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Identification of genes for leaf rust resistance in seedlings of wheat cultivars from the Yellow-Huai Basin in China and slow rusting observations in field trials

Huali Yang[1], Wenda Diao[1], Xiaocui Yan[1], Takele-Weldu Gebrewahid[2], Zaifeng Li[3], Zhanjun Yao[1]**

[1]*College of Agronomy, Hebei Agricultural University, North China Key Laboratory for Crop Germplasm Resources of China's Education Ministry, Baoding, Hebei, P.R. China*
[2]*College of Agriculture, Aksum University, Shire-Indaslassie, Tigray, Ethiopia*
[3]*College of Plant Protection, Agricultural University of Hebei/Biological Control Center for Plant Disease and Pests of Hebei Province, Baoding, Hebei, P.R. China*

**Corresponding authors: lzf7551@aliyun.com; yzhj201@aliyun.com*

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Abstract: Wheat leaf rust is a devastating disease worldwide. Identification of leaf rust resistance genes in seedlings and of genes for slow rusting are important in resistance breeding and for gene deployment to control the disease. A total of 108 wheat cultivars from the Beijing and Shandong province and a set of 36 differentials, mostly near-isogenic lines in the background of Thatcher with known leaf rust resistance genes, were tested with 20 *Puccinia triticina* pathotypes (FHJS1, FGBQ, PGJQ, SHJT, FHGQ, PHTT1, FHGQ, FHGQ, PHJS, THSM, FHSQ, PHST, PRSQ, FNTQ, PHGM, KHGQ, PHTT2, TGTT, FHJS2, NHHT) at the seedling stage in the greenhouse. The cultivars and differentials were also planted in the field to test their slow rusting resistance using a mixture of races at Baoding, Hebei province and Zhoukou, Henan province, for two consecutive years. Ten leaf rust resistance genes, *Lr1*, *9*, *10*, *19*, *20*, *24*, *26*, *34*, *37* and *46* were identified in the 57 commercial wheat cultivars, either singly or in combination, using molecular markers. Combined, the results from gene postulation and marker detection showed that one or more of the genes *Lr1*, *3*, *10*, *14a*, *14b*, *26*, *36*, *39*, *34* and *46*, were present in 57 cultivars, and that no known resistance gene was present in the remaining 51 cultivars. The resistance gene *Lr26* was present in 42 cultivars, and nine cultivars contained *Lr1*. *Lr46* was present in 10 cultivars, as indicated by the presence of the closely linked marker csLV46G22. Seven genotypes were identified as possibly carrying the gene *Lr39*. *Lr3* and *10* were found in six and four genotypes, respectively. The genes *Lr14b* and *34* were each present in three cultivars, while evidence for the presence of *Lr14a* and *36* was obtained in single genotypes. Finally, 12 cultivars showed slow rusting resistance at two locations in two crop seasons. The identification of leaf rust resistance genes in Chinese wheat cultivars will be helpful for gene deployment to control leaf rust.

Keywords: gene postulation; *Lr* genes; molecular marker; slow rusting resistance; *Puccinia triticina*; *Triticum aestivum*

Wheat leaf rust, caused by *Puccinia triticina* Eriks. (*Pt*), is one of the most important and widely distributed foliar diseases of wheat (*Triticum aestivum* L.) (McIntosh 1992; Cherukuri et al. 2005). It is adapted to a range of climates and can cause great yield losses in wide areas wherever wheat is grown (Winzeler

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et al. 2000). Leaf rust can seriously disrupt plant photosynthesis and cause yield losses of up to 40% of production (Khan et al. 2013). One of the most severe wheat leaf rust epidemics caused yield losses of up to 70% in northwestern Mexico during 1976-1977 (Dubin & Torres 1981). In China, four significant and damaging leaf rust epidemics have been documented, in 1969, 1973, 1975, and 1979 (Dong 2001). Utilizing genetically resistant cultivars is the most environmentally friendly, cost effective and long-term strategy for controlling the disease (Pink 2002).

Gene postulation provides an opportunity to quickly determine the potential resistance genes present in a large number of wheat lines. Gene postulation is widely used to identify leaf rust resistance (*Lr*) genes in various wheat cultivars or lines. Nine genes, *Lr1*, *3*, *3bg*, *10*, *13*, *14a*, *16*, *23* and *26*, were identified among 61 spring and 102 winter wheat cultivars from China (Singh et al. 1999). Fourteen *Lr* genes, *Lr1*, *2a*, *3bg*, *3ka*, *14a*, *16*, *17a*, *18*, *20*, *23*, *24*, *26*, *34* and *ZH84* were postulated with 24 *Pt* pathotypes in 65 genotypes, whereas known resistance genes were not identified in other 37 cultivars (Li et al. 2010). Twelve genes, *Lr1*, *3*, *3bg*, *3ka*, *11*, *13*, *14a*, *16*, *26*, *27*, *30* and *31* were identified to be present either individually or in combination in 84 Chinese wheat cultivars (Ren et al. 2015). Yan et al. (2021) postulated seven known *Lr* genes (*Lr1*, *13*, *18*, *14a*, *26*, *34* and *46*) 39 wheat accessions using 18 *Pt* races.

To identify *Lr* genes more accurately and efficiently, linked molecular markers have been developed in recent years. For instance, Prins et al. (2001) successfully transformed the amplified fragment length polymorphism (AFLP) marker for *Lr19* into a sequence-tagged site (STS) marker. Gupta et al. (2006) found that three sequence characterized amplified regions (SCAR) markers co-segregated with *Lr24.* Otherwise, other known *Lr* genes, including *Lr1* (Cloutier et al. 2007), *9* (Gupta et al. 2005), *10* (Schachermayr et al. 1997), *20* (Neu et al. 2002), *26* (Froidmont 1998; Mago et al. 2002; Chai et al. 2006), *37* (Bariana & McIntosh 1993), *34* (Dyck 1987), and *46* (Singh et al. 1998) could be identified using corresponding STS, SCAR or cleaved amplified polymorphic sequence (CAPS) markers.

To date, 79 *Lr* genes have been catalogued in wheat (McIntosh et al. 2016). Most of them are major genes and are believed to interact with the pathogen in a gene-for-gene relationship (Ash et al. 1996; McIntosh et al. 2003). These genes can be easily overcome by a change in *Pt* (Park et al. 1995). However, the pyramiding of a few resistance genes in a single cultivar could increase the range of resistant spectrum (Roelfs et al. 1992). Currently, only *Lr9*, *19*, *24*, and *28* are effective against prevalent Chinese *Pt* races (Yuan et al. 2007). Therefore, it is very important to find new resistance genes to deal with the rapidly evolving pathogen population. Some *Lr* genes conferring resistance at the adult plant stage rather than seedling resistance have also been characterized (Singh & Rajaram 1992). These genes include *Lr34* (Dyck 1987), *Lr37* (Dyck 1987), *46* (Singh et al. 1998), *67* (Herrera-Foessel et al. 2011; Hiebert et al. 2010), *68* (Herrera-Foessel et al. 2012). The *Lr34* gene was found in a Canadian cultivar PI58548 (Dyck 1977) and was located on chromosome 7D (Dyck 1987). Further research revealed that this locus is closely associated with the yellow rust resistance gene *Yr18* and the powdery mildew resistance gene *Pm38* (Schnurbusch et al. 2004; Lillemo et al. 2008). Gene *Lr46* is located on chromosome 1B of cultivar Pavon76 (Singh et al. 1998) and is closely linked to the stripe gene *Yr29* and powdery mildew resistance gene *Pm39* (Lillemo et al. 2008). These genes are characterized by inducing a longer pathogen latent period, smaller uredinia size, a lower infection frequency, a shorter period of sporulation and a lower spore density (Caldwell 1968; McIntosh & Bakeret al. 1968). Although slow rusting resistance has been used for durable control of leaf rust, there is little information on adult-plant resistance for leaf rust in modern wheat cultivars, and at least some of these genes have been overcome by virulence changes in *Pt* (Park & McIntosh 1994).

In China, leaf rust often occurs in the Yellow and Huai River Valley regions, and more information on resistance genes is required to control this disease effectively (Han et al. 2010). As part of this process, it is important for sustainable gene deployment and breeding new resistant cultivars to identify *Lr* genes in the currently released Chinese wheat cultivars. In this study, a set of Chinese wheat cultivars from Beijing and Shandong province were investigated to identify the seedling and adult plant *Lr* genes present, and to evaluate the effectiveness of these genes under field conditions.

MATERIAL AND METHOD

Plant materials and *Pt* pathotypes. The one hundred and eight wheat cultivars from Beijing and Shandong province (Table S1 in Electronic Supplementary Material (ESM)) were tested for response to 20 *Pt* pathotypes at the seedling stage in the greenhouse, and at adult plant growth stages in the field. A set

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of 36 differential lines carrying known leaf rust resistance genes were used as checks to postulate resistance genes. Zhengzhou 5389 and the International Maize and Wheat Improvement Center (CIMMYT) line Saar (Lillemo et al. 2008; Zhang et al. 2009) were included as highly susceptible and slow rusting checks, respectively. Tweenty *P. triticina* races used in multirace comparisons (Table 1) are maintained at the Biological Control Center for Plant Diseases and Plant Pests of Hebei, Hebei Agricultural University, China. These races were designated following the coding system of Long and Kolmer (1989) with the addition of a fourth letter for the reactions to a fourth quarter of differentials (http://www.ars.usda.gov/spzvserFiles/ ad_hoc/36400500cerealrusts/pt_nomen.pdf).

Seedling test. A set of 36 differential lines, 108 wheat cultivars and the susceptible check Zhengzhou 5389 were sown (6 to 8 seeds each) in a plastic pot (35 × 24 cm) in the greenhouse in 2016 (Table 1 and 2). When the first leaf was fully expanded, inoculations were performed through brushing urediniospores from the fully infected susceptible cultivar Zhengzhou 5389 onto all seedlings. Inoculated seedlings were immediately placed in plastic-covered cages and incubated at 15 °C and 100% relative humidity (RH) for 24 h. Then they were transferred to growth chambers maintained with 14 h light/10 h darkness at 18 to 25 °C with 70% RH. Infection types were scored at 12 to 14 days after inoculation according to the 0–4 scale system (Roelfs et al. 1992) when leaf rust was fully developed for the susceptible check Zhengzhou 5389. Cultivars with infection type (IT) 0-2+ were considered to be resistant, and those with IT 3 and 4 susceptible. Gene postulation was recorded using the method reported by Dubin et al. (1989).

Field testing. The 110 wheat cultivars, including Saar and Zhengzhou 5389, were planted in Baoding, Hebei province and Zhoukou, Henan province in a randomized complete block design in the field during 2015 to 2017 growing seasons. Approximately 50 seeds of each line were sown in a plot of 1.5 m length and 25 cm between rows. To spread urediniospores of *Pt*, the susceptible check Zhengzhou 5389 was sown adjacent and perpendicular to the test rows. Field inoculation was conducted using a mixture of equal amount of uredinospores from PHTT, THSM and TGTT *Pt* races suspended in 0.03% Tween 20 onto the spreader rows at tillering stage. Disease severity was first scored 4 weeks after inoculation using the modified scale system described by Cobb (Peterson et al. 1948), with three successive scoring at weekly intervals. The final disease severity (FDS) was used for statistical analysis.

Identification of *Lr* genes with molecular markers. Genomic DNA was extracted based on the modified cetyltrimethylammonium bromide (CTAB) method with minor modifications (Gill et al. 1991). A total of 12 STS and SCAR markers for 10 known *Lr* genes (Table S2 in ESM) were used to test the 108 wheat cultivars. PCR reactions were performed in 10 µL reaction mixture containing 1 µL genome DNA, 1 µL 10× PCR buffer, 2 mM dNTPs, 0.5 µM each forward and reverse primers, 6.7 µL ddH_2O and 1 U *Taq* DNA polymerase. PCR conditions: pre-denaturing at 94 °C for 5 min, 35 cycles (94 °C, 45 s; 52–68 °C, 45 s; 72 °C, 1 min), final extension at 72 °C for 10 min. The PCR products were separated on a 1.5% agarose gel or 12% polyacrylamide gel electrophoresis (PAGE) according to the size of product.

Statistical analysis. SAS 9.1.3 software was used to perform an analysis of variance with cultivars, environments that were the combination of location and year, and their interaction effects as fixed, and replicates nested in environments as random. The least significant difference (LSD) was used to compare the FDS values of the wheat cultivars. Genotypes with high ITs to mixed pathotypes at the seedling stage but low FDS values (no significantly higher FDS value than the slow rusting control cv. SAAR) in the field were considered to have slow rusting resistance.

RESULTS

Leaf rust resistance gene identification based on seedling reactions and molecular marker detection. Variation in infection types for 36 differential lines (Table 1) indicated that it was possible to identify 19 *Lr* genes (*Lr1*, *2a*, *2b*, *3ka*, *10*, *14a*, *15*, *17*, *26*, *30*, *18*, *21*, *23*, *3*, *36*, *39*, *3bg*, *44*, *14b*) in the 108 wheat cultivars at the seedling stage using the 20 *Pt* pathotypes. It was not possible to identify seven genes (*Lr2c*, *16*, *11*, *B*, *13*, *33*, *45*) using the 20 pathotypes due to the high infection types recorded with all the pathotypes. Ten genes (*Lr9*, *24*, *19*, *20*, *28*, *29*, *42*, *47*, *51*, and *53*) could not be detected in any tested cultivar due to low infection types to all the tested races. According to gene postulation, 10 *Lr* genes (*Lr1*, *3*, *10*, *14a*, *14b*, *Lr26*, *36*, *39*, *34*, *46*), either singly or in combination, were postulated in 57 genotypes, whereas 51 cultivars may contain unknown *Lr* genes (see further) or lacked any *Lr* gene effective against the pathotypes used.

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Table 1. Seedling infection types[a] of 36 wheat differential lines with known leaf rust resistance genes inoculated with 20 *Puccinia triticina* (*Pt*) isolates

Infection types (*Lr* genes)	*Pt* pathotypes																			
	FHJS1	FGBQ	PGJQ	SHJT	FHGQ	PHTT1	FHGQ	FHGQ	PHJS	THSM	FHSQ	PHST	PRSQ	FNTQ	PHGM	KHGQ	PHTT2	TGTT	FHJS2	NHHT
RL6003 (*Lr1*)	0	0	3	3	0	3	0	0	3	3	1	4	3	1	4	0	4	3	1	3
RL6016 (*Lr2a*)	1	1	1	3	1	1	1	0	;	3	1	1	1	1	1	3	1	4	1	2
RL6047 (*Lr2C*)	3	3	3+	3	3	3	3	3	3	3+	3	3	3+	3	3+	3	3+	3+	3	3
RL6002 (*Lr3*)	3	3+	3+	2	3	3	3	3	3	3+	3	3+	3+	3+	3+	3	4	4	4	2
RL6010 (*Lr9*)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
RL6005 (*Lr16*)	3	3	3+	3+	3	3	3+	3+	3	3+	3	3	3	3	3	4	3+	4	3+	3
RL6064 (*Lr24*)	;	;	;	;	;	;	;	;	;	;	;	;	;	;	;	;	;	;	;	;
RL6078 (*Lr26*)	3	0	1	3	3	3	3	3	3+	3	3+	3+	3	3	3	3	4	0	3	3
RL6007(*Lr3ka)*	1	2	2	1	1	3	2	2	1	3	3	3	3	3	;1	2	3	3	2+	2
RL605(*Lr11*)	3	2	3+	3	3	3+	3+	3	3	3	3+	3	3	3	3+	4	3	3+	3	3
RL6008 (*Lr17*)	3	2	3	3	2	3	2	2	3	3	3	3	3	3	2	2+	3	4	3+	2
RL6049 (*Lr30*)	2	1	2	1	2	3	1	1	2	2	2+	2	1	3+	2	1	3	3+	2+	3
RL6051 (*LrB*)	3+	3+	3+	3	3	3+	3+	3	3+	3	3	3	3	3	3	3+	3	4	3+	3
RL6004(*Lr10)*	3	3	3+	3	3	3	3	3	3+	2	3	3	3+	3	1,2	3	3+	4	3+	3
RL6013(*Lr14a)*	3	X	X	3	X	3+	2	X	3+	X	X	3	X	X	X	X	X	3	3	3	3
RL6009 (*Lr18*)	1	1	2	3	2	3	1	2	2	3	2	3	2	2	3+	2	3	3+	2+	3
RL6019 (*Lr2b*)	2	3	2+	3	3	2	3	3	2	3	3	1	3	2+	2	3	3	3	3	1+
RL6042(*Lr3bg)*	3	3	3	2	3	3	3+	3	3	3	3	3	3	3	1,2	3+	3+	3	3+	1+
RL4031 (*Lr13*)	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3+	3+	3+	3+	3+	3
RL6006(*Lr14b)*	3	3	3+	3+	3+	3	3+	3	3	3+	3+	2+	3+	3+	X	3+	3+	X	3	3+
RL6052 (*Lr15*)	1	1	1	1	3	1	;	1	3	3	2	3	1	2+	3	1	1	4	1	0
RL6040 (*Lr19*)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
RL6092 (*Lr20*)	;	1	;	;	2	;1	1	;	3	;	1	3+	;	;	;	1	1	;	;	;
RL6043 (*Lr21*)	3	2	3	2+	3	1	3	3	1	1	3	2	2	3	3	2	3	4	3	3
RL6012 (*Lr23*)	3	3	3+	2	3	3	3	3	1	2	3	3	3	3+	2	2+	3	3+	3	3
RL6079 (*Lr28*)	0	0	0	0	0	0	0	0	0	0	0	;	0	0	0	0	0	0	;	0
RL6080 (*Lr29*)	1	;1	2	2	1	1	2+	2	1	1	2	1	2	1+	1	1	2	0	1	1
RL6057 (*Lr33*)	3	3	3+	3+	3	3	3+	3	3+	3	3	3	3	3+	3+	3+	3	4	3	3
E84018 (*Lr36*)	3	3	2	3	3	3	3	3	3+	1	3	3	1	2	2	1+	2	;	1+	3
KS86NGRC02 (*Lr39*)	1	1	1,2	3+	3	3+	2	2	3+	3	2	3	2	2	3	2	3	4	1	3

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Table 1 to be continued

Infection types (*Lr* genes)	*Pt* pathotypes																			
	FHJS1	FGBQ	PGJQ	SHJT	FHGQ	PHTT1	FHGQ	FHGQ	PHJS	THSM	FHSQ	PHST	PRSQ	FNTQ	PHGM	KHGQ	PHTT2	TGTT	FHJS2	NHHT
KS91WGRC11 (*Lr42*)	1	2	1	2	2	;	2	0	;	1	0	0	2	2	1	0	0	2	1	0
RL6147 (*Lr44*)	3	2+	2	3	3	3+	2	3+	1	2	2	;1	1	3	1	2	2	;1	3	3
RL6144 (*Lr45*)	3	3+	3+	3	3	3+	3	3	3+	3+	3	3	;	4	3	3+	3+	4	3	3+
PAVON76 (*Lr47*)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
C78.5 (*Lr51*)	;	0	0	;	;	0	1	0	0	0	;	1	;	;	;	;	;	0	;	;
98M71 (*Lr53*)	0	0	0	0	0	0	0	0	0	0	0	;	0	0	0	0	;	0	0	;

[a]According to the 0–4 Stakman scale modified by Roelfs et al. (1992); infection types (ITs): 0 = immune; ; = hypersensitive fleck with no sporulation; 1 = small uredinia with necrosis; 2 = small uredinia with chlorosis; 3 = moderate size uredinia without chlorosis or necrosis; 4 = large uredinia without chlorosis or necrosis; + = slightly larger uredinia; – = slightly smaller uredinia; X = heterogeneous infection that appears several its in one leaf; FHJS1and FHJS2: the same naming of different *Pt* pathotypes

Gene *Lr26* was postulated in 42 of the 108 tested wheat cultivars, with a frequency of 38.9% (Table 2). Twenty cultivars containing *Lr26* singly, including Lumai 20, Zhongmai 875, Jingshuang 2, displayed low ITs with three *Lr26* avirulent pathotypes (FGBQ, PGJQ, TGTT) and high infection types with the remaining 17 races. *Lr26* with other known or unknown *Lr* genes were present in 22 genotypes because they conferred resistant to other pathotypes except to *Lr26* avirulent pathotypes. All cultivars carrying *Lr26* were also tested using ω-secalin and *Glu-B3* markers (Table S2 in ESM) for the presence or absence of the 1BL/1RS translocation. Nine wheat cultivars with low ITs against nine *Lr1* avirulent pathotypes were postulated to have *Lr1*. Five cultivars (Nongda 212, Yannong 24, Shannong 413863, Nongda 211, Jimai 2) carried *Lr1* and *26* with other genes based on IT pattern. The presence of *Lr1* was confirmed by the molecular marker for *Lr1*.

Seven cultivars (Yannong 24, Nongda 211, Shannong 413863, Jimai 2, Taishan 1 (high), Taishan 5, Taishan 1) were postulated to contain *Lr39* as they showed low reactions to 10 *Lr39* avirulent pathotypes. Meanwhile, these seven cultivars were also postulated to carry *Lr1* based on gene postulation. *Lr39* was present in combination with *Lr26* and other genes in four lines (Yannong 24, Shannong 413863, Nongda 211, and Jimai 2).

Gene *Lr3* was postulated in six genotypes with low ITs to two pathotypes that were avirulent to *Lr3* (SHJT and NHHT). Jining 3 is likely to carry gene *Lr3* alone because it displayed only low ITs with the two pathotypes. A further five genotypes were postulated to have *Lr3* and other *Lr* genes. *Lr10* was postulated in Taishan 9819, Taishan 1, Laizhou 137, and Fengyou 6 which were resistant to two *Lr10* avirulent pathotypes THSM and PHGM. Three cultivars (Taishan 9819, Laizhou 137 and Fengyou 6) may contain *Lr10* combined with *Lr14b* as they also showed low ITs to all the *Lr14b* avirulent pathotypes (PHST, PHGM and TGTT). *Lr10* in these cultivars was also confirmed by molecular marker for *Lr10*. *Lr14a* was only detected in one cultivar (Taishan 1). This cultivar expressed low ITs to 11 *Lr14a* avirulent pathotypes and also contained *Lr1*, *39*, *10*. Gene *Lr36* was postulated in combination with *Lr26*, *10* and *14b* in Taishan 9819. Gene *Lr46* was concluded to be present in 10 genotypes based on molecular marker analysis with csLV46G22. Six lines carried *Lr46* alone or combination with unknown *Lr* genes. *Lr46* was found in combination with *Lr26* in three genotypes (Shannong 11, Jingshuang 16 and Lunxuan 987). *Lr34* was concluded to be present in three genotypes based

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Table 2. Seedling infection types and presence or absence of leaf rust (*Lr*) resistance genes based on gene postulation using 20 *Puccinia triticina* (*Pt*) pathotypes and molecular markers of 108 wheat cultivars

Infection types				*Pt* pathotypes																			
No.	*Lr* gene based on gene postulation	*Lr* gene based on gene marker detection	*Lr* gene(s)	FHJS1	FGBQ	PGJQ	SHJT	FHGQ1	PHTT1	FHGQ2	FHGQ3	PHJS	THSM	FHSQ	PHST	PHRQ	FNTQ	PHGM	KHGQ	PHTT2	TGTT	FHJS2	NHHT
1	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3+	3	4	3	3+	4	3+	3	3+	3+	3	3	3+	4	0	3+	4
2	*Lr26*, +	*Lr26*	*Lr26*, +	3	;1	2	3+	3	4	3	4	4	3	3	3	3	3	3	3	3	0	3+	3
3	*Lr26*, +	*Lr26*	*Lr26*, +	3+	0	1+	3	3	4	3	4	4	3	3+	3	3	3	3	3	3	1	3	3
4	*Lr26*, +	*Lr26*	*Lr26*, +	4	0	2+	4	3	4	3	4	3+	3+	3	3	3	3	3	3	3+	1	3	3
5	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2+	4	3	4	3	4	3	3+	3	3	3	3	3	3	3	0	3	3
6	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3+	3+	4	3	3+	3+	3+	3	3	3	3	3	3+	3	0	3+	3+
7	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3+	3	4	3+	3+	3+	3+	3	3	3	3	3	3+	3	;	3	3+
8	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3+	3	4	3+	3+	3+	3+	3	3	3	3	3	3	3	1	3	3
9	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3	3+	4	3	3+	3	3	3	3+	3	3	3	3	3	1	3	3
10	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3+	3+	4	3	3	3+	3	3	3	3	3	3+	3	3	1	3	3+
11	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	2	3+	3+	3+	3	3+	3+	3+	3	3+	3	3	3+	3	3	;	3	3+
12	*Lr26*, +	*Lr26*	*Lr26*, +	3+	0	2	4	3+	4	3	3+	3	3+	3+	3+	3	3	3	3	3	;	3	3
13	*Lr26*, +	*Lr26*	*Lr26*, +	3+	0	2	3+	3+	3	3	3	3+	3	3	3+	3	3	3	3	3	1	3	3
14	*Lr26*, +	*Lr26*	*Lr26*, +	3+	;	2	3	3+	3+	3	3	3+	3	3	3	3	3	3	3	3	1	3	3
15	*Lr26*, +	*Lr26*	*Lr26*, +	3+	;	2	3	3	3+	3	3	3	3	3	3	3	3	3	3	3	2	3+	3
16	*Lr26*, +	*Lr26*	*Lr26*, +	3+	0	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	1	3	3
17	*Lr26*, +	*Lr26*	*Lr26*, +	3+	1	2	3	3	3+	3	3	3	3	3	3	3	3	3	3	3	1	3	3
18	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	2	3+	3	4	3+	3	3	3+	3	3	3	3	3	3+	3	;	3	3+
19	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	1+	3	3	3	3	3	3+	3	3+	3	3	3	3	3	3	1	3+	3+
20	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	2	3	3+	3	3	3	3	3+	3	3	3	3	3	3	3	1	3	3
21	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	1	3	3	3	3	3+	3	3+	3	4	3	3+	3+	3	3+	0	2	3+
22	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	2	3	3	4	3	3+	4	3+	3+	3+	3	3	3+	3+	2	0	3+	4
23	*Lr26*, +	*Lr26*	*Lr26*, +	3	;	1+	3+	2	4	3	3+	4	3+	3	3	2	3	3	3	3	1	3+	3
24	*Lr26*, +	*Lr26*	*Lr26*, +	3+	1	2	4	3	4	3	4	4	3+	3	3	3	3	3	2	3	0	3	3
25	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	2	3+	3	4	3	3	3+	3+	3	3+	3	3	3	3+	2	1	3+	3+

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Table 2 to be continued

Infection types				*Pt* pathotypes																			
No.	*Lr* gene based on gene postulation	*Lr* gene based on gene marker detection	*Lr* gene(s)	FHJS1	FGBQ	PGJQ	SHJT	FHGQ1	PHTT1	FHGQ2	FHGQ3	PHJS	THSM	FHSQ	PHST	PRSQ	FNTQ	PHGM	KHGQ	PHTT2	TGTT	FHJS2	NHT
26	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3+	3	4	3+	3+	3+	3+	3	3	3	2+	3	3	3C	0	3	3+
27	*Lr26*, +	*Lr26*	*Lr26*, +	1+	;1	2	3+	1	3+	2	3	4	3+	1	3+	2	1	3+	2	2	;	2	3+
28	*Lr26*, +	*Lr26*	*Lr26*, +	3	1	1	3	2	3	3	1	3	3	2	3+	3	3+	3	3	2	2	3+	4
29	*Lr26*, +	*Lr26*	*Lr26*, +	3	0	2	3	2	4	3	3+	3+	3	3+	3+	3	2+	3	3	3	0	3	3
30	*Lr26*, +	*Lr26, Lr46*	*Lr26, Lr46*	3	0	1+	4	3	4	3	4	4	3+	3	3	3	3	3	3	3	0	3	3
31	*Lr26*, +	*Lr26, Lr46*	*Lr26, Lr46*, +	3	;	2	3+	3+	4	3	3	3	3+	3+	3+	3	3	3	3+	3	1	0	3+
32	*Lr26, Lr3*, +	*Lr26, Lr46*	*Lr26, Lr46, Lr3*	3+	0	1	0	3	4	3	4	3+	3	3	3	3	3	3	3	3	1	3	2
33	*Lr26, Lr1*, +	*Lr26, Lr1*	*Lr26, Lr1*, +	1	0	1	3+	1	3+	2	2	3+	3+	1	3	3	2	3+	1	1	1	2	3
34	*Lr26, Lr1, Lr39*, +	*Lr26, Lr1*	*Lr26, Lr1, Lr39*, +	0	0	1	3	0	4	0	1	4	3+	1	3+	2	;1	3+	;	3+	2	;	3+
35	*Lr26, Lr1, Lr39*, +	*Lr26, Lr1*	*Lr26, Lr1, Lr39*, +	0	0	2	4	0	4	0	0	3	3	1	3	1	0	3	;	3	0	0	3
36	*Lr26, Lr1, Lr39*, +	*Lr26, Lr1*	*Lr26, Lr1, Lr39*, +	1	0	1	3	1	2+	2	1	3	3	1	3+	2	2	3	1	2	0	1	4
37	*Lr26, Lr1, Lr39*, +	*Lr26, Lr1*	*Lr26, Lr1, Lr39*, +	1	1	1	3+	2	2	1	1	3	3+	2	3+	1	1,2	3	1	1	;	2	3
38	*Lr26, Lr3*, +	*Lr26*	*Lr26, Lr3*, +	3	0	1	1	2+	4	2	3+	3	2+	2+	3	3	3	3	2	3	;	3	2
39	*Lr26, Lr3*, +	*Lr26*	*Lr26, Lr3*, +	;	0	1	1	2	4	1	3	3+	1	1	2	2	3	3	1	1,2	;	1	1+
40	*Lr26, Lr10, Lr14b*, +	*Lr26, Lr10*	*Lr26, Lr10, Lr14b*, +	1+	0	2	4	2	4	3	2	4	2	3	2	2	2	2	1	3	0	1	3
41	*Lr26, Lr10, Lr14b*, +	*Lr26, Lr10*	*Lr26, Lr10, Lr14b*, +	3+	0	2	3	3+	4	3	3	3	1	3	;	3	2	1	3	3	1	2	3
42	*Lr26, Lr10, Lr14b, Lr36*, +	*Lr26, Lr10*	*Lr26, Lr10, Lr14b, Lr36*, +	2	0	2	3+	1	4	2	2	4	2	3	2	2	2	2	2	1	;	1+	4
43	+	*Lr46*	*Lr46*, +	3	3+	3	4	3	4	3	4	4	3+	3+	3	3	3	3	3+	3+	3	3+	3
44	+	*Lr46*	*Lr46*, +	3+	3+	3	3+	3	4	3	4	4	3+	3+	3	3	3	3	3+	3	4	3	3
45	+	*Lr46*	*Lr46*, +	3+	3+	3+	3+	3	3+	3	4	4	3+	3+	3	3	3+	3	3+	3+	3+	3	3
46	+	*Lr46*	*Lr46*, +	3	3+	3+	3+	3+	4	3	4	3	3	3+	3+	3	3	3	3+	3+	3+	3	3+
47	+	*Lr46*	*Lr46*, +	3+	3+	3	2	3	4	3	4	4	3+	3+	3	3	3	3	3	3	4	3	3
48	+	*Lr46*	*Lr46*, +	3	;	1	3	3	4	3	3+	3+	3+	3	3	3	3+	3	0	3+	3	3	3
49	+	*Lr46, Lr34*	*Lr46, Lr34*, +	3	3	3	3	3	3+	3	4	3	3+	3+	3	3	3	3	3	3+	3+	3	3+

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Table 2 to be continued

Infection types				*Pt* pathotypes																			
No.	*Lr* gene based on gene postulation	*Lr* gene based on gene marker detection	*Lr* gene(s)	FHJS1	FGBQ	PGJQ	SHJT	FHGQ1	PHTT1	FHGQ2	FHGQ3	PHJS	THMT	FHSQ	PHTQ	PHRS	FNTQ	PHGM	KHGQ	PHTT2	THTT	FHJS2	NHHT
50	*Lr1*, +	*Lr1*	*Lr1*, +	0	0	2	4	0	4	0	0	3+	3+	1	3+	3	;1	3	1	3+	4	0	4
51	*Lr1*, *Lr39*, +	*Lr1*	*Lr1*, *Lr39*, +	1	1	1	3+	2	3+	1	2	3+	1	2+	1+	1	1	3	2	2	3+	;1	3
52	*Lr1*, *Lr39*, *Lr3*, +	*Lr1*	*Lr1*, *Lr39*, *Lr3*, +	0	;	2	1	;	4	1	;	4	3+	1	3+	2	1	3+	;	3+	3+	1	2
53	*Lr1*, *Lr39*, *Lr10*, *Lr14a*, +	*Lr1*, *Lr10*	*Lr1*, *Lr39*, *Lr10*, *Lr14a*, +	1	;	1	3+	1	4	1	2	3+	1	1	2+	1	1	2	1	2	3+	2	3
54	*Lr3*, +	None	*Lr3*, +	3	3+	3+	2	4	3+	3	3	3	3	3+	3+	3	3	3	3	3	3+	3+	2
55	*Lr3*, +	None	*Lr3*, +	3	3+	3	2	3	3	3	3+	3	3	3+	3+	3	3	3+	3	4	2	3	2
56	+	*Lr34*	*Lr34*, +	3+	3+	3	3	4	4	3	3	3	3	3+	3	3	3	3	3	3	3	3+	3+
57	+	*Lr34*	*Lr34*, +	1	3	3	3+	3	4	3	3	3	3	3	3	3	3	3	3	3	1	3	3
58	+	none	unknown seedling resistance gene	3	3	3	2	3	3	3	3	3	3+	3	3+	3	2	3	3	3+	3	3	3+
59	+	none	unknown seedling resistance gene	3	3	3	3	3	4	3	3+	4	3	3	3+	3	3+	3+	3	4	2	2	3
60	+	none	unknown seedling resistance gene	3	3	2+	3	2	4	3	3+	4	3+	3	3+	3	3	3+	3+	3	3+	2+	3+
61	+	none	unknown seedling resistance gene	3	3+	2	3+	3	4	3	4	4	3+	3	3	3	3	3	3	3	3+	3	3
62	+	none	unknown seedling resistance gene	0	;	2	3+	3+	4	0	;	3+	3+	1	3+	3	1	3	0	3	3	0	3+
63	+	none	unknown seedling resistance gene	3	3+	3	3+	3	3	3	4	4	3+	3	3	3	2	3	3C	3	1	3C	3
64	+	none	unknown seedling resistance gene	3	3	3+	3+	3+	4	3+	3+	2	3+	3	3	3	3	3	3+	3	3+	3	3+
65	+	none	unknown seedling resistance gene	3+	3+	3+	4	3+	4	4	3+	3	3+	3	3+	3	4	3	3+	2	4	3+	4
66	+	none	unknown seedling resistance gene	3	3	3	3+	3+	4	3+	3+	3	3+	3	3	1	3	3	3	3	3	3	3
67	+	none	unknown seedling resistance gene	3	1	2	4	1	4	2	3	4	3	2+	3+	2+	2	3+	2+	3+	3+	1+	3+

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Table 2 to be continued

Infection types				*Pt* pathotypes																				
No.	*Lr* gene based on gene postulation	*Lr* gene based on gene marker detection	*Lr* gene(s)	FHJS1	FGBQ	PGJQ	SHJT	FHGQ1	PHTT1	FHGQ2	FHGQ3	PHJS	THSM	FHSQ	PHST	PRSQ	FNTQ	PHGM	KHGQ	PHTT2	TGT	FHJS2	NHT
68	+	none	unknown seedling resistance gene	3	0	3	3	3	3+	3	3	3+	3+	2+	3+	3	3	3+	3	3	0	3	3
69	+	none	unknown seedling resistance gene	3+	3+	3+	1	3+	4	3+	3	3+	3	3+	3	3	3	3	3	3	3+	3+	3
70	+	none	unknown seedling resistance gene	3+	3	3	2	3	3	3	3	3	3	3	3+	3	3	3	3	3	3+	3	3
71	+	none	unknown seedling resistance gene	3+	3	3+	3+	3	3	3+	2	3	3	2	3	2+	3	3	1	1	0	1	3
72	+	none	unknown seedling resistance gene	1	2	3	3	2	3+	3	3	2	3	2+	2	2	2	3	2+	3	3	3	3+
73	+	none	unknown seedling resistance gene	3	3	3+	3+	3+	4	3	3	3	2	3+	2	3	3	3	3	3	3+	3+	3
74	+	none	unknown seedling resistance gene	3	3	2	3	3+	3+	3	3	3	1	3	3	3	3	3	3	3	2	3	3
75	+	none	unknown APR gene	3+	3	3+	3+	3+	3+	3+	4	3	3+	3+	3	3	4	3	3	3+	3	3+	4
76	+	none	unknown APR gene	3	3	3	3	3	4	3	3+	4	3	3	3+	3	3+	3+	3	3+	3+	3+	3+
77	+	none	unknown APR gene	3	3+	3+	3	3	4	3	4	4	3+	3+	3+	3	3	3	3+	4	4	3	4
78	+	none	unknown APR gene	3	3+	3	3+	3	4	3	4	4	3+	3+	3+	3	3	3	3	3	3+	3	3
79	+	none	unknown APR gene	3+	3	3	3+	3	4	3	4	4	3+	3	3	3	3	3	3	3	4	3	3
80	+	none	unknown APR gene	4	3+	3+	3+	3	4	3+	4	4	4	3+	3	3	3	3	3	3	3+	3	3+
81	+	none	unknown APR gene	3+	3	3+	3	3	4	3	3+	3+	3+	3	3+	3	3	3	3+	3+	3+	3	4
82	+	none	unknown APR gene	3	3	3+	4	3	4	3	3	3+	3	3	3+	3	3	3	3	3+	3	3	3

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Table 2 to be continued

Infection types				*Pt* pathotypes																			
No.	*Lr* gene based on gene postulation	*Lr* gene based on gene marker detection	*Lr* gene(s)	FHJS1	FGBQ	PGJQ	SHJT	FHGQ1	PHTT1	FHGQ2	FHGQ3	PHJS	THSM	FHSQ	PHST	PHRS	FNTQ	PHGM	KHGQ	PHTT2	TGT	FHJS2	NHHT
83	+	none	unknown APR gene	3	3	3+	3+	3	4	3	3	3	3	3	3	3	3	3	3	3	3	3+	3
84	+	none	unknown APR gene	3	3	3+	4	3+	4	3	3+	3	3	3	3	3	3	3	3	3+	3	3	3
85	+	none	unknown APR gene	3	3	3	3+	3	4	3	4	3	3	3	3	3	3	3	3	3+	3	3+	3
86	+	none	unknown APR gene	3	3+	3+	3+	3+	4	3	4	4	3+	3	3+	3	3	3	3+	3+	3	3	3+
87	+	none	unknown APR gene	3	3+	3+	4	3+	4	3	4	4	3+	3	3	3	3	3	3+	3+	3	3+	3+
88	+	none	unknown APR gene	3	3+	3+	3+	3+	4	3	3+	4	3+	3+	3	3	3	3	3+	3+	3	3	3+
89	+	none	unknown APR gene	3	3	3	3+	3	4	3+	3+	4	3	3	3+	3	3	3	3+	3	3	3+	3+
90	+	none	unknown APR gene	3	3	3+	3	3+	4	3+	3+	3	3+	3	3	3	3	3	3+	3	3+	3	3+
91	+	none	unknown APR gene	3	3	3	3	3+	4	3	3+	3	3+	3	3	3	3	3	3	3+	3	3	3
92	+	none	unknown APR gene	3	3	3+	3	3+	3	3+	3+	3+	3	3	3+	3	3	3	3	3	3+	3	3+
93	+	none	unknown APR gene	3	3	3	3	3+	3	3	3+	3+	3	3	3+	3+	3	3	3	3+	3+	3	3
94	+	none	unknown APR gene	3	3	3	3	3+	4	3	3	3	3	3	3	3	3	3+	3	3	3	3	3+
95	+	none	unknown APR gene	3+	3+	3+	3	3+	4	3	3+	3	3	3+	3	3	3	3	3	3	3+	3+	3
96	+	none	unknown APR gene	3+	3	3	3	3	3	3	3	3	3	3	3+	3	3	3	3	3+	3+	3	3
97	+	none	unknown APR gene	3+	3+	3+	3	3+	4	3+	3	3+	3	3	3	3	3	3	3+	3	3+	3	3

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Table 2 to be continued

Infection types				*Pt* pathotypes																			
No.	*Lr* gene based on gene postulation	*Lr* gene based on gene marker detection	*Lr* gene(s)	FHJS1	FGBQ	PGJQ	SHJT	FHGQ1	PHTT1	FHGQ2	FHGQ3	PHJS	THSM	FHSQ	PHST	PRSQ	FNTQ	PHGM	KHGQ	PHTT2	TGT	FHJS2	NHT
98	+	none	unknown APR gene	3+	3+	3+	3+	3+	4	3+	3	3	3	3	3	3	3	3	3	3	3+	3	3
99	+	none	unknown APR gene	3+	3	3	3	3+	3	3+	3	3	3	3	3+	3	3	3	3+	3+	4	3+	4
100	+	none	unknown APR gene	3+	3+	3+	3+	3+	3+	3+	3	3	3+	3+	3	3	3	3	3+	3+	3	3+	3
101	+	none	unknown APR gene	3	3	3+	3	3	3	3	3	3	3	3	3+	3	3	3	3	3	3+	3	3+
102	+	none	unknown APR gene	3	3	3+	3	3+	3	3	3	3	3	3	3	3	3	3	3	3	3+	3	3+
103	+	none	unknown APR gene	3	3	3+	3+	3+	3	3+	3	3	3+	3	3	3	3	3	3	3	3+	3	3+
104	+	none	unknown APR gene	3	3	3	3+	3+	3+	3	3	3+	3	3+	3	3	3	3	3	3	3+	3+	3+
105	+	none	unknown APR gene	3	3	3	3	3	3+	3+	3	3+	3	3+	3+	3	3+	3	3	4	4	4	4
106	+	none	unknown APR gene	3	3	3	3+	3+	3	3	3	3+	3	3+	3	3+	3	3	3	3	3+	3+	3
107	+	none	unknown APR gene	3	3+	3+	3+	4	3+	3+	3	3+	3	3+	3	3	3	3	3+	3	3+	3+	3
108	+	none	unknown APR gene	3	3	3	3	4	3+	3	3	3	3+	3+	3	3	3	3	3	3	3+	3	3
109	–	none	CK	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4

Infection types (ITs): 0 = immune; ; = hypersensitive fleck with no sporulation; 1 = small uredinia with necrosis; 2 = small uredinia with chlorosis; 3 = moderate size uredinia without chlorosis or necrosis; 4 = large uredinia without chlorosis or necrosis; + = slightly larger uredinia; – = slightly smaller uredinia; X = heterogeneous infection that appears several its in one leaf; FHJS1and FHJS2: the same naming of different *Pt* pathotypes; [a]gene postulated by gene postulation and molecular detection; [b]gene just postulated by gene postulation; [c]gene just postulated by molecular detection; [+]have unknown resistance gene; [–]have no gene ; APR – adult plant resistance; CK – susceptible check

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on the presence of molecular marker csLV34, and these genotypes showed symptoms of tip necrosis at the adult stage. Two genotypes contained *Lr34* in addition to unknown gene and one line has *Lr46* and *34* together.

The reactions of 17 cultivars were not in accordance with the response of any known *Lr* gene or any gene combination, and consequently the genetic composition of these lines could not be postulated. A further 34 cultivars displayed high ITs to all pathotypes, indicating that they lacked *Lr* genes that were effective against the pathotypes used.

The 108 wheat cultivars were also used to verify the specificity of the STS or SCAR markers linked to *Lr1*, *9*, *10*, *19*, *20*, *24*, *26*, *34*, *37*, and *46*. The presence of *Lr1, 10, 26* by gene postulation was confirmed by molecular detection. *Lr9*, *19*, *20*, and *24* were not present in any of these cultivars based on the seedling reaction and molecular marker detection. It was not possible to postulate *Lr34* and *46* in the seedling stage because they are adult plant resistance (APR) genes. Therefore, *Lr46* was detected in 10 cultivars followed by *Lr34* in three cultivars based on the result from molecular marker detection.

Slow rusting resistance in field. The variance analysis results showed that the genotypes interactions and the environment interactions were highly significant differences but replicates interaction was not significant. At the same time, highly significant differences were found for wheat genotypes and environment for FDS in the field trials, but its effect on variation was much less than the genotypic differences. Therefore, these suggested that the expression of wheat leaf rust resistance was mainly influenced by genotypes and environments (Table 3). In the two crop seasons, Zhengzhou 5389 scored FDS value of 85% and 95% in the two years, indicating that development for leaf rust disease was good. And slow rusting check SAAR scored FDS value of 1% and 3% in the two cropping seasons, respectively. On the basis of seedling and field evaluation, 12 wheat cultivars (Table 4) that were susceptible at the seedling stage but had low FDS in the field for all the environments were concluded to have slow rusting resistance.

DISCUSSION

The gene-for-gene hypothesis (Flor 1956) provides a basis for resistance gene postulation, so it is widely used in gene analysis to leaf rust (Yuan et al. 2007). Although this approach generates substantial genetic information in a short time, there are obvious limitations in this study due to our inability to detect all *Lr* genes (Boroujeni et al. 2011). Molecular markers, including gene identification and marker assisted selection, have been effective tools in plant breeding.

Our results indicated that at least 10 named *Lr* genes (*Lr1*, *3*, *10*, *14a*, *14b*, *26*, *36*, *39*, *34*, *46*) and several unidentified *Lr* genes are responsible for race-specific seedling resistance to leaf rust among 108 tested wheat cultivars and breeding lines. *Lr26* and *Lr1* were present either individually or in combination in 38.9% and 8.3% of the tested 108 Chinese wheat cultivars, respectively. Singh et al. (2001) and Li et al. (2010) found that *Lr26* and *Lr1* accounted for a large proportion comparing with other genes. This indicates that these two genes have been used widely in Chinese wheat breeding programs.

Lr26 on 1BL/1RS was derived from *Secale cereale*. The 1BL/1RS translocation was introduced to Chinese wheat breeding programs during the early 1970 s, after which *Lr26* became a major source of resistance to leaf rust. Many founder parents of the modern wheat cultivars contained *Lr26*, such as Lovrin 10, Lovrin 13, Kavkaz, Predgornaia and Neuzucht (He et al. 2001), leading to a high fre-

Table 3. Analysis of variance of final disease severity (FDS) in 110 wheat cultivars including slow rusting cultivar Saar and susceptible Zhengzhou 5389 checks tested in the two growing seasons

Source of variation	df	MS	*F* value	*P*
Cultivar	109	5 444.5363	74.35*	< 0.0001
Environment	1	10 877.1019	148.54*	< 0.0001
Replication	1	21.7149	0.03	0.5863
Cultivar × environment	109	244.4350	3.34**	< 0.0001
Cultivar × replication	109	55.0002	0.75	0.9644
Error	440	73.228		

df – degree of freedom; MS – mean squared error; R^2 = 0.999; *,**significance at the 0.05 and 0.01 probability

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Table 4. Infection types (ITs)[a] in the seedling test with a mixture of PHTT, TGTT and THSM pathotypes and mean final disease severity (FDS) in the field experiments with the same pathotype in the two points and two growing seasons for 12 wheat genotypes with slow rusting resistance to leaf rust

No.[b]	Cultivar (genotype)	ITs of seedlings to the pathotype mixture	FDS (%)			
			BD1	BD2	ZK1	ZK2
CK-1	Saar	4	1	3	1	3
CK-2	Zhengzhou 5389	4	80	95	85	95
8	Lunxuan 987	4	12.5	8	7.5	15
9	Yannong 23	4	10	5	3	1
10	Laizhou 953	4	7.5	5	12.5	10
11	Nongda 211	4	7.5	7.5	3	10
21	Weimai 8	3	7.5	5	10	15
31	Jingshuang 16	4	15	3	10	8
34	Yannong 24	4	10	8	1	3
40	Qingmai 6	4	15	15	20	10
58	Shannongfu 63	3	12.5	12.5	3	1
67	Yanyou 361	4	15	8	7.5	15
76	Zhongyou 335	4	15	12.5	5.5	7.5
94	Zimai 12	4	10	7.5	10	12.5
LSD	(P= 0.05)		13			

[a]According to the 0–4 Stakman scale; 4 – large uredinia; [b]line numbers corresponding to those in Table S1 in ESM; CK-1 – slow rusting check (Saar); CK-2 – susceptible check (Zhengzhou 5389); BD – Baoding, Hebei province; ZK – Zhoukou, Henan province; 1 – first year (2014–2015) planting season; 2 – second year (2015–2016) planting season

quency of *Lr26* in Chinese wheat cultivars and lines. In this study, *Lr26* in four cultivars Fengkang 2, Jiangshuang 16, Laizhou 137 and Jingdong 8 was derived from Lovrin 10. *Lr26* in Shannong M17 was derived from Laizhou 137 (Lovrin 10/Lumai 14). *Lr26* in Lumai 5, Lumai 11, Lumai 15, Jinan 16 and Lumai 8 might derived from Aimengniu (Aifeng 3//Mengxian 201/Neuzucht). *Lr26* in the cultivar Jimai 2 might be derived from Zhoumai 9 (Singh et al. 1999). Due to single cultivation and the race variation, *Lr26* gradually lost resistance.

In the present study, gene postulation combined with molecular marker detection showed that nine of the 108 wheat cultivars contained *Lr1*. *Lr1* was present widely in Chinese cultivars (Singh et al. 1999). For example, Li et al. (2010) found that seven wheat cultivars contained *Lr1*. Sichuan cultivars 351-15 and SW8588 also contained *Lr1* using genetic analysis (Zhou et al. 2012). In the study *Lr1* in Taishan 1 was originated from Bima 4 (Liu et al. 2014). *Lr1* in Yannong 19 and Yannong 24 might be derived from Shan 229 (Yan et al. 2017). Genes *Lr3*, *10*, *14a*, *14b*, *36* and *39* were present in some cultivars (Table 2). However, these postulated results require further verification because few avirulent pathotypes are available for verification.

The seedling resistance in 51 genotypes could not be determined because 17 showed resistance to many pathotypes and a further 34 genotypes showed high ITs with all pathotypes used. It could be better to use different pathotypes or additional differential lines with other resistance genes to postulate the genes in these cultivars.

The result based on the analysis of seedling ITs and field FDS showed that 12 of the 108 tested wheat cultivars conferred slow rusting resistance (Table 4). Seven cultivars, Weimai 8, Yannong 24, Laizhou 137, Lunxuan 987, Laizhou 953, Nongda 211 and Jingshuang 16, contained the *Lr26* gene in combination with other known or unknown genes; two cultivars (Lunxuan 987, Jingshuang 16) of those contained *Lr46* based on molecular detection. Three genotypes (Shannongfu 63, Yannong 23, Yanyou 361) had unknown genes. Two cultivars (Zhongyou 335 and Zimai 18) were susceptible at the seedling, but showed slow rusting resistance in the field. In the current study, 10 genotypes contained *Lr46* gene and three genotypes contained *Lr34* gene, but only

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three lines with *Lr46* displayed slow rusting resistance in the 2-year-field trials, indicating that *Lr34* or *46* combined with other minor resistance genes could provide high resistant levels of leaf rust than *Lr34* or *46* alone.

CONCLUSION

In the study, ten *Lr* genes, *Lr1*, *3*, *10*, *14a*, *14b*, *26*, *36*, *39*, *34* and *46*, either singly or in combination were identified in 57 lines. *Lr9*, *19*, *20*, *24* and *37* were not identified in all the tested cultivars. The known gene *Lr9*, 19, *24*, *28*, *29*, *47*, *51* and *53* were effective at all plant growing stages due to low infection types to all the tested races. Three and ten genotypes were possessed *Lr34* and *Lr46*, respectively. And the main resistance genes *Lr1*, *Lr26*, *Lr34* and *Lr46* accounted for 39%, 9%, 8% and 3% of the tested wheat materials respectively. Twelve cultivars showed slow leaf rusting resistance in the field. Therefore, these results contribute to the integration of resistance genes into the chinese facultative wheat genotypes and improve the genetic diversity of wheat varieties.

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