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ORIGINAL PAPER

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Fusarium head blight resistance in hexaploid wheat (*Triticum aestivum*)-*Lophopyrum* genetic lines and tagging of the alien chromatin by PCR markers

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Abstract The objective of this research was to identify Fusarium head blight (FHB) resistance in wheat (*Triticum aestivum*)-*Lophopyrum* genetic lines that might complement FHB resistance in common wheat; and to identify DNA markers that can be used to tag the resistance gene in the alien chromatin (E or el_2 genome) for the development of improved wheat cultivars. FHB resistance was evaluated in 19 Chinese Spring-*Lophopyrum elongatum* (EE) substitution lines, two Thatcher-*L. ponticum* (el_1 and el_2) substitution lines, and four Thatcher-*L. ponticum* translocation lines. Significant resistance was identified in the substitution lines 7E(7A), 7E(7B), and 7E(7D). The homoeologous chromosome, $7el_2$,also showed resistance in the Thatcher genetic background. Both the Thatcher-$7el_1$ substitution and translocation lines were susceptible, like Thatcher, indicating that there is no resistance gene on the $7el_1$ chromosome. Simple sequence repeat (SSR) and cleaved amplified polymorphic sequences (CAPS) in homoeologous group 7 chromosomes were used to identify DNA markers located on 7E and $7el_2$. As expected, the transferability of wheat SSR markers to *Lophopyrum* is low. Of the 52 SSR markers that we tested, only five were found to be co-dominant on 7E of *L. elongatum* versus 7A, 7B, and 7D, one of which is also positive on $7el_2$. A CAPS marker, derived from the RFLP probe PSR129, can serve as a dominant marker for $7el_2$ chromatin.

Keywords Wheat · Lophopyrum · Fusarium head blight · Substitution · Translocation · SSR · CAPS

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Introduction

Fusarium head blight (FHB) caused by the fungus *Fusarium graminearum* has posed a serious threat to wheat production in the United States, not only causing yield and grain quality losses, but the fungus also produces a mycotoxin, deoxynivalenol (McMullen et al. 1997). Breeding wheat resistant to FHB is the method of choice to minimize crop and grain quality losses caused by the disease. Germplasm screening is the first step in a breeding program for the integration of FHB resistance into commercial wheat cultivars. However, the number of resistance genes is limited. Currently, the most effective and widely used source of FHB resistance is a QTL located on chromosome 3BS of the Chinese line, Sumai 3, and its derivatives (Waldron et al. 1999; Buerstmayr et al. 2002; Zhou et al. 2002; Shen et al. 2003b). Other QTLs in some European cultivars such as Fundulea 201R (Shen et al. 2003a) and Renan (Gervais et al. 2003) have also been reported, but their effects are not as strong and stable as that in Sumai 3 on chromosome 3BS. Exploitation of species related to wheat for resistance to FHB is necessary to broaden genetic diversity and achieve high levels of FHB resistance.

Lophopyrum (also called *Thinopyrum,* or *Agropyron*) has been an important source of resistance to several pathogens. Examples include yellow dwarf virus resistance in *Thinopyrum intermedium* (Sharma et al. 1995), leaf rust resistance in *Th. ponticum* (Sharma and Knott 1966), and yellow rust resistance in *Lophopyrum elongatum* (Ma et al. 2000). Jauhar and Peterson (2000) reported that *L. elongatum* confers resistance to *F. graminearum* that causes FHB in small grains. A set of *Triticum aestivum* (AABBDD)-*Lophopyrum elongatum* (EE) substitution lines in cv. Chinese Spring genetic background have already been developed (Dvorak 1980; Dvorak and Chen 1984; Tuleen and Hart 1988). Separately, two wheat-*Lophopyrum* substitution lines in the Thatcher genetic background 7D-$7el_1$ (Knott 1968), which was derived from the substitution line Agrus (Caldwell et al. 1956) and 7D-$7el_2$ (Knott et al. 1977), were developed.

Translocation lines were derived from the substitution line 7D-7el$_1$ (Sharma and Knott 1966) and from 7D-7el$_2$ (Kibirige-Sebunya and Knott 1983). The translocation break point and the size of the integrated *Lophopyrum* chromatin in the wheat-*Lophopyrum* translocation lines have been characterized (Kim et al. 1993). The objective of this research was to identify FHB resistance in wheat (*Triticum aestivum*)-*Lophopyrum* genetic lines that might complement FHB resistance in common wheat; and to identify DNA markers that can be used to tag the resistance in the alien (E and el$_2$ genome) chromatin for the development of improved wheat cultivars.

Materials and methods

Plant materials

Nineteen of a possible 21 disomic substitution lines in the cv. Chinese Spring (*Triticum aestivum*) background, each of which has one chromosome of *Lophopyrum elongatum* replacing the corresponding homoeologous Chinese Spring chromosome, were included in this study. The designation of the substitution lines is the respective number of the EE chromosome followed by the replaced Chinese Spring chromosome in parentheses (Dvorak 1980). Each *L. elongatum* chromosome was represented in three different lines, replacing the corresponding homoeologous chromosome of the A, B, and D genomes of wheat, except that 4E(4B) and 5E(5A) were missing.

Wheat (*T. aestivum*) cv. Thatcher, two substitution lines (K11463 and K2620) with chromosome 7 from two sources of *L. ponticum* replacing the Thatcher 7D chromosome, and four translocation stocks (K11695, KS10-2, KS24-1, and KS24-2) were also used in this study. These two sources of *Lophopyrum* chromosomes, el$_1$ and el$_2$, have been well characterized and the break points of the translocation lines between 7el$_1$-7D and 7el$_2$-7D were established using cytogenetic and molecular methodologies (Kim et al. 1993). The relationship among genomes E, el$_1$, and el$_2$ was reported by Dvorak (1975), who showed that they were homoeologous, and that the frequency of pairing was low (13.6%) between 7E and 7el$_1$, but much higher (50–80%) between 7el$_1$ and 7el$_2$.

FHB evaluation

The two sets of *Lophopyrum*-derived wheat lines were evaluated for type II FHB resistance (Schroeder and Christensen 1963) by single floret inoculation. The inoculum was a mung bean culture of a local isolate of *F. graminearum*. About 500 macrospores in 10 μl of deionized H_2O were placed into the basal floret of the third or fourth spikelet from the tip of spikes. Immediately after inoculation, a plastic bag was stapled over the inoculated spikes for 3 days to maintain high humidity. Three weeks after inoculation, disease severity, measured as the number of diseased spikelets in the inoculated spikes, was recorded. The various lines and their parents were evaluated for disease in three to four experiments conducted in a greenhouse. Data were analyzed by an unbalanced analysis of variance using the GLM program of SAS (SAS Institute, version 8.0, 1999). Both lines and experiments were considered to be random effects. Thus, the F-test was run against the mean square of the interaction term. The mean values of the lines were compared with those of their corresponding recurrent parent, Chinese Spring or Thatcher. Fisher's least significance test was used as the critical value of difference.

SSR and CAPS assays

After FHB resistance was localized to chromosome 7 of *Lophopyrum*, emphasis was placed on 7E, 7el$_1$, and 7el$_2$ for chromatin tagging using SSR and CAPS markers. DNA was extracted from the Chinese Spring, Thatcher, and the substitution and translocation lines containing chromosome 7 of *Lophopyrum*. A roller machine was used to extract DNA (Clarke et al. 1989). In brief, leaves of young seedlings were squeezed between rollers while 600 μl of a cTAB solution (TrisHCl 83.5 mM, NaCl 1.17 M, EDTA 16.7 mM, cTAB 1.67%, pH 8.0) was dripped on to them. The extract was collected in 1.5 ml Eppendorf tubes and incubated in a water bath at 65°C for 15 min, followed by extraction with chloroform-octanol and DNA precipitation. The DNA was then collected using pipette tips, dried, and dissolved in 1 ml sterile deionized water. Four microliters of this suspension were used in each PCR reaction.

Wheat SSR markers located on group 7 chromosomes were screened for polymorphisms among Chinese Spring and its three substitution lines, and Thatcher and its substitution and translocation lines. All PCR amplifications were conducted in a 25 μl reaction mix, consisting of 1×PCR buffer (Promega, Madison, Wis.), 1.5 mM $MgCl_2$, 2.0 mM dNTPs, 250 μM oligonucleotide primers, 4 μl DNA solution (about 40 ng), and 1 U *Taq* polymerase. The reactions were performed in an MJ Research Thermal Cycler (PTC-100^{TM} Thermal Controller). The samples were denatured at 94°C for 2 min, followed by 35 cycles consisting of 94°C for 30 s, 60°C for 40 s, 72°C for 1 min, with a final extension at 72°C for 7 min. PCR products were resolved on 4% Metaphor agarose gels containing ethidium bromide and visualized with a UV transilluminator.

For analysis of the CAPS marker *Xpsr129* (Marais et al. 2001), the PCR reactions were set up as for the SSR assays, but the PCR parameters were as follows: 94°C for 45 s, 60°C for 1 min, 72°C for 1.5 min. The PCR products were digested with the restriction enzyme *Hae*III and the resulting digestion products were separated in 1% agarose gels and visualized under UV light.

Results

FHB resistance derived from diploid *L. elongatum*

Chinese Spring is moderately resistant to FHB. Disease severity, expressed as the number of diseased spikelets at 21days after inoculation of one floret, was 4.5, 4.2 and 7.5 respectively in the three experiments, close to the overall mean disease severity of 4.3, 4.2, and 6.7 for all lines in these experiments (Table 1). Analysis of variance showed that there was no significant difference in the means between the first two experiments. However, the disease scores were significantly higher in the third experiment. In each experiment, there was considerable variation among the lines, which ranged from highly resistant to highly susceptible (Table 1). Lines 7E(7A), 7E(7B), and 7E(7D) consistently had the lowest disease severity. In most plants containing 7E, only the inoculated floret showed a small lesion, and some of these inoculated florets produced a seed. Occasionally, the disease spread to the second floret in the same spikelet, but not beyond. Even in experiment 3, in which the overall mean was highest, the disease severities of the three 7E substitution lines were 0.5, showing its stability across environments. In all three experiments, we included the cultivar Ning 7840, whose FHB resistance QTLs have been mapped and validated (Zhou et al. 2002; Guo et al. 2003; Zhou et al.

Table 1 Reaction to *Fusarium graminearum* in Chinese Spring and the 19 Chinese Spring-*Lophopyrum elongatum* substitution lines. Disease severity (*N*) was measured as number of diseased spikelets below and including the inoculated floret, 21days after inoculation. Experiments 1, 2, and 3 were conducted in March–April 2002, September–November 2002, and November–December 2002 respectively

Line	Experiment 1		Experiment 2		Experiment 3	
	N	Disease	*N*	Disease	*N*	Disease
1E(1A)	16	3.5±3.0	14	4.9±2.8	11	4.5±2.7*
1E(1B)	17	2.4±2.0	18	2.1±1.7*	13	5.0±3.9
1E(1D)	15	6.4±4.6	14	5.3±3.1*	11	8.3±6.2
2E(2A)	19	3.7±2.4	11	1.6±1.0*	10	6.5±3.8
2E(2B)	14	5.1±4.1	13	6.3±3.2*	11	5.3±4.0
2E(2D)	18	8.1±3.7**	18	7.4±3.0**	12	11±1.4*
3E(3A)	16	4.3± 3.2	9	3.3±2.8	10	9.9±3.8
3E(3B)	14	5.2±3.7	13	4.0±4.0	14	7.8±3.4
3E(3D)	21	8.6±4.0**	11	6.4±4.3*	10	13.6±1.5**
4E(4A)	12	3.0±1.3	13	2.3±1.4*	5	5.4±3.4
4E(4D)	14	4.4±3.3	17	5.3±3.6*	11	7.4±3.8
5E(5B)	15	3.6±3.5	16	4.0±2.9	6	5.1±4.1
5E(5D)	12	4.3±4.4	19	9.4±2.1**	–	No data available
6E(6A)	15	6.9±2.7*	20	6.7±3.3*	10	8.5±2.7
6E(6B)	16	4.9±3.4	18	8.1±2.6**	11	10.9±3.2*
6E(6D)	10	5.4±3.4	16	4.9±3.9	10	8.6±3.5
7E(7A)	13	0.6±0.2**	15	0.6±0.2**	13	0.5±0.2**
7E(7B)	19	0.6±0.2**	18	0.6±0.3**	12	0.5±0.1**
7E(7D)	20	0.7±0.2**	18	0.7±0.3**	9	0.5±0**
Chinese Spring	24	4.5±3.8	21	4.2±2.8	15	7.5±3.3
Overall	320	4.3±3.8	342	4.2±3.7	204	6.7±4.7
$LSD_{0.05}$	–	2.2	–	1.9	–	2.8
$LSD_{0.01}$	–	2.9	–	2.6	–	3.8

* Significantly different from Chinese Spring at P=0.05
** Significantly different from Chinese Spring at P=0.01

2003), as a resistant control. It appears that the resistance of the 7E substitution lines restricts the spread of the disease to a greater degree than that seen in Ning 7840. In experiment 3, the resistance of Ning 7840 was overcome by the pathogen in two plants, which showed disease severity scores of 3 and 5 respectively.

Analysis of variance showed significant differences among both the different lines and the different experiments, and also interactions between lines and experiments (data not shown). Therefore comparisons were made between Chinese Spring and its substitution lines in individual experiments. Although variations were seen, the disease resistances of the substitution lines in all three experiments, lines 7E(7A), 7E(7B), and 7E(7D), were consistently more resistant than Chinese Spring, while lines 2E(2D) and 3E(3D) were consistently more susceptible (Table 1). The disease scores of 6E(6A) and 6E(6B) were significantly higher than that of Chinese Spring in two experiments, but not in the other. Despite the significant interactions observed between the lines and the experiments, the resistance of the three 7E substitution lines was prominent. Lines 2E(2D) and 3E(3D) were the most susceptible (Fig. 1). Therefore, chromosomes 2D and 3D of Chinese Spring are likely to have a resistance QTL that inhibits the spread of FHB after infection.

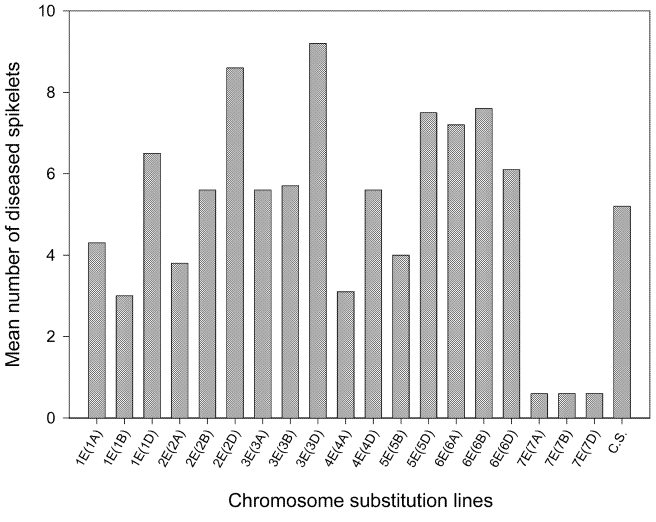

Fig. 1 Mean disease severity of Chinese Spring and its *Lophopyrum elongatum* substitution lines across three experiments. Disease was scored as number of diseased spikelets below and including the inoculated floret

FHB resistance derived from decaploid *L. ponticum*

The data from four experiments (three of which were conducted together with the Chinese Spring-*L. elongatum* substitution lines) are summarized in Table 2. The recurrent parent Thatcher was moderately susceptible to FHB; its mean disease severity ranged from 5.4–8.5 in the four experiments. The two different sources of *L. ponticum* chromosome 7 showed different responses to *Fusarium graminearum*. Lines K2620 (el_2 substitution line), KS24-1, and KS24-2 (el_2 translocation lines) consistently displayed lower disease severities than that of Thatcher. The performance of line KS10-2 was not consistent across the four experiments; its disease severity was significantly lower than that of Thatcher in two experiments, but not in the other two. The $7el_1$ deriva-

Table 2 Reaction to *Fusarium graminearum* in cv. Thatcher and its *Lophopyrum ponticum* derivatives. Disease severity (*N*) was measured as the number of diseased spikelets below and including the inoculated floret 21days after inoculation. This set of lines was evaluated in four repeated experiments conducted in November–December 2001, together with the three experiments summarized in Table 1

Lines	Experiment 0		Experiment 1		Experiment 2		Experiment 3	
	N	Disease	*N*	Disease	*N*	Disease	*N*	Disease
K11463	7	10.7±1.5	7	6.8±3.2	12	6.2±3.3	11	7.4±2.8
K2620	11	1.5±1.6**	9	2.4±2.9*	20	1.4±1.0**	9	1.3±0.8**
11695	6	9.2±1.2	17	9.2±4.7	4	7.8±5.3	10	9.4±3.1
KS10-2	8	1.3±1.2**	13	3.1±3.1	15	4.3±3.0	15	3.7±2.4**
KS24-1	8	2.8±2.2**	11	2.6±2.5*	10	1.7±0.7**	18	4.2±3.0**
KS24-2	10	2.7±2.5**	18	1.8±1.3**	12	1.3±0.7**	22	4.2±3.0**
Thatcher	11	6.9±3.8	17	5.6±3.1	14	5.4±3.7	18	8.5±3.7
Overall	61	4.6±4.1	92	4.6±4.1	87	3.5±3.2	103	5.5±3.8
$LSD_{0.05}$	–	2.4	–	2.6	–	2.3	–	2.3
$LSD_{0.01}$	–	3.2	–	3.4	–	3.0	–	3.0

* Significantly different from Thatcher at P=0.05
** Significantly different from Thatcher at P=0.01

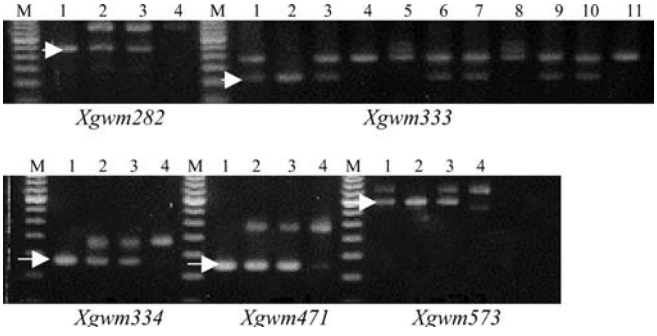

Fig. 2 SSR markers for *Lophopyrum* chromosome 7. *Arrows* indicate the DNA fragments amplified from 7E and $7el_2$. *1* 7E(7A); *2* 7E(7B); *3* 7E(7D); *4* Chinese Spring; *5* K11463; *6* K2620; *7* KS10-2; *8* K11695; *9* KS24-1; *10* KS24-2; *11* Thatcher; *M* 20 bp DNA ladder

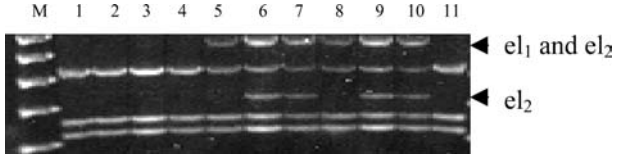

Fig. 3 CAPS marker for *L. ponticum*. The locus *Xpsr129* was amplified followed by digestion with *Hae*III. *Arrows* indicate the specific bands from the $7el_1$ and $7el_2$ chromosomes. *1* 7E(7A); *2* 7E(7B); *3* 7E(7D); *4* Chinese Spring; *5* K11463(el_1); *6* K2620(el_2); *7* KS10-2(el_2); *8* K11695(el_1); *9* KS24-1(el_2); *10* KS24-2(el_2); *11* Thatcher; *M* 20 bp DNA ladder

tives, substitution line K11463 and translocation line K11695, were both susceptible to *F. graminearum* in all four experiments. In certain experiments the disease was more severe in these el_1-derived lines than in the recurrent parent Thatcher. Analysis of variance showed that there were significant interactions between the lines and the experiments (F=1.92, P=0.01), largely due to the unstable response of KS10-2. The variance between the experiments was not significant (F=1.5, P=0.25).

PCR markers for 7E and $7el_2$

A total of 52 SSR markers located on wheat group 7 were screened to identify markers on chromosomes 7E and $7el_2$. Primers were synthesized based on publicly available sequences (Röder et al. 1998; Pestsova et al. 2000) and sequences provided from Cregan (personal communication). Most markers were not amplified from the 7E, $7el_1$, and $7el_2$ chromosomes. Typically, one band was missing with regard to the corresponding substitution chromosome. Only five SSR markers, *Xgwm282, Xgwm333, Xgwm344, Xgwm471,* and *Xgwm573*, produced a novel band compared to those from Chinese Spring (Fig. 2). Since lines 7E(7A), 7E(7B), and 7E(7D) are three replicates of the 7E chromosome, the presence of this band in all three lines indicates that this band originates from the 7E genome. The marker *Xgwm333* also produced a band of the same size (130 bp) in $7el_2$ as in 7E, but this band is absent in $7el_1$. Instead, $7el_1$ shows two specific bands of about 160 bp and 170 bp (Fig. 2).

A pair of primers designed according to the sequence of the RFLP probe PSR129 (Marais et al. 2001) amplified a DNA fragment of the same size from all lines. After digestion with restriction enzyme *Hae*III, three patterns of bands were displayed that distinguished the 7E, $7el_1$, and $7el_2$ chromosomes (Fig. 3). 7E showed no difference from 7D (Chinese Spring and Thatcher), which both had five bands; $7el_2$ had a specific band of 300 bp, and el_1 and el_2 had a common specific band of 500 bp. This probe is located on the homoeologous chromosomes 7A, 7B, and 7D in wheat, as well as in *Lophopyrum*. Apparently, mutations have occurred that result in the internal polymorphism that we observed in 7D, $7el_1$, and $7el_2$.

Discussion

FHB resistance in wheat-*Lophopyrum* genetic lines

Resistance to FHB is quantitatively expressed. The expression of resistance is determined by the interactions between the host, pathogen, and environment. The Standard deviations of disease scores are usually large, especially in intermediately resistant and susceptible lines. Thus, repeated experiments and large sample sizes are necessary to be certain of the disease resistance status.

In this study, we carried out three experiments for the evaluation of the Chinese Spring-*L. elongatum* substitution lines. The average sample size was 15 plants from each line for each individual experiment. In addition, the *L. elongatum* chromosomes, from 1E to 7E, were represented three times (except 4E and 5E, which were only represented twice) in the Chinese Spring genetic background, resulting in a total of 45 plants which were tested for each E-genome chromosome. The consistent resistance seen in lines 7E(7A), 7E(7B) and 7E(7D) across the three experiments clearly indicates that chromosome 7E of *L. elongatum* confers excellent type II resistance to the pathogen *F. graminearum*. Also, the significantly increased susceptibility in lines 2E(2D) and 3E(3D) suggests two hypotheses: either 2D and 3D from Chinese Spring carry resistance genes, or 2E and 3E of *L. elongatum* carry susceptibility genes. Since lines 2E(2A), 2E(2B), 3E(3A), and 3E(3B) did not show significant negative effects on the resistance to FHB, we suggest that Chinese Spring has resistance genes on chromosomes 2D and 3D. Yao et al. (1997) reported that when chromosome 2D of Chinese Spring was replaced by that of Sumai 3, the substitution line was more susceptible than Chinese Spring, and our data confirm their observations.

Four experiments for the evaluation of Thatcher-*L. ponticum* substitution and translocation lines were also conducted, and the average sample size was nine plants per line per experiment. The reduced disease score in the el_2 genetic lines for the substitution line K2620 and the translocation lines KS24-1 and KS24-2 support the hypothesis that $7el_2$ carries resistance to FHB. Since KS24-1 and KS24-2 are Robertsonian translocations, 7DS.$7el_2$ (Kim et al. 1993), the resistance region is located on the long arm of $7el_2$. The substitution line K11463 and the translocation line K11695 contain $7el_1$ chromatin. Data from these lines showed no reduction in their disease scores compared to that of Thatcher (Table 2), indicating that $7el_1$ does not carry resistance to FHB. The disease severity of line KS10-2 was significantly lower than that of Thatcher in two experiments at the 0.01 confidence level, but not in the other two experiments. In this line, the whole short arm and half of the long arm of chromosome 7 have been replaced by $7el_2$ (Kim et al. 1993). This line needs to be tested further for FHB resistance. If confirmed, the resistance locus should be on the proximal half of the long arm of chromosome $7el_2$. It is not known if the resistance of 7E and $7el_2$ are allelic.

PCR markers for the alien chromatin

Genetic characterization and mapping of the FHB resistance locus of *Lophopyrum* is important for the utilization of this source of resistance genes in wheat breeding. DNA markers are convenient tools for gene tagging to identify progeny that carry the desired traits. Using SSR primers designed from wheat sequences, we screened 52 primer pairs located on group 7 chromosomes, but of these, only five markers amplified a distinct band from 7E (Fig. 2). The transferability of these SSR markers between wheat and *Lophopyrum* is about 10% (5 out of 52). Homoeologous chromosomes rarely recombine; thus, any of the positive E chromatin markers are useful to indicate the presence or absence of FHB resistance. We also compared the banding patterns of these markers in wheat lines such as Ning 7840 and F201R that have been used as resistance sources in our breeding program. All markers detected polymorphisms between the different wheat lines and their 7E derivatives, so these markers are useful for pyramiding the different sources of FHB resistance. Among the five SSR markers that are positive in 7E, *Xgwm333* amplifies a DNA fragment that is also present in $7el_2$, but not in $7el_1$. The CAPS marker *Xpsr129*, previously reported in $7el_1$ (Marais et al. 2001), produced a $7el_2$-specific DNA fragment in addition to the expected el_1-specific fragment compared 7A, 7B with 7D. Thus, in total we have identified five SSR markers for chromosome 7E, and one SSR and one CAPS marker for chromosome $7el_2$. According to the SSR genetic map (Röder et al. 1998), *Xgwm471* and *Xgwm573* are located near the distal end of the short arm of 7A and 7B, respectively. *Xgwm333* is located at the centromere of 7B, while *Xgwm282* and *Xgwm344* are located in the middle of the long arms of 7A and 7B, respectively. If colinearity is applicable between wheat and *Lophopyrum*, these markers will be useful to identify recombinants and to physically map the resistance gene(s).

Breeding perspectives

The introgression of chromosome segments that carry useful genes derived from related species into cultivated wheat is a strategy frequently used in wheat breeding programs. The 1A/1R or 1B/1R translocations are excellent examples, and many disease resistance genes such as those for leaf rust and yellow dwarf viruses also have been introduced into wheat. The use of resistance gene(s) from *Lophopyrum* for controlling FHB is promising. Wheat-*Lophopyrum* recombinant lines that have FHB resistance have already been developed. The Chinese Spring-*L. elongatum* 7E substitution lines showed normal fertility. The plant type of KS24-2 is similar to Thatcher and also has normal fertility, so it may be a more desirable parent for breeding than KS24-1. The FHB resistance of these wheat-*Lophopyrum* recombinant lines is not allelic to any of the FHB resistance QTLs reported in wheat and, thus, can be pyramided.

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