenes Website. The present Supplement will be offered to editors/curators for similar listing.

Revisions

10. Laboratory Designators for DNA markers

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csl	Lagudah, E.S.* Division of Plant Industry CSIRO*, GPO Box 1600 Canberra ACT 2601 Australia	*pgh*	Shimosaka, E. Laboratory of Plant Genecology* Hokkaido* National Agricultural Experiment Station 1, Hitsujigaoka, Toyohira-ku Sapporo, 062-8555 Japan
kvl	Collinge, D.B. Section for Plant Pathology Dept. of Plant Biology Royal Veterinary and Agricultural University* Thorvaldsenvej 40 1871 Frederiksberg C Denmark	*rgs*	(Rice etiolated shoot* clones) Sasaki, T. (see *rgc*)
msu	Talbert, L.E. Plant Sciences Department Montana State University Bozeman, MT 59717 USA	*sun*	Sharp, PJ. Plant Breeding Institute University of Sydney* PMB 11, Camden NSW 2570 Australia

Morphological, Physiological, Molecular and DNA Traits

Following the first paragraph insert: More than 20 NILs involving genes affecting a range of traits are described in {0066}. These are not yet incorporated into the Catalogue.

Gross Morphology : Spike Characteristics
3. Sphaerococcum
Revision:
The naturally-occurringsphaerococcum gene in chromosome 3D and various mutant alleles conferring a similar phenotype form a homoeologous series. The sphaerococcoidalleles are either recessive or incompletely dominant. All three mapped loci are closely linked to the respective centromeres {0030}.The "a" alleles are allocated to Chinese Spring or "normal" wheats.

S-A1 {0029}.	3A {0056}.	**v:**	CS {0029}.
S-A1a {0029}.		**v:**	CS {0029}; common wheats {0029}.
S-A1b {0029}.	*S3* {0056}.	**v:**	MS 1453 {0056}.
	ma: *Xgwm2-3A*(S) - 5.1 cM - *S-A1* - 6.6 cM - *Xgwm720-3A* (L) {0030}.		
S-B1 {0029}.	3B {0030}.	**v:**	CS {0029}.
S-B1a {0029}.		**v:**	CS {0029}; common wheats {0029}.
S-B1b {0029}.	*S2* {0030}.	**v:**	MSK 2452 {0056}; MSK 2454 {0056}.
	ma: *Xgwm685-3B*(S) - 4.2 cM - *S-B1* - 0.5 cM - *Xgwm566* /*Xgwm845* /cent {0030}.		
S-D1 {0029}.	3D {1292,0030}; 3DS {1193,1194}; 3DL {692}.	**v:**	CS {0029}.
S-D1a {0029}.		**v:**	CS {0029}; common wheats {0029}.
S-D1b {0029}.	*s1* [*sp1* {1286}].	**i:**	S-615*11/*T.sphaerococcum* var. *rotundatum*{1500}.
		s:	CS*7/*T.sphaerococcum rubiginosum* 3D {1304}.
		v:	Sphaerococcum wheats {0029}.
S-D1c {0029}.	*S1* {0056}.	**v:**	MS 3287 {0056}.
	ma: *Xgdm72-3D*(S) - 8.0 cM - *S-D1* - 2.9 cM - *Xgwm456-3D*/cent {0030}.		

Temporary designation
s2 [*sp2* {1286}]. Partially dominant {1286}. Sphaerococcum simulator {1286}.
Sphaerococcum-like tetraploid wheats were reported {122,475,1282,1286}, but comparisons between them, or with *s2*, were not made. Whereas Schmidt & Johnson {1281} reported a single recessive factor controlling the sphaerococcum character intetraploid wheat, Joppa {621} using the same stock found that two recessive genes were necessary to produce this phenotype.

Ear length (new section under 'Spike characteristics')

QEl.ocs-5A.1 {0068}.	5AL {0068}.	**v:**	CS(*T. spelta* 5A)/CS(Cappelle-Desprez 5A) RI mapping population {0079}.
	ma: Associated with *Xbcd9* {0068}.		

Awnedness
1. **Dominant Inhibitors**
1.1 Hooded
Hd. Add: '**ma:** *Xcdo1387-4A* – 8.2 cM – *Hd* – 7.2 cM – *Xpsr163* {0047}.'

Boron Tolerance

Bo3. 4A {0012}.

Crossability with Rye, *Hordeum* and *Aegilops* spp.

1. Common wheat

Current section

2. Tetraploid wheat

The Chinese tetraploid, Ailanmai, possesses recessive crossability genes on chromosomes 1A, 6A and 7A with the 6A gene being the least effective {0017}.

DNA Markers

Group 1S

Amendments:

Xcdo534-1B. Revise the last column to '(6A,B ,D, 7A).'.
Xglk558-1D. Add '(5D).' in the last column.
Xpsr549-1A.2. Add '(5A).' in the last column.
Xpsr908-1B. Add '(6D).' in the last column.
Xpsr1327-1D. Revise the first column to '*Xpsr1327-1A* [{0031}], *1D* {410}.', add '[*Xpsr1327b-1A* {0031}].' in the second column and '(3B).' in the last column.
Xsfr1(Lrk10)-1A. Revise the first column to '*Xsfr2(Lrk10)-1A.*', and add '(3B,D).' in the last column.
Xsfrp1(Lrk10)-1A. Revise the first column to '*Xsfrp2(Lrk10)-1A.*'.

Add:

Xcnl5-1A [{0059}].		AG10F/AG10R.	
Xglk301-1B [{0031}].	[*Xglk301a-1B* {0031}].	pTag301.	(2D, 3D, 5D, 7A,B).
Xglk317-1B [{0031}].	[*Xglk317a-1B* {0031}].	pTag317.	(3D, 4D, 5A, 6A).
Xgwm264-1B.1 [{9929}[1],{0003}[2]].	[*Xgwm264-1B* {9929}, *Xgwm264c-1B* {0003}].	WMS F264/ WMS R264.	(1B, 3B).
Xgwm498-1A {0035}[2].		WMS F498/WMS R498.	(1BL).
Xgwm656-1A {0035}[2].		WMS F656/WMS R656.	
Xkvl901(Chs)-1B,D [{0091}].	[*Chs-1B,D* {0091}].	pBH72-O8 {0098}.	
Xkvl902(Fmt)-1B,D [{0091].	[*Fmt-1B,D* {0091}].	pBH72-F1 {0099}.	
Xpsr593-1B [{0031}].	[*Xpsr593a-1B* {0031}].	PSR593.	(2B, 4B, 7B).
Xpsr642-1B {0031}.		PSR642.	
Xpsr960-1B [{0031}].		PSR960.	
Xsfr3(LRR)-1A,B [{0031}].	[*CD9a,b-1A* {0031}].	CD9.	

Group 1L

Amendments:

Xbcd265-1A,B,D. Add '(4A).' in the last column.
Xglk163-1B,D. Add '(2D, 4D, 5AS, 5BL).' in the last column.
Xglk558-1B. Add '(5D).' in the last column.
Xgwm131-1B. Revise the first column to '*Xgwm131-1B.1* [{9929,0003}].', add '[*Xgwm131-1B* {9929}, *Xgwm131a-1B* {0003}].' in the second column, and add '(1B).' in the last column.
Xgwm498-1B. Add '(1AS).' in the last column.
Xmwg710-1A,B,D. Add '(7BL).' in the last column.
Xpsr172(Lhcb1)-1A. Add '(2B).' in the last column.
Xpsr549-1A.1. Add '(5A).' in the last column.
Xpsr1327-1A. Add '(1AS, 3B).' in the last column.

Add:

Xkvl903(Chi2)-1B [{0091}].	[*Cht22-1B* {0091}].	pBH72-N12 {0092}.	
Xgwm131-1B.2 [{0003}].	[*Xgwm131b-1B* {0003}].	WMS F131/WMS R131.	(1B, 3B).
Xgwm636-1B [{0003}].	[*Xgwm636b-1B* {0003}].	WMS F636/WMS R636.	(2A).
Xpsr924-1A,B,D {0043}.		PSR924.	(2B).

Group 1
Amendments:
Xglk558-1D. Add '(5D).' in the last column.
Xglk652-1D. Add '(3A).' in the last column.
Xpsr386-1A. Add '(2A).' in the last column.
Xwg232-1A. Add '(5B,D).' in the last column.

Add:

Xgwm264-1B.2 [{0003}[2]].	[*Xgwm264a-1B* {0003}].	WMS F264/WMS R264	(1B, 3B).
Xpsr967-1A,B {598}.		PSR967.	(4B, 5A, 6AS, 6BL).
Xwpg501(Pdi)-1B {0064}.		pTAPDI501.	(4A,B,D).

Group 2S
Amendments:
Xbcd102-2D. Add '(6A).' in the last column.
Xcdo456-2A.1,A.2,D. Revise the first column to '*Xcdo456-2A.1,A.2* {1060}, *2B* [{0074}], *2D* {1060}.', add '[*Xcdo456b-2B* {0074}].' in the second column and add as a note 'The arm location of *Xcdo456-2B* was not reported in {0074}.'.
Xfbb40-2B. Revise the last column to '(6A,D). '.
Xgwm210-2D. Revise the first column to '*Xgwm210-2B* {9929}, *2D* {1225}.'.
Xgwm264-2B. Delete.
Xgwm636-2A. Add '(1B).' in the last column.
Xpsr549-2B. Add '(5A).' in the last column.
Xpsr566-2A,D. Revise the first column to '*Xpsr566-2A* {256}, *2B* [{0031}], *2D* {256}.' and add '[*Xpsr566b-2B* {0031}].' in the second column.
Xpsr593-2B. Add '(1B).' in last column.
Xpsr908-2A,D. Add '(6D).' in the last column.

Add:

Xcsu182(Sod)-2B [{0091}].	[*Sod-2B* {0091}].	CSU182.	
Xglk197-2A {0031}[1], *2B* {9926}[4].		pTag197.	(7B).
Xglk222-2A {0031}, *2D* {822}.		pTag222.	(5B,D).
The arm location of *Xglk222-2D* was not reported in {822}.			
Xglk302-2B [{822,0049}].	[*Xglk302b-2B* {822,0049}].	pTag302.	(4A,D).
The arm location of *Xglk302-2B* was not reported in {822}.			
Xglk398-2B.1,B.2 [{822,0049}].	[*Xglk398a,b-2B* {822,0049}].	pTag398.	
The arm location of *Xglk398-2B.1,B.2* was not reported in {822}.			
Xglk400-2B {822,0049}.		pTag400.	
The arm location of *Xglk400-2B* was not reported in {822}.			
Xglk407-2B {822,0049}.		pTag407.	(5A).
The arm location of *Xglk407-2B* was not reported in {822}.			
Xglk471-2B [{822,0049}].	[*Xglk471b-2B* {822,0049}.	pTag471.	
Xglk546-2B.2	[*Xglk546f-2B* {822,0049}.	pTag546.	(3B, 5A, 6B, 7A,

[{822,0049}].			7B).
The arm location of *Xglk546-2B.2* was not reported in {822}.			
Xglk661-2B [{822,0049}].	[*Xglk661c-2B* {822,0049}.	pTag661.	(4A,B,D).
The arm location of *Xglk661-2B* was not reported in {822}.			
Xglk687-2B {822,0049}.		pTag687.	
The arm location of *Xglk687-2B* was not reported in {822}.			
Xglk703-2B {822,0049}.		pTag703.	
The arm location of *Xglk703-2B* was not reported in {822}.			
Xglk2002(Hst2a-1)-2A,B,D [{0049}].	[*Hst2a-A1,B1,D1* {0049}].	pwcH2A-4.	
Xgwm71-2B [{0035}][2].	[*Xgwm71a-2B* {0035}.	WMS F71/WMS R71.	(2AS, 2A, 3D).
Xgwm122-2A {9929}[1],{0035}[2].		WMS F122/WMS R122.	
The arm location of *Xgwm122-2A* was not reported in {9929}.			
Xgwm275-2A {9929}[1],{0035}[2].		WMS F275/WMS R275.	
The arm location of *Xgwm275-2A* was not reported in {9929}.			
Xgwm448-2A {9929}[1],{0035}[2].		WMS F448/WMS R448.	
The arm location of *Xgwm448-2A* was not reported in {9929}.			
Xgwm547-2B {0035}[2].		WMS F547/WMS R547.	(3B).
Xksu904(Per2)-2A,B [{0091}].	[*Per2-2A,B* {0091}].	pox22.3 {0093}.	
Xpsr172(Lhcb1)-2B [{0031}].	[*Xpsr172-2B* {0031}.	PSR172.	(1A, 5A,B,D, 7A,B,D).
Xpsr386-2A [{0031}].	[*Xpsr386c-2A* {0031}].	PSR386.	(1A, 3B, 5A, 7A).

Group 2L

Amendments:

Xfba111-2D. Revise the last column to '(6A,B,D).'.

Xglk554-2A,B. Revise the first column to '*Xglk554-2A* [{822}][1],{154}[1], *2B* [{822,0049}[1]], *2D* {9926}[4].', revise the second column to '[*Xglk554a-2A* {822}, *Xglk554c-2B* {822,0049}].', and add '(3B).' in the last column..

Xglk558-2B,D. Add '(5D).' in the last column.

Xgwm265-2A. Add '(4A).' in the last column.

Xgwm356-2A. Add '(6A).' in the last column.

Xpsr919-2A,B,D. Add '(3B).' in the last column.

Xpsr934-2A,B,D. Add '(4A).' in the last column.

Add:

Xbcd377-2A {0078}.		BCD377.	
Xcdc2(Sod3)-2A,B,D [{0054}].		SOD3.1 & SOD3.2.	
Xcnl6-2B [{0059}],{0060}.		AG24F/AG24R.	(7D).
Xglk76-2A [{822}], *2B* [{822,0049}].	[*Xglk76a-2A* {822}, *Xglk76b-2B* {822,0049}].	pTag76.	
The arm locations of *Xglk76-2A,B* were not reported in {822}.			
Xglk331-2B {822,0049}.		pTag331.	(4A).
The arm location of *Xglk331-2B* was not reported in {822}.			
Xglk370-2B {822,0049}, *2D* {1034}.		pTag370.	(4A).
The arm locations of *Xglk370-2B,D* were not reported in {822} and {1034}.			
Xglk529-2B [{822,0049}], *2D* [{822}].	[*Xglk529a-2A* {822,0049}, *Xglk529b-2D* {822}].	pTag529.	

The arm locations of *Xglk529-2B,D* were not reported in {822}.

Xglk539-2B {822,0049}.		pTag539.	
The arm location of *Xglk539-2B* was not reported in {822}.			
Xglk592-2B {822,0049}.		pTag592.	
The arm location of *Xglk592-2B* was not reported in {822}.			
Xglk600-2A [{822}], *2B* [{822,0049}].	[*Xglk600a-2A* {822}, *Xglk600b-2B* {822,0049}.	pTag600.	(4A).
The arm locations of *Xglkj600-2A,B* were not reported in {822}.			
Xglk605-2B {822,0049}.		pTag605.	
The arm location of *Xglk605-2B* was not reported in {822}.			
Xglk609-2B [{822,0049}], *2D* [{822}].	[*Xglk609b-2B* {822,0049}, *Xglk609a-2D* {822}.	pTag609.	(3A).
The arm locations of *Xglk609-2B,D* were not reported in {822}.			
Xglk618-2B {822,0049}.		pTag618.	
The arm location of *Xglk618-2B* was not reported in {822}.			
Xglk632-2A [{822}],*2B* [{822,0049}], *2D* {0031}.	[*Xglk632a-2A* {822, *Xglk632b-2B* {822,0049}].	pTag632.	
The arm locations of *Xglk632-2A,B* were not reported in {822}.			
Xglk653-2A [{822}], *2B* [{822,0049}], *2D* {1034}.	[*Xglk653a-2A* {822}, *Xglk653b-2B* {822,0049}].	pTag653.	
The arm locations of *Xglk653-2A,B* and *Xglk653-2D* were not reported in {822} and {1034}.			
Xglk664-2A [{822}], *2B* [{822,0049}], *2D* {0031}.	[*Xglk664a-2A* {822}, *Xglk664b-2B* {822,0049}].	pTag664.	
The arm locations of *Xglk664-2A,B* were not reported in {822}.			
Xglk699-2B {822,0049}.		pTag699.	
The arm location of *Xglk699-2B* was not reported in {822}.			
Xglk740-2A [{822}], *2B* [{822,0049}].	[*Xglk740b-2A* {822}, *Xglk740a-2B* {822,0049}.	pTag740.	
The arm locations of *Xglk740-2A,B* were not reported in {822}.			
Xgwm372-2A {0035}[2].		WMS F372/WMS R372.	
Xksu905(Wip)-2A [{0091}].	[*Wip-2A* {0091}].	5C05B11 {0094}.	
Xkvl906(Cbp2)-2A [{0091}].	[*Cbp2-2A* {0091}].	pBH72-B8 {0092}.	(7B).
Xksu908(Cbp1)-2B [{0091}].	[*Cbp1-2B* {0091}].	RRI 10 {0095}.	(6B).
Xksu909(Chi1)-2B [{0091}].	[*Cht1a-2B* {0091}].	Chi-G11 {0096}.	(3A).
Xksu910(Tha1)-2D [{0091}].	[*Tha1-2D* {0091}].	CR5 {0097}.	(4A, 6B, 7A,B,D).
XksuF41-2A.1,.2 {0078}.		pTtksuF41.	(2B,D).
Xmwg526-2A.1,.2 {0078}.		MWG526.	(3D).
Xpsr924-2B {0031}.		PSR924.	(1A,B,D).
Xpsr644-2B [{0031}].	[*Xpsr644b-2B* {0031}].	PSR644.	(5A).
Xpsr1200-2A,B,D {0043}.		PSR1200.	(3A).
Xwmc41-2D {0015}.		WMC 41F/WMC 41R {0080}.	

Group 2

Amendments:

Xglk76-2A,B. Delete (moved to 2L).

Xglk163-2D. Add '(1B, 5AS, 5BL).' in the last column.

Xglk197-2B. Delete (moved to 2S).

Xglk222-2D. Delete (moved to 2S).
Xglk278-2D. Revise the first column to '*Xglk278-2A.1,A.2,B* [{0031}], *2D* {9926}[4].' and add '[*Xglk278a,b-2A, Xglk278c-2B*{0031}].' in the second column.
Xglk293-2D. Revise the first column to '*Xpsr293-2A* [{0031}], *2D* {822}.' and add '[*Xpsr293b-2A* {0031}].' in the second column.
Xglk301-2D. Add '(1B, 7B)' in the last column.
Xglk302-2B. Delete (moved to 2S).
Xglk331-2B. Delete (moved to 2L).
Xglk370-2B,D. Delete (moved to 2L.
Xglk398-2B(1),(2). Delete (moved to 2S).
Xglk400-2B. Delete (moved to 2S).
Xglk407-2B. Delete (moved to 2S).
Xglk471-2B. Delete (moved to 2S).
Xglk529-2B,D. Delete (moved to 2L).
Xglk539-2B. Delete (moved to 2L).
Xglk546-2B(1),(2). Revise the first column to '*Xglk546-2B.1* [{822}]' and add '*Xglk546-2B.2* was moved to 2S'.
Xglk554-2A,B,D. Delete (moved to 2L).
Xglk578-2B. Delete (moved to 2S).
Xglk592-2B. Delete (moved to 2L).
Xglk600-2A,B. Delete (moved to 2L).
Xglk605-2B. Delete (moved to 2L).
Xglk609-2B,D. Delete (moved to 2L).
Xglk610-2A. Revise the first column to '*Xglk610*-2A {822}, *2B.1,B.2,D* [{0031}].' and add '[*Xglk610a,b-2B, Xglk610c-2D*{0031}].' in the second column.
Xglk618-2B. Delete (moved to 2L).
Xglk632-2A,B. Delete (moved to 2L).
Xglk653-2A,B,D. Delete (moved to 2L).
Xglk661-2B. Delete (moved to 2S).
Xglk664-2A,B. Delete (moved to 2L).
Xglk684-2A. Revise the first column to '*Xglk684-2A* {822}, *2B* [{0031}].' and add '[*Xglk684b-2B*{0031}].' in the second column.
Xglk687-2B. Revise the first column to '*Xglk687-2A* [{0031}], *2B* {822}.' and add '[*Xglk687a-2A* {0031}].' in the second column.
Xglk699-2B. Revise the first column to '*Xglk699-2A* [{0031}], *2B* {822}.' and add '[*Xglk699b-2A* {0031}].' in the second column.
Xglk703-2B. Delete (moved to 2S).
Xglk740-2A,B. Delete (moved to 2L).
Xgwm122-2A. Delete (moved to 2S).
Xgwm210-2D. Delete (the 1999 amendments for this entry should have been made in the 2S group).
Xgwm275-2A. Delete (moved to 2S).
Xgwm448-2A. Delete (moved to 2S).
Xwg405-2D. Add '(6D).' in the last column.

Add:

Xbcd18-2B.1,.2,.3 [{0074}].	[*Xbcd18a,b,c-2B*{0074}].	BCD18.	
Xbcd907-2B{0074}.	[*Xbcd907b-2B*{0074}].	BCD907.	(3B,D, 7A).
Xbcd1086-2B[{0074}].	[*Xbcd1086b-2B*{0074}].	BCD1086.	
Xpsr129-2A [{0031}].	[*Xpsr129a-2A* {0031}].	PSR129.	(7A,B,D).
Xpsr961-2B {0031}.		PSR961.	
Xsfr4(NBS)-2A [{0031}].	[*PL_AP-2A* {0031}].	PL_AP.	

Group 3S
Amendments:
Xbcd907-3B,D. Add '(2B, 7A).' in the last column.
Xgwm114-3D. Delete (moved to 3L).

Add:

Xbcd907-3B.1,.2 {0078}.		BCD907.	(2B, 3D, 7A).
Xcsu358(Pal)-3B [{0091}].	[*Pal-3B* {0091}].	CSU358.	
Xgdm72-3D {0030}.		ASMS F72/ASMS R72.	
Xglk2007(Bzb2-1)-3A,B,D [{0049}].	[*Bzb2-A1,B1,D1* {0049}].	IHBP-1b(c38).	
Xgwm685-3B {0030}.		WMS F685/WMS R685.	
XksuA1-3B {0078}.		pTtksuA1.	(1B, 5B, 7D).
Xmsu1-3B [{0076}].		XJ5U/XJ5R.	
Xmsu2-3B [{0076}].		XJ26U/XJ26R.	
Xmsu3-3B [{0076}].		XJ28U/XJ28R.	
Xrgc970-3A,B [{0031}].	[*Xrgc970b-3A, Xrgc970a-3B* {0031}].	RGC970.	
Xpsr1200-3A {0031}.		PSR1200.	(2A,B,D).
Xpsr1327-3B [{0031}].	[*Xpsr1327a-3B* {0031}].	PSR1327.	(1AS,DS, 1AL, 3B, 4A,5D).
Xsfr2(Lrk10)-3B,D [{0031}].	[*Lrk10-3B,D* {0031}].	Lrk10.	(1A).

Group 3L

Amendments:

Xglk609-3A. Add '(2B,D).' in the last column.

Xgwm547-3B. Add '(2B).' in the last column.

XksuD19-3D. Revise the first column to '*XksuD19-3B* {0078}, *3D* {1061}.'

Xpsr549-3A. Add '(5A).' in the last column.

Xpsr904-3A,D. Revise the last column to '(6AS, 6DL).'.

Add:

Xabcp174-3B [{0087}].		ABC174 3f/ABC174 1r.	
Xbcdp131-3D [{0087}].		BCD131 1f*/BCD 1r.	
Note : In order to get a 3D-specific amplicon, the BCD131 1f primer was modified by removing the 5' T and adding a C at the 3' end {0087}.			
Xbcd187-3B {0078}.		BCD187.	
Xbcd195-3B {0078}.		BCD195.	
Xcdo251-3B {0078}.		CDO251.	
Xcnl2-3D [{0059}],{0060}.		AC14F/AC14R	(7B).
Xcnl4-3D [{0031}].	[*AC29* {0031}].	AC29F/AC29R {0059}.	
Xglk118-3A {822},{0031}.		pTag118.	
The arm location of *Xglk118-3A* was not reported in {822}.			
Xglk577-3A {822},{0031}, *3B,D* {1034}.		pTag577.	
The arm location of *Xglk577-3A,B,D* was not reported in {822} and {1034}.			
Xglk645-3A {822},{0031}.		pTag645.	
The arm location of *Xglk645-3A* was not reported in {822}.			
Xglk652-3A [{0031}], *3B* [{822}[1],{0031}], *3D* {9926}[4].	[*Xglk652a-3A* {0031}, *Xglk652b-3B* {822}].	pTag652.	(1D).
The arm location of *Xglk652-3B* was not reported in {822}.			
Xglk2003(Bza1-1)-3B [{0049}].	[*Bza1-B1* {0049}].	IHBP-1a(1).	(5A,D, 6AL, 6BS, 7D).

Xglk2006(Bzb-1)-3A,B,D [{0049}].	[*Bzb1-A1,B1,D1* {0049}].	IHBP-1b(c1).	
Xgwm114-3D {9929,0039}}.		WMS F114/WMS R114.	(3B).
The arm location of *Xgwm114-3D* was reported incorrectly in {9929}.			
Xgwm638-3A {0035}[2].		WMS F638/WMS R638.	
Xgwm674-3A {9929}[1],{0035}[2].		WMS F674/WMS R674.	
The arm location of *Xgwm674-3A* was not reported in {9929}.			
Xgwm705-3B {0030}.		WMS F705/WMS R705.	
Xgwm707-3D {0030}.		WMS F707/WMS R707.	
Xgwm720-3A {0030}.		WMS F720/WMS R720.	
Xgwm751-3A {0030}.		WMS F751/WMS R751.	
Xgwm802-3B {0030}.		WMS F802/WMS R802.	
Xgwm853-3B {0030}.		WMS F853/WMS R853.	
Xksu909(Chi1)-3A [{0091}].	[*Cht1a-3A* {0091}].	Chi-G11 {0096}.	(2B).
Xkvl912(Prp)-3A [{0091}].	[*Prp-3A* {0091}].	HvPRPb {00100}.	
Xkvl914(Glb3)-3B.1,B.2,B.3,B.4,D [{0091}].	[*Glb3-3B.1,B.2,B.3,B.4,D* {0091}].	pBH72-I1 {0092}.	
Xpsr936-3A {0031}.		PSR936.	

Group 3
Amendments:
Xglk118-3A. Delete (moved to 3L).
Xglk301-3D. Add '(1B, 7B).' in the last column.
Xglk317-3D. Add '(1B).' in the last column.
Xglk558-3D. Add '(5D).' in the last column.
Xglk577-3A,B,D. Delete (moved to 3L).
Xglk645-3A. Delete (moved to 3L).
Xglk652-3B. Delete (moved to 3L).
Xgwm674-3A. Delete (moved to 3L).
Xmwg526-3D. Add '(2A).' in the last column.
Xpsr386-3B. Add '(2A).' in the last column.

Add:

Xglk554-3B [{0031}].	[*Xglk554b-3B* {0031}].	pTag554.	(2A,B,D, 5B).
Xgwm845-3B {0030}.		WMS F845/WMS R845.	
Xpsr919-3B [{0031}].	[*Xpsr919a-3B* {0031}.	PSR919.	(2A,B,D).
Xpsr1054-3B {0031}.		PSR1054.	

Group 4S (4AL:4BS:4DS)
Amendments:
Xbcd265. Revise the first column to '*Xbcd265-4B* {0047}, *4D* {1059}.'
Xcdo1338-4A. Add '(5B,D)' in the last column.
Xglk278-4D. Add '(2A,B).' in the last column.
Xpsr593-4B. Add '(1B).' in last column.
Xpsr1327-4A. Add '(1AS, 3B).' in the last column.
Xpsr1871(Pki)-4A,B,D. Revise the first column to '*Xkvl1871(14-3-3a)-4A,B,D*[{255},{0091].', add '[*Xpsr1871(Pki)-4A,B,D* {255}, *1433a-4A,B* {0091}].' in the second column, and revise the third column to 'pBT6-5tot (pHv14-3-3a)'.

Add:

Xglk348-4B [{0031}], *4D*	[*Xglk348a-4B, Xglk348b-4D*	pTag348.

[{822},{0031}]. {0031}, *Xglk348a-4D* {822}].
The arm location of *Xglk348-4D* was not reported in {822}.
Xgwm513-4B WMS F513/WMS R513.
{9929}[1],{0035}[2].
The arm location of *Xgwm513-4B* was not reported in {9929}.
Xgwm601-4A {0035}[2]. WMS F601/WMS R601.
Xkvl916(Oxo)-4A,B [*Oxo2-4A,B* {0091}]. pOXOXa {00102}.
[{0091}].
Xkvl917(Tha2)-4A [*Tha2-4A* {0091}]. pBH72-C6 {0092}. (7A,B).
[{0091}].
Xkvl918(Chi2)-4A,B [*Cht21-4A,B* {0091}]. pBH72-C4 {0092}.
[{0091}].

Group 4L (4AS:4BL:4DL)
Amendments:
Xgwm6-4B {1226}. Add '(5A, 4D).' in the last column.
It is possible that *Xgwm6-5A,4B,D* form a homoeologous series.
Xgwm6-4D {1226}. Add '(5A,4B).' in the last column.
It is possible that *Xgwm6-5A,4B,D* form a homoeologous series.

Add:
Xglk315-4A pTag315.
{822},{0031}.
The arm location of *Xglk315-4A* was not reported in {822}.
Xglk752-4A [*Xglk752a-4A* {822}]. pTag752. (6B).
[{822}],{0031}.
The arm location of *Xglk752-4A* was not reported in {822}.
Xglk2004(Bza2-1)-4A,B,D [*Bza2-A1,B1,D1* {0049}]. IHBP-1a(c14).
[{0049}].
Xgwm192-4A,B,D [*Xgwm192c,a,b-4A,B,D* WMS F192/ WMS R192.
[{0039}]. {0039}].
Xgwm397-4A {0031}. WMS F397/WMS R397.

Group 5AL:4BL:4DL
Amendments:
Xfba1-4B. Revise the last column to '(6BS,DS, 6AL).'.

Add:
Xgwm6-5A [{0031}]. [*Xgwm6b-5A* {0031}]. WMS F6/ WMS R6. (4B,D).
It is possible that *Xgwm6-5A,4B,D* form a homoeologous series.
Xkvl920(OxoLP)-4D [*Oxo1-4D* {0091}]. pBH6-903 {00103}.
[{0091}].

Group 4
Amendments:
Xglk163-4D. Add '(1B, 5AS, 5BL).' in the last column.
Xglk302-4A. Revise the first column to '*Xglk302-4A* [{822}], *4D* [{0031}].' and add '[*Xglk302b-4D* {0031}].' in the second column.
Xglk315-4A. Delete (moved to 4L).
Xglk317-4D. Add '(1B).' in the last column.
Xglk348-4D. Delete (moved to 4S).
Xglk752-4A. Delete (moved to 4L).
Xgwm513-4B. Delete (moved to 4S).
Xwg232-4A. Add '(5B,D).' in the last column.

Add:

Xglk331-4A {0031}.		pTag331.	(2B).
Xglk600-4A [{0031}].	[*Xglk600b-4A* {0031}].	pTag600.	(2A,B).
Xgwm111-4A [{0031}].	[*Xgwm111c-4A* {0031}].	WMS F111/WMS R111.	(7B,D).
Xpsr934-4A [{0031}].	[*Xpsr934a-4A* {0031}].	PSR934.	(2A,B,D).
Xcsl102(NBS-LRR)-4A [{0031}].	[*CD16.2-4A* {0031}].	Cd16.2 {0048}.	
Xpsr967-4B {598}.		PSR967.	(1A,B, 5A, 6AS, 6BL).
Xwpg501(Pdi)-4A,B,D {0064}.		pTAPDI501.	(1B).

Group 5S

Amendments:

Xcdo1338-5A. Revise the first column to '*Xcdo1338-5A* {282}[3], *5B,D* {0034}.

Xglk317-5A.1,.2, 5A. Add '(1B).' in the last column.

Xgwm192-5D. Delete.

Xpsr1327-5D. Add '(1AS, 3B).' in the last column.

Add:

Xcdo344-5A,B,D {0034}.		CDO344.	
Xfba114-5B {0034}, *5D* {1059,0034}.		FBA114.	
The arm location of *Xfba114-5D* was not reported in {1059}.			
Xbfa137-5D {1059,0034}.		FBA137.	(4A).
The arm location of *Xfba137-5D* was not reported in {1059}.			
Xglk163-5A {0031}.	[*Xglk163a-5A* {0031}].	pTag163.	(1B,D, 2D, 4D, 5BL).
Xkvl922(Tha3)-5B,D [{0091}].	[*Tha3-5,D* {0091}].	pBH72-K10 {0092}.	
Xmta9-5D {1239,0034}.		MTA9 {629}.	
The arm location of *Xmta9-5D* was not reported in {1239}.			
Xpsr549-5A {0031}.		PSR549.	(1AS, 1AL, 2B, 3A).
Xpsr644-5A [{0031}].	[*Xpsr644a-5A* {0031}.	PSR644.	(2B).
Xrgc3-5A {0034}.		RGC3.	
Xrgr2104-5A,D {0034}.		RGR2105.	

Group 5L

Amendments:

Xbcd265-5A. Add '(4A).' in the last column.

Xbcd926-5A. Revise the first column to '*Xbcd26-5A* {1059}, *5B,D* {0034}.'

Xbcd1087-5D. Revise the first column to '*Xbcd1087-5A,B* {0034}, *5D* {446}.'

Xcdo504-5A. Revise the first column to '*Xcdo504-5A* {419,282}[3], *5B* {1059}, *5D* {0034}.'

Xcdo584-5B. Revise the first column to '*Xcdo584-5A* {0068}, *5B* {1059}.'

Xcdo590-5A. Revise the first column to '*Xcdo590-5A* {9969}, *5B,D* {0034}.'

Xglk165-5B,5D.1,.2. Add '(7B).' in the last column.

Xglk222-5B,D. Add '(2A).' in the last column.

Xglk621-5D. Delete (moved to 4AL:5BL:5DL).

XksuA1-5B. Add '(3B).' in the last column.

Xpsr918-5D. Revise the first column to '*Xpsr918-5A,B* [{0031}], *5D* {1609}.' and add '[*Xpsr918b-5A, Xpsr918a-5B* {0031}].' in the second column.

Xrgc711-5A. Revise the first column to '*Xrgc711-5A* {9969}, *5B,D* {0034}.'

Xrz474-5A. Revise the first column to '*Xrz474-5A* {9969}, *5B,D* {0034}.'

Xrgr2311-5A. Revise the first column to '*Xrgr2311-5A* {9969}, *5D* {0034}.'

Xrgr3226-5A. Revise the first column to '*Xrgr3226-5A* {9969}, *5D* {0034}.'

Xrgr2404-5A. Revise the first column to '*Xrgr2404-5A* {9969}, *5B,D* {0034}.'
Xrgr2443-5A. Revise the first column to '*Xrgr2443-5A* {9969}, *5B,D* {0034}.'
Xrz630-5A. Revise the first column to '*Xrz630-5A* {9969}, *5B,D* {0034}.'
Xrz698-5A. Revise the first column to '*Xrz698-5A* {9969}, *5B,D* {0034}.'

Add:

Xcdo520-5A,D {0034}.		CDO520.	
Xcdo1088 {0068}.		CDO1088.	
Xglk163-5B [{0031}].	[*Xglk163b-5B* {0031}].	pTag163.	(1B,D, 2D, 4D, 5AS).
Xglk2003(Bza-2)-5A,D [{0049}].	[*Bza1-A2,D2* {0049}].	IHBP-1a(1).	(3B, 6AL, 6BS, 7D).
Xksu919(Lpx)-5A,B [{0091}].	[*Lpx-5A,B* {0091}].	6C02E12 {0094}.	(4A).
Xksu921(Mpc1)-5A [{0091]].	[*Mpc1-5A* {0091}].	c1 {0094}.	(7D).
Xksu923(Pr1)-5D [{0091}].	[*Pr1-5D* {0091}].	CR1 [0097}.	
Xpsp128-5A,B,D {0086}.		PSP128F1/PSP128R1.	
Xpsp128-5D {0086}.		PSP128F2/PSP128R2.	
Xrgc1401-5A {0067}.		C1401.	
Xrgr2632-5A {0067}.		R2632.	
Xrgr2856-5A {0067}.		R2856.	
Xrgs1912-5A {0067}.		S1912.	
Xrz596-5B,D {0034}.		RZ596.	
Xwg232-5A,B,D {0034}.		WG232.	(1A, 4A, 7A).

4AL:5BL:5DL
Amendments:
Xgwm637-4A. Delete '**' and add '{0035}.' in the first colum. Delete Note: 'Whether*Xgwm637-4A* belongs to ... is uncertain.'.

Add:

Xglk558-5D [{0031}].	[*Xglk558a-5D* {0031}].	pTag558.	(1BL, 1DS, 1D, 2B,D, 3D, 6D, 7D).
Xgwm118-4A,5B [{0035}]^2.	[*Xgwm118a-4A, Xgwm118b-5B* {0035}]^2.	WMS F118/WMS R118.	
Xgwm265-4A {0035}^{2}.		WMS F265/WMS R265.	(4A).

Group 5
Amendments:
Xfba114-5D {1059}. Delete (moved to 5S).
Xfba137-5D. Delete (moved to 5S).
Xglk278-5A,B. Add '(2A,B).' to the last column.
Xglk301-5D. Add '(1B, 7B)' in the last column.
Xmta9-5D. Delete (moved to 5S).
Xpsr172(Lhcb1)-5A,B,D. Add '(2B).' in the last column.
Xpsr386-5A. Add '(2A).' in the last column.
Xwg232-5A.1,B. Add '(5D).' in the last column.
Xwg232-5A.2. Add '(5B,D).' in the last column.

Add:

Xpgh1(ELIP)-5A,B,D [{0053}].	[*Xwcr12-5A,B,D* {0053}].	WCR12.	

Xglk2001(Hst1-1)-5A,B,D [{0049}].	[*Hst1-A1,B1,D1* {0049}].	ITAHISTH1.	
Xpsr967-5A {598}.		PSR967.	(1A,B, 4B, 6AS, 6BL).
Xrgc1329-5A {0067}.		C1329.	
Xrgr1618-5A {0067}.		R1618.	
Xrgc2540-5A {0067}.		C2540.	

Group 6S

Amendments:

Xabc173-6A,D. Revise the first column to '*Xabc173-6A* {9927}^{2}, *6B* {0081}^{1}, *6D* {900}^{1}.'.

Xcdo534-B,D. Revise the first column to '*Xcdo534-6A* {0081}^{1}, *6B* {860}^{1},{9927}^{2}, *6D* {900}^{1}.' and revise the last column to '(1B, 7A).'.

Xcdo1380-6B. Replace '6BS' with '6BL' in 'A 6BS *Xcdo1380-6B* locus was mapped in {9921}.'.

Xcmwg652-6A,B,D. Revise the first column to '*Xcmwg652-6A* {900}^{1}, *6B* {9927}^{2},{0081}^{1}, *6D* {9926}^{4},{0081}^{1}.'.

Xcmwg653-6A. Delete (moved to 6L).

Xcmwg684-6B. Add '(6D).' in the last column.

Xcmwg690-6A,B. Revise the first column to '*Xcmwg690-6A,B* {9927}^{2}, *6D* {0081}^{1}.'.

Xfba1-6D. Revise the first column to '*Xfba1-6B* {0081}, *6D* {900}.', and add '(6AL).' in the last column.

Xfba67-6B. Revise the first column to '*Xfba67-6A* {0081}, *6B* {900}, *6D* {0081}.'.

Xfba85-6A,D. Revise the first column to '*Xfba85-6A* {900}, *6B* {0081}, *6D* {900}.'.

Xfba148-6A. Revise the first column to '*Xfba148-6A* {900}, *6D* {0081}.'.

Xfba187-6D. Revise the first column to '*Xfba187-6B* {0081}, *6D* {900}.'.

Xfba328-6B. Delete (moved to 6L).

Xfbb354-6D. Revise the first column to '*Xfbb354-6A,B* {0081}, *6D* {900}.'.

Xfbb399-6B. Revise the first column to '*Xfbb399-6A* {0081}, *6B* {900}, *6D* {0081}.'.

Xglk479-6B. Revise the first column to '*Xglk479-6A* {822,0049}^{1}, *6B* {900}^{1}, *6D* {9926}^{4}.' and add 'The arm locations of *Xglk479-6A* and *Xglk479-6D* were not reported in {822} and {9926}.'.

Xglk537-6A. Revise the first column to '*Xglk537-6A* {822,0049}^{1}, {9927}^{2}.'

Xglk562-6A,B. Revise the first column to '*Xglk562-6A* {822,0049}^{1}, *6B* {9927}^{2}.'

XksuG48-6A,B,D. Revise the first column to '*XksuG48-6A* {187}^{2}, *6B* {187}^{2},{0081}^{1}, 6D {448}^{4},{444,862}^{1}.'

Xmwg966-6A. Revise the first column to '*Xmwg966-6A* {9927}^{2}, *6B* {0081}^{1}.'.

Xpsr141(Pgk2)-6A,B,D. Revise the first column to '*Xpsr141(Pgk2)-6A,B,D* [{598}]^{b}, *6D.1,.2* {0081}.'.

Xpsr301-6A,B,D. Revise the first column to '*Xpsr301-6A* {598}, *6B.1* [{598}],{0081}, *6D* {598}.', add '[*Xpsr301-6B* {598}]' in the second column and add '(6BL).' in the last column.

Xpsr899-6A,B,D. Delete '*6B* {900}' from the entry.

Xpsr904-6A. Add '(6DL).' in the last column.

Xpsr967-6A. Add '(6BL).' in the last column.

Xtam10-6A,B. Revise the first column to '*Xtam10-6A.1* [{245}]^{1}, *6B* {245}^{1},{187}^{2}.', add '[*Xtam10-6A* {245}].' in the second column and add '(6AL).' in the last column.

Add:

Xcdo365-6B {0071}.		CDO365.	
Xcnl3-6B [{0059}],{0060}.		AC22F/AC22R.	
Xfba381-6B,D.2 {0081}.		FBA381.	(6DL).
Xglk172-6A [{822,0049}].	[*Xglk172a-6A* {822,0049}].	pTag172.	(7A,B).
The arm location of *Xglk172-6A* was not reported in {822}.			
Xglk752-6B [{822}],{0081}.	[*Xglk752b* {822}].	pTag752.	(4A).
The arm location of *Xglk752-6B* was not reported in {822}.			
Xglk2003(Bza1-4)-6B [{0049}].	[*Bza1-B4* {0049}].	IHBP-1a(1).	(3B, 5A,D, 6AL, 7D).
Xglk2005(Bza3-1)-6A,B,D [{0049}].	[*Bza3-A1,B1,D1* {0049}].	IHBP-1a(17).	
Xgwm82-6A {0035}^{2}.		WMS F82/WMS R82.	

Xksu924(Ppo)-6A,D [{0091}].	[*Ppo6A,D* {0091}].	7C02D02 {0094}.	
Xksu925(Hrp)-6A [{0091}].	[*Hrp-6A* {0091}].	5C05D01 {0094}.	
Xksu926(Rip)-6D [{0091}].	[*Rip-6D* {0091}].	5C04F01 {0094}.	
Xpsp551-6B {0086}.		PSP551F1/PSP551R1.	
Xrz476-6B {0081}.		RZ476.	(7B).
Xuta1(Psif)-6A,B,D {0081}.		p26 {999}.	
Xuta2(Psif)-6D {0081}.		p28 {999}.	

Group 6L

Amendments:

Xabc163-6A. Revise the first column to '*Xabc163-6A* {282}^{3}, *6D* {0081}^{1}.'.

Xabc175-6A,D. Revise the first column to '*Xabc175-6A* {9927}^{2},{0081}^{1}, *6D* {900}^{1}.'.

Xabg388-6A. Revise the first column to '*Xabg388-6A* {282}^{3}, *6B* {0081}^{1}.'.

Xbcd1-6B. Revise the first column to '*Xbcd1-6A* {0081}^{1}, *6B* {187}^{2},{0081}^{1}, *6D* {0081}^{1}.'.

Xbcd357-6D. Revise the first column to '*Xbcd357-6B* {860,0071}, *6D* {900}.', delete '(6B).' from the last column and add 'The arm location of *Xbcd357-6B* was not reported in {860}.'.

Xcdo772-6A,B. Revise the first column to '*Xcdo772-6A* {900}, *6B* {9921}, *6D* {0081}.'.

Xcmwg674-6A. Revise the first column to '*Xcmwg674-6A* {9927}^{2},{0081}^{1}, *6B,D* {0081}^{1}.'.

Xcmwg684-6A. Revise the first column to '*Xcmwg684-6A* {282}^{3},{9927}^{2},{0081}^{1}, *6B.2* [{9927}^{2},{0081}^{1}], *6D* [{0031}]^{1}.' and revise the second column to '[*Xcmwg684-6B* {9927,0081}, *Xcmwg684b-6D* {0031}].'.

Xcsb112(Dhn5)-6A,B. Revise the first column to '*Xcsb112(Dhn5)-6A,B* [{187}]^{2}.' and add '[*Xcsb112(Dhn5-6B.2,D.2* {0081}].' to the second column.

XEsi35-6A,B,D. Revise the first column to '*Xucd109(Esi35)-6A,B,D* [{278}].' and add '[*XEsi35-6A,B,D* {278}].' in the second column.

Xfba81-6D. Revise the first column to '*Xfba81-6B* {0081}, *6D* {900}.'.

Xfba111-6A,B. Revise the first column to '*Xfba111-6A,B* {900}, *6D* {0081}.'.

Xfba381-6D. Revise the first column to '*Xfba381-6D.1* [{900}]', add '[*Xfba381-6D* {900}].' in the second column, and add '(6BS,6DS).' in the last column.

Xfbb40-6A. Revise the first column to '*Xfbb40-6A* {900}, *6D* {0081}.'.

Xfbb57-6B. Revise the first column to '*Xfbb57-6A* {0081}, *6B* {900}, *6D* {0081}.'.

Xfbb82-6A,B. Revise the first column to '*Xfbb82-6A,B* {900}, *6D* {0081}.'.

Xfbb164-6B. Revise the first column to '*Xfbb164-6A* {0081}, *6B* {900}.'.

Xfbb169-6B,D. Revise the first column to '*Xfbb169-6A* {0081}, *6B,D* {900}.'.

Xfbb170-6A. Revise the first column to '*Xfbb170-6A* {900}, *6B* {0081}.'.

Xfbb221-6A. Revise the first column to '*Xfbb221-6A* {900}, *6B* {0081}.'.

Xfbb327-6B. Revise the first column to '*Xfbb327-6B* {900}, *6D* {0081}.'.

Xglk334-6A. Revise the first column to '*Xglk334-6A* {882,0049}^{1}, *6B* {9927}^{2}, *6D* {0081}^{1}.' and revise the note to 'The arm location of *Xglk334-6A* was not reported in {822}.'.

Xfbb364-6B. Revise the first column to '*Xfbb364-6B* {0081}, *6B* {900}, *6D* {0081}.'.

Xglk547-6A. Revise the first column to '*Xglk547-6A* {9927}^{2}, *6A.1,.2,.3* [{822,0049}]^{1}, *6B* [{822}]^{2}.', revise the second column to'[*Xglk547a,b,d-6A.1,.2,.3* {822,0049}, *Xglk547c-6B*{822}].', and add 'The arm locations of *Xglk547-6A.1,.2,.3,B* were not reported in {822}.'.

Xglk705-6A,B. Revise the first column to '*Xglk705-6A* {9927}^{2},{0081}^{1}, *6B* {822,0081}^{1}.'

Xglk762-6A. Revise the first column to '*Xglk762-6A* {822,0049}^{1}, {9927}^{2}.' and add 'The arm location of *Xglk762-6A* was not reported in {822}.'.

Xmwg19-6A. Revise the first column to '*Xmwg19-6A* {9927}^{2},{0081}^{1}, *6B,D* {0081}^{1}.'.

Xmwg21-6A. Revise the first column to '*Xmwg21-6A* {9927}^{2}, *6B* {0081}^{1}.'.

Xmwg74-6A,B. Revise the first column to '*Xmwg74-6A* {9927}^{2}, *6B* {900}^{1}, *6D* {0081}^{1}.'.

Xmwg798-6A,B. Revise the first column to '*Xmwg798-6A* {282}^{3},{0081}^{1}, *6B* {9927}^{2},{0081}^{1}, *6D* {0081}^{1}.'.

Xmwg2029-6A,B. Revise the first column to '*Xmwg2029-6A* {9927}^{2},{0081}^{1}, *6B* {9927}^{2},{0081}^{1}, *6D* {0081}^{1}.'.

Xpsr463(Prk)-6A,B,D. Add '{0081}.' as reference in the first column.

Xpsr908-6B. Revise the first column to '*Xpsr908-6B* {256,598}, *6D* {0081}.'.
Xuta1(Psif)-6B.1,.2. Delete.
Xwg341-6B.1,.2. Revise the first column to '*Xwg341-6B.1,.2* [{444}], *6B.3* {0081}.'

Add:

Xabg1-6B,D {0081}. ABG1.

Xabg20-6A {282}[3],{0081}[1], *6B* {0081}, *6D* {900,0081}[1]. ABG20 {664}.

The arm location of *Xabg20-6A,D* was not reported in {282} and {900}.

Xbcd102-6A {0081}, *6B* {860,0071}. BCD102. (2D).

Xbcd276-6B,D {0081}. BCD276.

Xcmwg644-6A,B,D {0081}. cMWG644.

Xcmwg653-6A,B,D {0081}. cMWG653.

A *Xcmwg653-6A* locus was previously mapped 1.5 cM proximal to the centromere on 6AS {9927}. It is likely that *Xcmwg653-6A* is, in fact, located on the long arm of 6AL {0081}.

Xcmwg716-6D {0081}. cMWG716.

Xfba1-6A {0081}. FBA1. (4B, 6BS,DS).

Xfba328-6B {0081}. FBA328.

A *Xfba328-6B* locus was previously mapped close to the centromere on 6BS {900}. The precise arm location had not been confirmed using ditelosomic analysis, and this locus may, in fact, be located on 6BL.

Xfba397-6A {900,0081}, *6B,D* {0081}. FBA397.

Xfbb215-6A {900}, *6D* {0081}. FBB215.

The arm location of *Xfbb215-6A* was not reported in {900}.

Xfbb283-6A {900,0081}. FBB283. (3B).

The arm location of *Xfbb283-6A* was not reported in {900}.

Xglk259-6A {822,0049}. pTag259. (1D).

The arm location of *Xglk259-6A* was not reported in {822}.

Xglk299-6A [{822,0049}], *6D* [{822}]. [*Xglk299a-6A* {822,0049}, *Xglk299b-6D* {822}]. pTag299.

The arm locations of *Xglk299-6A,D* were not reported in {822}.

Xglk512-6A [822,0049}]. [*Xglk512a-6A* {822,0049}]. pTag512. (4A).

The arm location of *Xglk512-6A* was not reported in {822}.

Xglk724-6A [{822,0049}], *6B,D* [{822}]. [*Xglk724e-6A* {822,0049}, *Xglk724c,b-6B,D* {822}]. pTag724. (3A,B,D, 5A).

The arm locations of *Xglk724-6A,B,D* were not reported in {822}.

Xglk756-6A [{822,0049}]. [*Xglk756b-6A* {822,0049}]. pTag756. (2D, 3B, 5A,D).

The arm locations of *Xglk756-6A* was not reported in {822}.

Xglk2003(Bza1-3)-6A [{0049}]. [*Bza1-A3* {0049}]. IHBP-1a(1). (3B, 5A,D, 6BS, 7D).

Xgwm356-6A {0035}[2]. WMS F356/WMS R356. (2A).

XksuD1-6B {860,0081}, *6D* {448}[1,4],{0081}[1]. pTtksuD1.

The arm locations of *XksuD1-6B* and *XksuD1-6D* were not reported in {860} and {448}.

Xksu908(Cbp1)-6B [{0091}]. [*Cbp1-6B* {0091}]. RRI 10 {0095}. (2B).

Xksu910(Tha1)-6B [{0091}]. [*Tha1-6B* {0091}]. CR5 {0097}. (2D, 4A, 7A,B,D).

XksuG51-6D {448}[4],{444,0081}[1].

The arm location of *XksuG51-6D* was not reported in {448} and {444}.

Xmwg514-6A,D {0081}. MWG514.

Xmwg872-6A,B,D {0081}.		MWG872.	
Xmwg911-6D {0081}.		MWG911.	
Xmwg2100-6B {0081}.		MWG2100.	
Xpsr301-6B.2 {0081}.		PSR301.	(6AS,BS,DS).
Xpsr904-6D {0081}.		PSR904.	(3A,D, 6AS).
Xpsr967-6B {0081}.		PSR967.	(1A,B, 4B, 5A, 6AS).
Xtam10-6A.2 {0081}.		TAM10.	(6AS,BS).
Xwg405-6D {0081}.		WG405.	(2D).

Group 6
Amendments:
Xabg20-6A,D. Delete (moved to 6L).
Xbcd102-6B. Delete (moved to 6L).
Xbcd357-6B. Delete (moved to 6L).
Xfba397-6A. Delete (moved to 6L).
Xfbb215-6A. Delete (moved to 6L).
Xfbb283-6A. Delete (moved to 6L).
Xglk172-6A. Delete (moved to 6S).
Xglk259-6A. Delete (moved to 6L).
Xglk299-6A,D. Delete (moved to 6L).
Xglk317-6A. Add '(1B).' in the last column.
Xglk479-6A,D. Delete (moved to 6S).
Xglk512-6A. Delete (moved to 6L).
Xglk547-6A(1),(2),(3),6B. Delete (moved to 6L).
Xglk558-6D. Add '(5D).' in the last column.
Xglk724-6A,B,D. Delete (moved to 6L).
Xglk752-6B. Delete (moved to 6S).
XksuD1-6B,D. Delete (moved to 6L).
Xtam10-6A. Revise the last column to '(6AS,BS, 6AL).'.

Add:

Xwmc104-6B {0032}.	WMC F104/WMC R104 {0037}.	

Group 7S
Amendments:
Xcdo534-7A. Revise the last column to '(1B, 6A,B,D).'.
Xglk184-7A,D. Delete (moved to 7AS:4AL:7DS).
Xglk301-7A. Revise the first column to '*Xglk301-7A* [{553}],{822}, *7B* {0031}.', add '(1B).' in the last column, and add 'The arm location of*Xglk301-7A* was not reported in {822}.'

Add:

Xcnl1-7B [{0059}].		AC1F/AC1R.	
Xkvl906(Cbp2)-7B [{0091}].	[*Cbp2-7B* {0091}].	pBH72-B8 {0092}.	(2A).
Xkvl930(Pr1)-7B,D [{0091}].	[*Pr1b-7B,D* {0091}].	HvPr1b {00104}.	
Xpsr952-7B {0031}.		PSR952.	
Xpsr955-7B {0031}.		PSR955.	

7AS:4AL:7DS
Amendments:
Xbcd907-7A. Add '(2B).' in the last column.

Xglk576-7A. Add '(7BL).' in the last column.
Xmwg710-7A,4A,7D. Add '(7BL).' in the last column.
Xpsr573-4A. Revise the first column to '*Xpsr573-4A* {255}, *7D* {0031}'

Add:

Xcnl6-7D [{0059}],{0060}.		AG24F/AG24R.	(2B).
Xglk184-7A,D [{553}].	TAG184-7A,D {553}].	pTag184 {822}.	

The map position of *Xglk184-7D* in {0031} indicated that this locus belongs to the 7AS:4AL:7DS group.

Xksu910(Tha1)-4A [{0091}].	[*Tha1-4A* {0091}].	CR5 {0097}.	(2D, 6B, 7A,B,D).
Xksu919(Lpx)-4A [{0091}].	[*Lpx-4A* {0091}].	6C02E12 {0094}.	(5A,B).

It is not clear whether *Xksu919(Lpx)-4A* belongs to the group 7AS:4AL:7DS or to the group 4AL:5AL:5BL.

Xpsp160-7A,4A {0086}.	PSP160F1/PSP160R1.
Xsun1-7A,D(Wx) {0077}.	SUN 1F/ SUN 1R.

The primers SUN 1F/SUN 1R amplify across an $(AT)_n$ microsatellite at the 3'end of waxy genes.

Group 7L
Amendments:
Xglk197-7B. Add '(2A).' in the last column.
XksuA1-7D. Add '(3B).' in the last column.
Xpsr129-7A,B,D. Add '(2A).' in the last column.
Xpsr593-7B. Add '(1B).' in last column.
Xgwm111-7D. Revise the first column to '*Xgwm111-7B* [{0031}], *7D* {9929}.', add '*Xgwm111a-7B* {0031}.' in the second column, and add '(4A).' in the last column.
Xrz476-7B. Add '(6B).' in the last column.

Add:

Xbzh232(Tha)-7A,B {[{0031}].	[*Xpwir232a,b-7A,B* {0031}].	pWIR232 {0061}.	
Xcdc1(Sod1)-7A,B,D {0054}.		SOD1.1 & SOD1.2.	
Xcnl2-7B [{0059}],{0060}.		AC14F/AC14R.	(3D),
Xglk165-7A {0031}.		pTag165 {822}.	(5B,D).
Xglk576-7B {0031}.		pTag576 {822}.	(7AS).
Xglk2003(Bza1-5)-7D [{0049}].	[*Bza1-D5* {0049}].	IHBP-1a(1).	(3B, 5A,D, 6AL, 6BS).
Xgwm260-7A {0035}[2].		WMS F260/WMS R260.	
Xksu910(Tha1)-7A,B,D [{0091}].	[*Tha1-7A,B,D* {0091}].	CR5 {0097}.	(2D, 4A, 6B).
Xkvl917(Tha2)-7A,B [{0091}].	[*Tha2-7A,B* {0091}].	pBH72-C6 {0092}.	(4A).
Xkvl927(Grp94)-7A.1,A.2, B [{0091}].	[*Grp94-7A.1,A.2,B* {0091}].	HvGRP94 {0092}.	
Xksu928(Chi1)-7B,D [{0091}].	[*Cht1b-7B,D* {0091}].	Barchi3 {0096}.	
Xksu929(Cat)-7B [{0091}].	[*Cat-7B* {0091}].	5C05D01 {0094}.	
Xmwg710-7B [{0031}].	[*Xmwg710a-7B* {0031}].	MWG710.	(1A,B,D, 7A,4A,7D).
Xpsr927-7B [{1181}],{0031}.	[*Xpsr927.1* {1181}].	PSR927.	(4A,D).

The arm location of *Xpsr927-7B* was not reported in {1181}.

Group 7
Amendments:
Xglk301-7A. Delete (moved to 7S).
Xglk558-7D. Add '(5D).' in the last column.
Xglk598-7B. Delete (moved to 7L).
Xpsr172(Lhcb1)-7A,B,D. Add '(2B).' in the last column.
Xpsr386-7A. Add '(2A).' in the last column.
Xpsr927-7B. Delete (moved to 7L).
Xwg232-7A.1. Add '(5B,D).' in the last column.
Xwg232-7A.2. Add '(5B,D).' in the last column.

Add:

Xmst101-7D {0032}.		MST F101/MST R101{0038}.	
Xksu921(Mpc1)-7D [{0091}].	[*Mpc1-7D* {0091].	c1 {0094}.	(5A).

Dormancy (seed)
Add at the bottom of the section: 'Tolerance to preharvest sprouting (PHS) in the cross SPR8198 x HD2329 was shown to be associated with *Xwmc104-6B* and *Xmst101-7D*, and may thus be controlled by two genes {0032}.'

Ear emergence

QEet.ocs-4A.1 {0047}.	4AL {0047}.	**v:**	CS/CS(Kanto107 4A) mapping population.
		ma:	Associated with *Wx-B1.*
QEet.ocs-5A.1 {0068}.	5AL {0068}.	**v:**	CS(*T. spelta* 5A)/CS(Cappelle-Desprez 5A) RI mapping population {0079}.
		ma:	Associated with *Xcdo584* and morphological locus *Q* {0068}.

Earliness *per se*
Genes for earliness *per se* {0023} affect aspects of developmental rate that are independent of responses to vernalization and photoperiod.

Eps-A1a {0024}.	3A {0023}; 3AL {0024}.	**v:**	Chinese Spring {0024}.
Eps-A1b {0024}.		**v:**	Timstein {0024}.

Temporary symbols:

EpsWi {0025}.	3A {0025}.	**v:**	Cheyenne*7/Wichita {0025}.
epsCnn {0025}.		**v:**	Cheyenne {0025}.

QEet.ocs-5A.2 {0026}.	5AL {0026}.	**ma:**	*Xcdo 412-5A - Xbcd9-5A* region {0026}.

Free-threshing habit (new section)
QTL loci mapped include:

QFt.mgb-5A {0046}.	5AL {0046}.	**tv:**	Messapia/*T. dicoccoides* MG4343 mapping population {0046}.
		ma:	Associated with *XksuG44-5A.*
QFt.mgb-6A {0046}.	*6A* {0046}.	**tv:**	Messapia/*T. dicoccoides* MG4343 mapping population {0046}.
		ma:	Associated with *Xpsr312-6A.*

Frost Resistance
Add: Responses to cold exposure and their genetics are reviewed in {0020}.

Glaucousness (Waxiness/Glossiness)
Epistatic inhibitors
W1[I]. **ma:** *Xcdo456 -2B* - 4 cM - *W1[I]* {0001}.

Height
Reduced Height
Add to preamble for *Rht-1:*
The *Rht-1*: homoeoloci are orthologous with the *D8* locus in maize and the *GAI* locus in *Arabidopsis.* They encode proteins resembling nuclear transcription factors and are involved in the sensing of gibberellin levels {0019}.

Rht-A1a {0019}. **v:** Chinese Spring {0019}. All wheats are assumed to be monomorphic.

Rht-B1g {0019}. **v:** Highbury mutants M3 103-3 and M3 103-9 {0019}. Allele *Rht-B1g* is a fast neutron-induced mutation of *Rht-B1b* and produces a tall gibberellin responsive phenotype {0019}.

Rht-D1b. **ma:** *Xpsr1871* - 1cm - *Rht-D1b* - 4cM - *Xpsr821* (*PhyA*) {0019}.

Add below *QHt.fra-1B*

QHt.ocs-4A.1 4AL {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.
ma: Associated with *Xpsr119-4A* and *Wx-B1* {0047}.

QHt.ocs-4A.2 4AS {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.
ma: Associated with *Xbcd1738-4A* and *Hd* {0047}.

QHt.ocs-5A.1 [{0068}]. [*Qt.ocs-5A.1* {0068}]. 5AL {0068}. **v:** CS(*T. spelta* 5A)/CS(Cappelle-Desprez 5A) RI mapping population {0079}.
ma: Associated with *Xcdo1088 – Xbcd9* {0068}.
This QTL coincided with a QTL for culm length, *QCl.ocs-5A.1* {0068}.

Leaf Tip Necrosis
Add at the end of the section:
'QTL for leaf tip necrosis were identified in {0050} and were named according to the rules for Wheat Gene Nomenclature by the catalogue curators.'

QLtn.sfr-1B [{0050}]. 1BS {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.
ma: Associated with *Xgwm18-1B* and *Xglk483-1B* {0050}.

QLtn.sfr-3A [{0050}]. 3A {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.
ma: Associated with *Xpsr570-3A* and *Xpsr543-3A* {0050}.

QLtn.sfr-4B.1 [{0050}]. 4B {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.
ma: Associated with *Xpsr921-4B* and *Xpsr593-4B* [{0050}].

QLtn.sfr-4B.2 [{0050}]. 4B {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.
ma: Associated with *Xpsr593-4B* and *Xpsr112-4B* [{0050}].

QLtn.sfr-4D [{0050}]. 4DL {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}
ma: Associated with *Xpsr302-4D* and *Xpsr1101-4D* [{0050}].

QLtn.sfr-5A [{0050}]. 5A {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.
ma: Associated with *Xpsr549-5A* and *Xglk163-5A* [{0050}].

QLtn.sfr-6A [{0050}]. 6A {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.

ma: Associated with *Xpsr563-6A* and *Xpsr966-6A* [{0050}].

QLtn.sfr-7B.1 [{0050}]. 7B {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.

ma: Associated with *Xpsr350* and *Xbzh232(Tha)-7B* [{0050}].

QLtn.sfr-7B.2 [{0050}]. 7B {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.

ma: Associated with *Xglk750-7B* and *Xmwg710-7B* [{0050}].

QLtn.sfr-7D [{0050}]. 7DS {0050}. **v:** Forno/*T. spelta* var. Oberkulmer mapping population {0050}.

ma: Associated with *Xpsr160-7D* and *Xgwm44-7D* [{0050}].

Lodging (new section)

QTL for lodging were identified in {0052} and were named according to the rules for Wheat Gene Nomenclature by the catalogue curators.[1]

QLd.sfr-1B [{0052}]. 1BS {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr949-1B* and *Xgwm18-1B* {0052}.

This QTL coincides with QTL for reduced height, increased culm stiffness and broader leave width {0052}.

QLd.sfr-2A [{0052}]. 2AS {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr958-2A* and *Xpsr566-2A* [{0052}].

This QTL coincides with QTL for reduced height, increased culm stiffness, broader leave width, more erect growth habit, later ear emergence and increased culm thickness {0052}.

QLd.sfr-2D [{0052}]. 2D {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr933-2D* and *Xglk529-2D* [{0052}].

QLd.sfr-3A [{0052}]. 3AS {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr598-3A* and *Xpsr570-3A* {0052}.

This QTL coincides with QTL for increased culm stiffness and reduced culm thickness {0052}.

QLd.sfr-4A [{0052}]. 4AS {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xgwm397-4A* and *Xglk315-4A* {0052}.

This QTL coincides with QTL for reduced height, increased culm stiffness and more erect growth habit {0052}.

QLd.sfr-5A [{0052}]. 5AL {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr918-5A* and *Xpsr1201-5A* [{0052}].

This QTL coincides with QTL for reduced height, increased culm stiffness, reduced leave width, more erect growth habit, later ear emergence and increased culm thickness {0052}.

QLd.sfr-5B [{0052}]. 5BL {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr370-5B* and *Xpsr580-5B* [{0052}].

This QTL coincides with QTL for increased culm stiffness, broader leaf width and more erect growth habit {0052}.

QLd.sfr-6B [{0052}]. 6BL {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr964-6B* and *Xpsr142-6B* {0052}.

QLd.sfr-7B [{0052}]. 7BL {0052}. **v:** Forno/T. spelta var. Oberkulmer mapping population {0052}.

ma: Associated with *Xpsr927-7B* and *Xpsr350-7B* {0052}.

This QTL coincides with QTL for reduced height and later ear emergence {0052}.

Nucleolus Organizer Regions

Remove the *Nor-A1* entry and replace with :

Nor-A9 {00120}. [*Nor-A1* {221,367,835,1012}]. 1AS {282,276}. **v:** *T. spelta* {221,367,835,1012}. **dv:** *T. monococcum* {658,282,276}.

Remove the *Nor-A3* entry and replace with :

Nor-A10 [*Nor-A3* {1014,658}]. 5AS {282,276}. **dv:** *T. monococcum* {282,276}, *T. urartu* IPSR (PBI) Acc. A. {00120}.

Proteins

1. Grain Protein Content

Amendments:

QGpc.ndsu-6Bb . Add '{0071}' as reference for the QTL.

Add:

QGpc.ccsu-2D 2DL {0015}. **ma:** Association with microsatellite locus *Xwmc41-2D* accounted for 19% of the variation in grain protein content between PH132 and WL711 {0015}.

1. Enzymes

2.4. α-Amylase

Addat the end of *α-Amy-1* section:

'Synthesis of α-amylase isozymes controlled by *α-Amy-1*genes on chromosomes 6A and 6D is reduced in DT6BS compared to euploid CS. This result suggests the presence of a gene(s) on the long arm of chromosome 6B, which is (are) requiredfor GA-inducedα-amylase synthesis in the aleurone {0072}.'

3. Endosperm Storage Proteins

3.1. Glutenins

Towards the end of the preamble, between the phrases 'A system of naming the *Glu-A1-1, Glu-A1-2, Glu-B1-1* and *Glu-B1-2* alleles in *T. turgidum* var. *dicoccoides* is given in {796}.' and 'Following the first listing which considers the *Glu-1* set for hexaploid wheat as a single locus, there is a provisional listing based on x- and y- type glutenins. These are not referenced.',insert the following phrase, then the paragraphthat follows it:

'In {00116}, a comparison between spelt wheats (*T. spelta*) and bread wheat has been carried out for the glutenins using a nomenclaturesystem described in {00117}.

In {00105}, the evolution of the high molecular weight glutenin loci of the A, B, D and G genomes of wheat has been explored; 30 partial allele sequences were compared, designated by Greek letters (α,β,γ, etc.) (5 of which were cited as Schlumbaum, pers. comm.; the remaining 25 have been deposited in GenBank, accession nos. X98583-X98592, X98711-X98715 and Y12401-Y12410). These partial alleles derive from all six *Glu-1-1* and *Glu-1-2* loci in current-day samples taken from seven species of wheat, as well as from DNA extracted from charred grain of two samples from archaeologicalexcavations, of 3000 and 5000 years old respectively.'

The phrase following this insert, that is, as mentioned above, 'Following the first listing which considers the *Glu-1* set for hexaploid wheat as a single locus, there is a provisional listing based on x- and y- type glutenins. These are not referenced.'should now comprise a new paragraph.

At the end of the *Glu-A1* section, that is, between the phrase: 'The uncertainty in numbers is due to the very similar electrophoretic mobilities of some of the subunits compared with others observed either in this study or previously' and the entry for the *Glu-B1* locus, add the paragraph:

'In a study including emmer wheats (*T. dicoccon*) {00115}, new subunits named 1^+ and 2^- were found in accessions MG4378/1 and MG5380/1, respectively, and provisionally assigned to *Glu-A1*. Until confirmed, they are not included in the *Glu-A1* list.'

At the end of the *Glu-B1* section, that is, between the phrase: 'it was not conclusively clear how many of these alleles were distinct from each other, or from others previously observed.' and the entry for the *Glu-D1* locus, add the paragraph:

'In a study including emmer wheats (*T. dicoccon*) {00115}, new subunits named 7^{+} (in accessions MG5400/5 and MG30835/1), 8^{-} (in accessions MG5400/5, MG30835/1, MG5333/1 and MG5507) and 13^{-} (in accession MG5282/2) were found and provisionally assigned to *Glu-B1*. Until confirmed, they are not included in the *Glu-B1* list.'

In the *Glu-3* (LMW glutenin) section, after the paragraph 'Multiple alleles at each of the three *Glu-3* wheat loci were observed {479} and effects of allelic differences on bread making quality noted {483}.', add the paragraphs:

'A series of papers {00106, 00107, 00108 and 00109} describe considerable variation in primitive wheats not present in bread wheat (A genome species *T. boeoticum*, *T. urartu*, *T. thaoudar*, *T. aegilopoides*, *T. monococcum*, and D-genome species *T. tauschii*) for the low molecular weight subunits, sufficient to use them as a source for potentially changing flour properties in bread wheat.

In {00110}, variants for LMW glutenin subunits have been reported from study of twenty-four accessions of einkorn wheat (*T. monococcum* ssp. *monococcum*). Nine of these showed two electrophoretic bands for LMW subunits, arbitrarily designated 'a' and 'b', that appeared to be associated with good bread-making quality.

In {00111}, in a study of bread and durum wheats from Portugal, the authors used the nomenclature system described in {00113} for the LMW subunits in bread wheat, and that described in {00114} for the LMW subunits in durum wheat'

3.2. Gliadins

In the preamble section, after the 'Note' that ends with the phrase: 'and {1076} studies in *T. durum* (19 electrophoretic patterns, referring only to variation in the omega-gliadins, in 243 accessions).' add the following phrase:

'In {00110}, variants for ω-gliadins have been reported from study of twenty-four accessions of einkorn wheat (*T. monococcum* ssp. *monococcum*). In {00111}, in a study of bread and durum wheats from Portugal, the authors used the nomenclature system described in {00112} for the ω-gliadins. In {00116}, a comparison between spelt wheats (*T. spelta*) and bread wheat has been carried out for the gliadins using a nomenclature system described in {00118}.'

Gli-A1 [{1334}],{1125}.	[*Gld 1A* {1415}].	1AS {150,634,1334,1607}.	**s:** CS*/Cheyenne {634}. **v:** CS {150,1334,1607}.

Delete the previous corresponding entries and substitute the following:

Gli-A1a {988}.	**v:** CS {988}; Castan {991}; Mentana {9986}; Mara {9986}; Millewa {00119}.
Gli-A1c {988}.	**v:** Ukrainka {988}; Gazul {9985}; Sava {994}; Hopps {00119}.

Omission confirmed; this allelic designation will be used for a new allele in the future:

Gli-A1s {9981}.

Delete the previous corresponding entry and substitute the following:

Gli-A1t {9985}.	**v:** Jeja del País {9985}; Milturum 553 {9981}; Strela {9981}.

After *Gli-A1u* entry, add:

Gli-A1v {9981}.	**v:** Japhet {9981}; Rouge de Bordeaux {9981}.

Gli-B1 [{1607}],{1125}.	[*Gld 1B* {1243,1415}, Gld-B1,-B2,-B3,-B4,-B5,-B6 {420}].	1B {1607}, 1BS {150,634}.	**s:** CS*/Cheyenne {634}. **v:** CS {1607,150}.

Delete the previous corresponding entries and substitute the following:

Gli-B1d {988}.			v: Chopin {991}; Dneprovskaya 521 {988}; Petrel {991}; Tiberio {9986}; Yécora {9985}; Neepawa {995}; Suneca {00119}.
Gli-B1h {988}.			v: Krasnodonka {988}; Pepital {991}; Rudi {991}; Cabezorro {9985}; Tincurrin {00119}.
Gli-B1l {988}.			v: Clement {991}; Damier {991}; Fiocco {9986}; Avrora {9981}; Kavkaz {9981}.
Gli-B1t {9985}.			v: Jeja del País {9985}.

After *Gli-B1v* entry, add:

Gli-B1w {9981}.			v: Ardica {9981}; Barbilla (MCB-1017) {9981}.
Gli-D1 [{121}],{1125}.	[*Gld 1D* {1415}, Gld-D1,-D2,-D3 {420}].	1DS {121,150,634,1334,1607}.	s: CS*/Cheyenne {634}. v: CS {121,150,1334,1607}.

Delete the previous corresponding entry and substitute the following:

Gli-D1n {9981}.			v: Blanquillo de Toledo (MCB-0950) {9981}.

After the final entry in the *Gli-D1* list (currently *Gli-D1null*), and before the paragraph beginning 'Three alleles at each of the *Gli-1-1* (omega gliadin) loci were noted {1358}.', add:

Note: *Gli-B1l* encodes secalins often associated with the 1BL.1RS translocation.

Gli-A2 [{1334}],{1125}.	[*Gld 6A* {1415}].	6A {1334}, 6AS {1122}.	v: CS.

Delete the previous corresponding entries and substitute the following:

Gli-A2c {988}.			v: Siete Cerros 66 {988}; Prinqual {991}; Loreto {9986}; Escualo {9985}; Eagle {00119}.
Gli-A2d {988}.			v: Dneprovskaya 521 {988}; Mocho Sobarriba {9985}; Kenyon (biotype) {995}.
Gli-A2h {988}.			v: Hereward {988}; Apollo {991}; N. Strampelli {9986}; Montjuich {9985}; Basalt {9981}.
Gli-A2i {988}.			v: Lesostepka 75 {988}; Krasnodonka {988}.
Gli-A2m {988}.			v: Marquis {988}; Rex {991}; Suneca {00119}.
Gli-A2o {988}.			v: Castan {991}; Touzelle {991}; Lontra {9986}; Calatrava {9985}; Glenwari {9981}.

After *Gli-A2ab* entry, add:

Gli-A2ac {9981}.			v: Blanquillo de Barcarrota (MCB-0893) {9981}.
Gli-A2ad {9981}.			v: Hembrilla Soria (MCB-1298) {9981}.
Gli-A2ae {9981}.			v: Candeal de S.Lorenzo Parrilla (MCB-0932) {9981}.

Gli-A2af {9981}. v: Barbilla de Leon (MCB-1292) {9981}.

Gli-A2ag {9981}. v: Gluclub {9981}; Tincurrin {9981}.

Gli-A2ah {9981}. v: Candeal de Nava del Rey (MCB-0892) {9981}.

Gli-A2ai {9981}. v: Blanquillo (MCB-0908) {9981}.

Gli-B2 [{1607}],{1125}. [*Gld 6B* {1415}]. 6B {1607}, 6BS {1122}. v: CS.

Delete the previous corresponding entries and substitute the following:

Gli-B2d {988}. v: Akmolinka 1{988}; Tselinnaya 20 {988}; Friedland {991}; César {9981}.

Gli-B2f {988}. v: Maris Freeman {988}; Master {991}; Basalt {9981}.

Gli-B2g {988}. v: Galahad {988}; Cappelle-Desprez {991}; Capitole {991}.

Gli-B2i {988}. v: Insignia {988}; Robin {9981}.

Gli-B2n {988}. v: Solo {988}; Japhet {9981}, Rouge de Bordeaux {9981}.

Gli-B2o {988}. v: Mara {9986}; Hardi {9981}; Rivoli {991}; Pippo {9986}; Slavjanka {9981}; Odesskaya 16 {988}.

Gli-B2r {991}. v: Genial {991}; Arminda {991}; Estica {991}.

Gli-B2s {988}. v: Saratovskaya 36 {988}; Aquila {9981}.

Gli-B2ab {991}. v: Orepi {991}; Bordier {9981}.

After *Gli-B2af* entry, add:

Gli-B2ag {9981}. v: Jeja del Pais {9985}; Barbilla de Leon (MCB-1292) {Sp.,9981}.

Gli-B2ah {9981}. v: Rojo de Humanes (MCB-1262) {9981}; Grano de Miracolo {9981}.

Gli-B2ai {9981}. v: Blanquillo (MCB-0908) {9981}.

Gli-B2aj {9981}. v: Negrete de Málaga (MCB-1754) {9981}.

Gli-B2ak {9981}. v: HY320 {9981}; Leader {9981}.

Gli-B2al {9981}. v: Dankowska {991}.

Gli-B2am {9981}. v: TM-275 {9981}; Uralochka {9981}.

Gli-B2an {9981}. v: Eagle {9981}; Glenwari {9981}.

Gli-B2ao {9981}. v: Olympic {9981}; Mokoan {9981}.

Gli-B2ap {9981}. v: Veda {9981}; Magnif 27 {9981}.

Gli-B2aq {9981}. v: Winglen {9981}; Isis {9981}.

Gli-B2ar {9981}.			v: Arbon {9981}; Roazon {9981}.
Gli-B2as {9981}.			v: Strela {9981}; Sredneuralskaya {9981}.
Gli-B2at {9981}.			v: Ranee {9981}; Javelin 48 {9981}.
Gli-D2 [{1334}], {1125}.	[*Gld 6D* {1415}].	6D {1334}, 6DS {1122}.	v: CS.

Delete the previous corresponding entries and substitute the following:

Gli-D2f {988}.	v: Rempart {991}; Créneau {991}; Kirgizskaya Yubileinaya {988}.
Gli-D2h {988}.	v: Capitole {991}; Garant {991}; Thatcher {995}; Chinook {995}; Sadovo 1 {988}; Eagle {00119}
Gli-D2i {988}.	v: Insignia 49 {00119}; Lario {9986}.
Gli-D2k {988}.	v: Skala {988}; Crvencapa {994}; Kzyl-Bas {988}.

Omission confirmed; this allelic designation will be used for a new allele in the future:

Gli-D2l.

Delete the previous corresponding entries and substitute the following:

Gli-D2m {988}.	v: Marquis {988}; Rex {991}; Veronese {9986}; Yecora {9985}; Rinconada {9985}; Suneca {00119}.
Gli-D2q {988}.	v: Soissons {991}; Fournil {991}; E. Mottin {9981}; Volshebnitsa (biotype) {988}; Winglen {9981}; Cook {9981}.

After *Glu-D2aa* entry, add:

Gli-D2ab {9981}.	v: Rojo de Boadilla de Campos (MCB-1031) {9981}.
Gli-D2ac {9981}.	v: Albatros {9981}.
Gli-D2ad {9981}.	v: Hembrilla Soria (MCB-1298) {9981}.

5. Other proteins

1.1 Waxy proteins

At end of preamble add: 'All combinations of the null alleles were produced in Chinese Spring {0018}.'

Wx-A1

Add at the bottom of the section:

'The complete genomic sequence for the *Wx-A1a* allele from CS {0073} and the cDNA sequence for the *Wx-A1b* allele from Kanto 107 {0075} have been determined.'

Wx-B1

Wx-B1e {0027}. v: Blue Boy II {0027}; Canthatch {0027}; Eureka {0027}; Götz {0027}; Norin 44 {0027}; Turkey Red {0027}.

Add at the bottom of the section:

'The complete genomic sequence for *Wx-B1a* from CS has been determined {0073}.'

Wx-D1

Add at the bottom of the section:

'The complete genomic sequence for *Wx-D1a* from CS {0073} and the cDNA sequence for the *Wx-D1b* allele from Bai Huo {0075} have been determined.'

5.7. Starch granule proteins

Add at the bottom of the '***Sgp-1*** ' section: 'The proteins, designated Sgp-1, are starch synthases, encoded by *SsII-A1*, *SsII-B1* and *SsII-D1* {0042}.'

Add at the bottom of the '***Sgp-3*** ' section: 'The proteins, designated Sgp-3, are identical to wheat starch synthase I, encoded by *SsI-A1*, *SsI-A2* and *SsI-D1* {0041}.'

5.8. Puroindolines

Pina-A ^{m}1 {0083} 5A^{m}S {0083}. **dv:** *T. monococcum* DV92, G3116 {0083}

In *T. monococcum* ***Pina-A ^{m}1*** is completely linked to ***Gsp-A ^{m}1*** {0083}.

Pina-D1

Pina-D1a {452}.
Replace 'carrying the *Pinb-D1b* mutation {452,1035}.' with 'carrying a hardness mutation in puroindoline b {452,1035,0082}.'.
Pina-D1b {1035}.
Delete the sentence starting with '*Pinb-D1a* is present ...' and replace with 'Present only in some hard hexaploid wheats.'.

Pinb-A ^{m}1 {0083} 5A^{m}S {0083}. **dv:** *T. monococcum* DV92, G3116 {0083}

In *T. monococcum* ***Pinb-A ^{m}1*** is 0.1 cM proximal to ***Pina-A ^{m}1*** and both loci are less than 36 kb apart {0083}.

Pinb-D1

Pinb-D1a {452}.
Add reference '{0082}.' at the end of the sentence starting with *Pinb-D1a* is present ...'.
Pinb-D1b {1035}.
Delete the two sentences from '*Pinb-D1b* may be present ...' and replace with '*Pinb-D1b* is a "loss-of-function" mutation resulting from the replacement of a glycine by a serine at position 46 {452}.'.

Pinb-D1c {0082}. **v:** Avle {0082}; Reno {0082};Tjalve {0082}; Bjorke {0082}; Portal {0082}.
Pinb-D1c is a "loss-of-function" mutation resulting from the replacement of a leucine by a proline at position 60 {0082}.
Pinb-D1d {0082}. **v:** Bercy {0082}; Mjolner {0082}.
Pinb-D1d is a "loss-of-function" mutation resulting from the replacement of a tryptophan by a arginine at position 44 {0082}.

Pinb-D1b, *Pinb-D1c*, or *Pinb-D1d* are present in hard hexaploid wheats not carrying the *Pina-D1b* (null) mutation {452,1035,0082}.

5.9. Starch synthase

SsI-A1 {0041}. 7A {0041}.
SsI-B1 {0041}. 7B {0041}.
SsI-D1 {0041}. 7D {0041}.
Starch synthase I proteins are identical to the starch granule proteins Sgp-3 {0041}.

SsII-A1 {0042}. 7A {0042}.
SsII-B1 {0042}. 7B {0042}.

SsII-D1 {0042}. 7D{0042}.
Starch synthase II proteins are identical to the starch granule proteins Sgp-1 {0042}.

Quality Parameters
1. **Amylose content**
QAmc.ocs-4A.1 {0047}. 4AS {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.
ma: Associated with *Xbcd1738 and Xcdo1387*{0047}.

Response to Photoperiod
Following the first paragraph replace with:
Insensitivity is dominant.
There is an orthologous gene series on the short arms of homoeologous group 2. The "a" alleles confer the insensitive response {0063}, the contrasting allele may be referred to as "b".
Ppd-A1a {0063}. [*Ppd3*{1141}]. 2AL {1268}. **v:** C591 {0057}.
Ppd-B1a {0063}. [*Ppd2*{1566}]. 2BS {1566,1268,1269} **s:** Cappelle-Desprez*/CS 2B {0058}.
v: Chinese Spring {1268}; Spica {557}; Timstein {1269}. Sharbati Sonora *Ppd-A3* {887}.
ma: *Xpsr666* - 1.2cM - *Xpsr109* - 4.4cM - *Ppd-B1* - 4.8cM - *Xpsr804* ....Cent {0062}.
Ppd-D1a {0063}. [*Ppd1*{1566}]. 2DS {1268} [2DL pre-new arm nomenclature {1328}].
s: Cappelle Desprez*/Ciano 2D {1598}; Cappelle-Desprez*/Mara 2D {1598}. CS*/Ciano 2D *Ppd-B1*{1268}.
v: Akakomugi {1604}; Bezostaya 1 {1604}; Mara {1604}; Sava {1604}; Sonora 64 {1566}. Sharbati Sonora *Ppd-D1*{887}.
Two genes control photoperiod response in *T. turgidum* {788}.
Gene *Ppd-H2* on barley chromosome 2HS may be a member of the *Ppd-1* orthologous series {766}.

Response to Salinity
K^+/Na^+ discrimination
Add at the end of the 1st sentence: '*Lophopyrum elongatum* chromosome arms 1ES, 7ES, and 7EL enhance K^+/Na^+ selectivity in wheat under salt stress {0065}.

Tenacious Glumes
Tg2 {0046}. 2BS {0046}. Derived from *T. dicoccoides*
ma: *Tg2* is associated with *Xrsq805(Embp)-2B* and *Xpsr899-2B* {0046}.

Yield Components (new section)
50-grain weight
QFgw.ocs-4A.1 {0047}. 4A {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.
ma: Associated with *Xbcd265-4A* and *Xbcd1738-4A* {0047}.

Grain weight/ear
QGwe.ocs-4A.1 {0047}. 4AS {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.
ma: Associated with *Xbcd1738-4A* {0047}.

Plant yield
QYld.ocs-4A.1 {0047}. 4AS {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.
ma: Associated with *Xbcd1738-4A* {0047}.

Spikelet number/ear
QSpn.ocs-4A.1 {0047}. 4AS {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}.

ma: Associated with *Xbcd1738-4A* {0047}.

Tiller number/plant

QTn.ocs-4A.1 4AS {0047}. **v:** CS/CS(Kanto107 4A) mapping population {0047}. {0047}.

ma: Associated with *Xpsr163-4A* {0047}.

Pathogenic Disease/Pest Reaction

Reaction to *Diuraphis noxia*

Dn1. **v:** Betta DN {0004}; Caledon {0004}; Gariep {0004}; Limpopo DN {0004}; Tugela DN {0004}.

Dn5. **v:** Palmiet DN {0004}.

Reaction to *Erysiphe graminis*

Pm1. **ma:** AFLP marker 18M1 - various *Pm1* alleles 0.9 cM {0011}.

Pm1c. **v:** Blaukorn {0011}. **ma:** AFLP marker 18M2 was diagnostic for *Pm1c* {0011}.

Pm3d. **v:** Kanzler {0011}.

Pm3g [*Mlar* {854}]. 1A {0070}. {0070}.

ma: *Pm3g* - 5.2 cM - *Gli-A5* - 1.9 cM - *Gli-A1* {0070}.

Pm4a. **ma:** *Pm4a* - 3.5 cM - AFLP markers 4aM1 and 4aM2 {0011}. *Xbcd1231* was converted to a STS marker {0069}.

Pm4b. **v:** Atlantis {0011}; Boheme {0011}; Renan {0016}. RE714 {1220}.

Pm5. **v:** Greif *Pm6* {0011}.

Pm6. **i:** CI 13250/7*Prins {0069}; CI 12559/8*Prins {0069}; PI 170914/7*Prins {0069}.

v: Greif *Pm5* {0011}.

ma: close linkage with *Xbcd135* (1.5±1.4cM), *Xbcd307* (4.7±2.5cM) and *Xbcd266* (4.5±2.4cM) {0069}.

Pm8. Add just before 'crosses between three lines ...':
1BS/1RS recombinants 2.9 cM proximal to *Gli-B1/GluB3* {0084}. **i:** MA1 and MA2 four-breakpoint double translocation lines 1RS-1BS-1RS-1BS.1BL in Pavon {0084}.'

Pm13. 3B. **v:** add: R1B {0055}; R4A {0055}; R6A {0055}.

3D. **v:** add: R2A {0055}; R2B {0055}.

Add at the end of the section: '**ma:** *Pm13* was mapped to a translocated $3S^{l}S$ segment distal to *Xcdo460-3B* {0036}.

Pm17. Add: **v:** TAM202 {0021}; Niobrara {0021}; Nekota {0021}.

Pm21. Add: **ma:** RAPD $OPH17_{1400}$ and SCAR markers $SCAR_{1400}$ and $SCAR_{1265}$ associated with *Pm21* are described in {0014}.

Pm26 {0001}. Recessive {0001}. 2BS {0001}.

s: Bethlehem*8/*T. turgidum* var. *dicoccoides* 2BS {0001}. **tv:** *T. turgidum* var. *dicoccoides* TTD140 {0001}. **ma:** Co-segregation with *Xwg516* {0001}.

Pm27 {0002}. 6B (6B-6G) {0002}. **v:** Line 146-155-T {0002}.

tv: *T. timopheevii* var. *timopheevii* K-38555 {0022}.

ma: 6BS.....*Xpsr8/Xpsr964 - Pm27 - Xpsr154/Xpsr546* .....6BL {0002}. Co-segregation with *Xpsr3131* {0002}.

Pm28 {0022}. 1B {0022}. **v:** Meri {0022}

Mlre. RE714 *Pm4b* **tv:** *T. dicoccum* 119 {1220}.

Add: *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}.

Add: List in {0028} (Finnish wheats).

Add at the end of the section:
'QTL for resistance to powdery mildew were identified in {0051} and were named according to the rules for Wheat Gene Nomenclature by the catalogue curators.'

QPm.sfr-1A [{0051}]. 1A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr1201-1A* and *Xpsr941-1A* [{0051}].

QPm.sfr-1B [{0051}]. 1B {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.
ma: Associated with *Xsfr3(LRR)-1B* and *Xpsr593-1B* [{0051}].

QPm.sfr-1D [{0051}]. 1D {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr168-1D* and *Xglk558-1D* [{0051}].

QPm.sfr-2A [{0051}]. 2A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr380-2A* and *Xglk293-2A* [{0051}].

QPm.sfr-2D [{0051}]. 2D {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr932-2D* and *Xpsr331-2D* [{0051}].

QPm.sfr-3A [{0051}]. 3A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.
ma: Associated with *Xpsr598-3A* and *Xpsr570-3A* {0051}.

QPm.sfr-3D [{0051}]. 3D {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr1196-3D* and *Xsfr2(Lrk10)-3D* [{0051}].

QPm.sfr-4A.1 [{0051}]. 4A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.
ma: Associated with *Xgwm111-4A* and *Xpsr934-4A* [{0051}].

QPm.sfr-4A.2 [{0051}]. 4A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.
ma: Associated with *Xmwg710-4A* and *Xglk128-4A* [{0051}].

QPm.sfr-4B [{0051}]. 4B {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.
ma: Associated with *Xpsr593-4B* and *Xpsr1112-4B* [{0051}].

QPm.sfr-4D [{0051}]. 4D {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno{0051}.
ma: Associated with *Xglk302-4D* and *Xpsr1101-4D* [{0051}].

QPm.sfr-5A.1 [{0051}]. 5A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr644-5A* and *Xpsr945-5A* [{0051}].

QPm.sfr-5A.2 [{0051}]. 5A {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr1194-5A* and *Xpsr918-5A* [{0051}].

QPm.sfr-5B [{0051}]. 5B {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0051}.
ma: Associated with *Xpsr580-5B* and *Xpsr143-5B* [{0051}].

QPm.sfr-6B [{0051}]. 6B {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.

ma: Associated with *Xpsr167-6B* and *Xpsr964-6B* [{0051}].

QPm.sfr-7B.1 [{0051}]. 7B {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.

ma: Associated with *Xpsr593-7B* and *Xpsr129-7B* [{0051}].

QPm.sfr-7B.2 [{0051}]. 7B {0051}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0051}.

ma: Associated with *Xglk750-7B* and *Xmwg710-7B* [{0051}].'

This QTL corresponds to *Pm5* {0051}.

Reaction to ***Fusarium graminearum***

QTL for fusarium head blight were identified in {0078}.

QFhs.ndsu-2A {0078}. 2AL {0078}. **v:** Sumai 3/Stoa RI mapping population; the QTL was contributed by Stoa {0078}.

ma: Associated with *XksuH16-2A* (LOD>3).

QFhs.ndsu-3B {0078}. 3BS {0078}. **v:** Sumai 3/Stoa RI mapping population; the QTL was contributed by Sumai 3 {0078}.

ma: Associated with *Xbcd907-3B.2* (LOD>3).

A major QTL was associated with several linked AFLP markers tentatively located in chromosome 7BL of Ning 7840 {0005}.

Mesterházy et al. {0006} reported a strong genetic correlation in resistance to different species of*Fusarium.*

Reaction to ***Heterodera avenae***

Cre1. 2BL {1579, 1580}. **ma:** A PCR-based assay was developed from*Xglk605-2B* {1580}.

Reaction to ***P. graminis***

Sr31. Add at the bottom of the section:
'1BS/1RS recombinants 4.4 cM proximal to *Gli-B1/Glu-B3* {0084}. **i:** : MA1 and MA2 four-breakpoint double translocation lined 1RS-1BS-1RS-1BS.1BL in Pavon {0084}.'

Sr38. 6M^{v} = 2MS-6MS.6ML or 2MS-6ML.6MS {0009}.

Sr44. **v:** Several 7A-7Ai#1L translocations {0089}.

Reaction to ***P. recondita***

Lr13. Add at the bottom of the section :
'**ma:** *Xpsr912-2B* - 9.1 cM - *Lr13* - 7.9 cM - *Xbcd1709-2B* - 9.8 cM - Cent. {0088}.'

Lr19. L505 {1346}; Ps29 {1346}.

Lr23 . Add at the bottom of the section:
'A QTL, which is likely to correspond to *Lr23*, was identified in the Opata 85/W-7984 (ITMI) RI mapping population. The resistance was contributed by W-7984 {0090}. **ma:** association with *Xksu904(Per2)-2B* {0090}.'

Lr26. Add at the bottom of the section:
'1BS/1RS recombinants 4.4 cM proximal to *Gli-B1/Glu-B3* {0084}. **i:** : MA1 and MA2 four-breakpoint double translocation lined 1RS-1BS-1RS-1BS.1BL in Pavon {0084}.'

Lr34 . Add at the bottom of the section:
'A QTL, which is likely to correspond to *Lr34*, was identified in the Opata 85/W-7984 (ITMI) RI mapping population. The resistance was contributed by Opata 85 {0090}. **ma:** association with *Xwg834-7D* {0090}.'

Lr35. Add at the end of the section: 'Complete cosegregation between*Lr35* and RFLP loci *Xwg996-2B*, *Xpsr540-2B* and *Xbcd260-2B* was observed. The RFLP probe BCD260 was converted to a CAPS and STS marker {0045}.'

Lr37. 6M^{v} = 2MS-6MS.6ML or 2MS-6ML.6MS {0009}.

Lr48 {0085}. Adult plant resistance {0085}. Recessive {0085}. **v:** CSP44 *Lr34* {0085}.

Lr49 {0085}. Adult plant resistance {0085}. **v:** VL404 *Lr34* {0085}.

Genotype tests: Add: {0013} (Chinese cultivars).

Add at the end of the section :
'QTL for leaf rust resistance were identified in {0050} and were named according to the rules for Wheat Gene Nomenclature by the catalogue curators.'

QLr.sfr-1B 1BS {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0050}.
[{0050}].
ma: Associated with *Xpsr949-1B* and *Xgwm18-1B* {0050}.

QLr.sfr-2B 2B {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0050}.
[{0050}].
ma: Associated with *Xpsr924-2B* and *Xglk699-2B* [{0050}].

QLr.sfr-3A 3A {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0050}.
[{0050}].
ma: Associated with *Xpsr570-3A* and *Xpsr543-3A* {0050}.

QLr.sfr-4B 4B {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0050}.
[{0050}].
ma: Associated with *Xpsr921-4B* and *Xpsr593-4B* [{0050}].

QLr.sfr-4D 4DL {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0050}.
[{0050}].
ma: Associated with *Xglk302-4D* and *Xpsr1101-4D* [{0050}].

QLr.sfr-5D 5DL {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Oberkulmer {0050}.
[{0050}].
ma: Associated with *Xpsr906-5D* and *Xpsr580-5D* [{0050}].

QLr.sfr-7B.1 7B {0050}. **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0050}.
[{0050}].
ma: Associated with *Xpsr593-7B* and *Xpsr129-7B* [{0050}].

QLr.sfr-7B.2 **v:** Forno/T. spelta var. Oberkulmer mapping population; the resistance was contributed by Forno {0050}.
[{0050}].
ma: Associated with *Xglk750-7B* and *Xmwg710-7B* [{0050}].

Reaction to *P. striiformis*

Yr2. Change listing to: Heines VII *Yr25*. Heines Peko *Yr6 Yr25*.

Yr7 . Change to: Reichersberg 42 *Yr25*.

Yr9. Add new section between the 1B=1RS.1BL and 1R1B) sections:
'1BS/1RS recombinants 4.4 cM proximal to *Gli-B1/Glu-B3* {0084}. **i:** MA1 and MA2 four-breakpoint double translocation lined 1RS-1BS-1RS-1BS.1BL in Pavon {0084}.'

Yr17. 6M^{v} = 2MS-6MS.6ML or 2MS-6ML.6MS {0009}.
Add at the end of the section: '*Yr17* is closely linked to the scar marker SC-Y15, developed from RAPD marker OP-Y15$_{580}$, and to *Xpsr150-2M^{v}* {0044}.'

Yr25. Add: **v:** Carina {0010}; Hugenoot {0010}; Tugela-DN {0010}. Heines Peko *Yr2 Yr26* {0010}. Reichersberg 42 *Yr7* {0010}.
To the sentence at end of *Yr25*, delete last phrase and add: This prediction was confirmed for Heines VII, Heines Peko and Reichersberg 42 {0010} but the pathogen culture used in {0010} was not virulent on Clement (*Yr9*) or on Strubes Dickkopf where another, or a different gene, must be present.

YrH52 {0003}. 1BS {0003}. **tv:** *T. dicoccoides* H52 {0003}.
ma: distal ...*Yr15* - 9.6 cM - *YrH52* - 1.4 cM - *Nor-B1* - 0.8 cM - *Xgwm* 264a - 0.6 cM - *Xgwm18* {0003}.

Yrns-B1 3BS {0033}. **v:** Lgst.79-74 {0033}.
{0033}.
ma: *Xgwm493* (distal) – 21 cM – *Yrns-B1* {0033}.

Reaction to *Pyrenophora tritici repentis*

1. Insensitivity to tan spot toxin

tsn1. **v:** BR34 {0007}; CEP17 {0007}; Chinese Spring {0007}; Erik {0007}; 1A807 {0007}; 1A905 {0007};

tv: Altar 84 {0007}; D87450 {0007}.

tsn1. **v:** Cheyenne {0007}; Hope {0007}; Jagger {0007}; ND485 {0007}; Timstein {0007}.

tv: Ben {0007}; Medora {0007}.

ma: *tsn1* - 3.7 cM - *Xbcd1030* {0007}.

2. Resistance to chlorosis induction

***QTsc.ndsu-1A*.** Add '{0040}' to the references to the QTL and the marker association. Add at the end of the section: '*QTsc.ndsu-1A* confers resistance in both seedlings and adult plants.'

QTsc.ndsu-4A {0090}. 4AL {0090}. **v:** Opata 85/W-7984 (ITMI) RI mapping population; the resistance was contributed by W-7984 {0090}.

ma: Association with *Xksu916(Oxo2)-4A* and *Xksu915(14-3-3a)-4A*{0090}.

Reaction to *Schizaphis graminum*

***Gb2*.** **v:** Century {0008}; TAM107 {0008}; TAM200 {0008}; TAM202 {0008}.

Genetic Linkages

2DS *Rht8* - *Ppd1* 20.9cM {0062}.

Additions to Summary Table 1

Amc	Amylose content
Bza	Basic leucine zipper protein of family 1a
Bzb	Basic leucine zipper protein of family 1b
Cbp	Chitin-binding protein
Chi	Chitinase
Eet	Ear emergence time
El	Ear length
ELIP	Early light-inducible protein
Eps	Earliness *per se*
Fgw	50-grain weight
Fhs	Reaction to *Fusarium graminearum*
Fmt	Flavonoid O-methyltransferase
Ft	Free threshing
Gpc	Grain protein content
Grp	Grp94 protein (endoplasmic heat shock protein 'endoplasmin')
Gwe	Grain weight per ear
Hrp	Hydroxyproline-rich protein
NBS	Protein that contains a nucleotide binding site
Ld	Lodging
Lpx	Lipoxygenase
LRR	Protein that contains a leucine rich repeat
Mpc1	Myb protein c1
Oxo	Oxalate oxidase
OxoLP	Oxalate oxidase-like protein
Pal	Phenylalanine ammonia lyase
Pdi	Protein disulphide isomerase

Ppo	Polyphenol oxidase
Pr	Pathogenicity related protein
Prp	Proline-rich protein
Rip	Ribosome inactivating protein
Spn	Spikelet number per ear
Tn	Tiller number per plant
Wip	Wound-induced protein
Yld	Yield
14-3-3	14-3-3- protein

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Amendments.

617. Yildirim A, Jones SS, Murray TD & Line RF 2000 Evaluation of *Daspyrum villosum* populations for resistance to cereal eyespot and stripe rust pathogens. Plant Disease 84: 40-44.
618. Update with information listed in 9963. Delete 9963
619. McIntosh RA & Lagudah ES 2000 Cytogenetical studies in wheat XVIII. Gene *Yr24* for resistance to stripe rust. Plant Breeding 119: 81-83.
9925. Crop Science 39: 805-811.
9926. Theoretical and Applied Genetics 99:16-26.
9958. Theoretical and Applied Genetics 98:1132-1137.
9960. Proc. 8th Int. Symp. Preharvest Sprouting in Cereals (Weipert D ed.): 67-76.
9961. Theoretical and Applied Genetics 98:977-984.
9985. Plant Breeding (In press).

New.

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Editorial Remarks

As you see, Wheat Information Service is refreshed with new fashion from No.91 in the memorial year of millennium. The new printing-block, A4, enables to include more information within the limit of '5-printing pages' for research articles. The double-column setting should be easy to read, and comfortable for figures and tables. The page design is also renewed from an idea of conjunction of classic genetics and breeding with molecular informations. Please arrange your bookshelf fitting with the new size.

The editorial office hopes WIS will be active for the next century. Please recommend new subscribers especially young generation.

The present issue includes important information of Wheat Gene Catalogue which is a great effort of Dr. Bob McIntosh and others. The catalogue has appeared in WIS and Annual News Letter for the year supplement, and the formal reversion will be published when International Wheat Genetics Symposium will approve it which is held every five years (the next one will be on 2003 in Italy). Send proposal or correction to the editors on it. Japanese group is discussing a convenient version of gene symbols on the web-site KOMUGI (http://www.shigen.nig.ac.jp/wheat/wheat.html).

WIS is not only an article journal but also the exchange place for information. In addition to research articles, 'Research information' (short and informal paper) is also very welcome. Records and announcements of meeting or symposium should be welcome to be printed. Send the summary or program of these meetings related to wheat genetics and breeding, which will be provided within space capacity.

Toward the next century of food sufficiency and environmental safe.

Editorial Office:
K. Nishikawa, T. Sasakuma, H. Tujimoto and K. Furukawa

Kihara Memorial
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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'.

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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.

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