



Full Length Article

Genetic Analysis of Chinese Differential Cultivar Early Premium for Yellow Rust Resistance Genes

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ABSTRACT

To investigate the resistance genes and character of the wheat differential sets not only can monitor the dynamic development of races but also is the base of the study the physiological special. Moreover, it can improve the diagnosis of Chinese races and the resistance analysis to genic level. Early Premium, one of the Chinese wheat differential cultivars, contains unknown resistance genes to yellow rust. Genetic analysis, allelic analysis, and monosomic analysis are used in this study to determine the inheritance and chromosomal location of the major genes in Early Premium with races 2E16 and CYR18. The results are followed: Early Premium, at least, contains three major genes, which are different from known major all-stage resistance genes, and inherits by nucleus. The resistance to CYR18 is determined by three recessive complementary genes, and to 2E16 is also controlled by three recessive complementary genes. Chi-square analysis of the F₂ segregation data between a set of monosomic susceptible cultivars *Triticum spelta* and Early Premium revealed that the genes effective against race 2E16 are located on chromosomes 3B, 4D and 5D, and temporarily designated as *YrEP1*, *YrEP2* and *YrEP3*, respectively. © 2011 Friends Science Publishers

Key Words: Wheat yellow (stripe) rust; Differential cultivars; Resistance genes; Genetic analysis

INTRODUCTION

Wheat yellow (stripe) rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriksson (*Pst*), is an important wheat disease that exists all around the world. China is the biggest epidemic zone of wheat yellow rust in the world. Destructive epidemics of wheat yellow rust in China occurred in 1950, 1964, 1990 and 2002, which caused yield losses of 6.0, 3.2, 1.8, and 1.3 million tones, respectively (Li & Zeng, 2002; Wan *et al.*, 2004). Due to over-wintering and over-summering of the pathogen in China, an understanding of the yellow rust races and identification of newly evolved races is important. For the recent discovery of the perfect stage of *Pst* on *Berberis* spp. (Jin *et al.*, 2010), the importance of the sexual stage will be need further work. Presently, using wheat differential cultivars are still used to identify the virulence of races (Yang & Wu, 1990). A set of differential cultivars must have three characters: (a) stronger ability of discretion, that is, they can sensitively response to the virulence variation of physiologic races of wheat yellow rust; (b) stability to pathogens, they can not be easily affected by environmental conditions such as temperature and light; (c) the representation, they can represent the situation of varieties for different time as well as their use in the production (Wang *et al.*, 1963). In addition, identifying the resistant

gene and characterizing the wheat differential sets are the base of monitoring the dynamic development of races and the study of the physiological special (Yang & Stubbs, 1990).

The concept of wheat yellow rust differential cultivars was first internationally proposed by Gassner and Straib (1932). At present a set of differential cultivars proposed by Johnson is widely used in Europe and Australia (Australia additional Avecet R). North America and China are independent epidemic regions of yellow rust and each has their own set of differential cultivars. In addition, India also has its own differential cultivars. The composition of wheat yellow rust differential cultivars in China is open, constantly replenished important production lines or important wheat resistance resources, and properly adjusted. In 1940s, Fang (1944) first carried out the work of selection of wheat yellow rust differential cultivars. With wheat production and the change of races of *Pst*, the differential cultivars were also constantly undergone changes in number. The total number had reached 24. Currently, after many years research, combining the present practical conditions of our country, a set of Chinese wheat yellow rust differential cultivars was selected, including 19: Trigo Eureka, Fulhard, Lutescens 128, Mentana 2419, Virgilio, Abbondanza, Early Premium, Funo, Dannish 1, Jubilejinall, Fengchan 3, Lovrin 13, Kangyin 655, Suwon 11, Zhong 4, Lovrin 10, Hybrid 46,

Triticum spelta album and Guinong 22. The recent studies on resistant inheritance of wheat yellow rust differential cultivars are more in China, such as Yang and Stubbs (1990), Liu (1988, 1990), Wang *et al.* (1994), Zhao *et al.* (2006), Ma *et al.* (2006) and Gao *et al.* (2008).

The wheat cultivars Early Premium originated in Kansas, was introduced into China in 1946, and spread after 1950s in the Jizhong, Asia, the northern Henan and other regions (Zhuang *et al.*, 1994). Also it had been used to bred many wheat varieties, such as the Xuzhou 1, 3, 14, and Jinan 2. Despite the resistance to the current epidemic races in Early Premium has lost, it is still in use and plays a role in resistance identification to CYR18. The genetic background and gene(s) contained in it were not clear. However, Yang *et al.* (1994) postulated that there was unknown gene in it. In order to clearly identify Early Premium gene(s) composition, Classic genetics, allelic analysis and monosomic analysis are used in this study. Through genetic analysis based on races identification, the number of resistant gene(s), gene(s) interaction modes and resistance characteristics were analysed, as well as the similarities and differences with known gene(s) were evaluated. Through monosomic analysis, the unknown resistance gene(s) in Early Premium were located, and named by international uniform nomenclature to unknown gene(s), which could be useful for Chinese wheat scientists as well as other scientists working on rust resistance in different regions of the world.

MATERIALS AND METHODS

Wheat cultivars: Wheat varieties or lines were collected and preserved by the Institute of Plant Protection (IPP), Chinese Academy of Agricultural Sciences (CAAS), China. *T. spelta* monosomic lines were drawn from the Netherlands Research Centre for International Wheat yellow rust. The cultivars used in this study included Early Premium, Ming Xian169 (MX169) and known Yr gene carrier lines (Table I).

Pathogen materials: Two tested *Puccinia striiformis* f. sp. tritici (*Pst*) isolates, including CYR18 (from China) and 2E16 (from Indiana) were collected and preserved by IPP.

Development of crosses and generations: From 2000-2002, Early Premium, as female parent, was crossed with known genes carrier lines (male parent), the seed was sown to get the F₁ seeds and the F₁ plants were self-pollinated to obtain F₂ seeds, which were used in allelic analysis research; MX169 as female and male was crossed with Early Premium. The F₁ plants were self-pollinated to produce the F₂ seeds and backcrossed with MX169 to obtain the backcross generation (BC₁), that were used for genetic inheritance analysis; A set of *T. spelta* monosomic as female were crossed with Early Premium to obtain the F₁ seeds, which were confirmed monosomics by cytological analysis and self-pollinated to obtain the F₂ seeds.

Chromosome examination of monosomics: The squash technique of pollen mother cell (PMC) was used (Burson & Bennett, 1970). When the flag leaf, whose distance is about

5-7 cm with next leaf (called pulvinus), appears and the ear length is about 4-5 cm, this stage is called cell meiosis. The young spike is cut off, placed in Carnoy solution (100% ethanol-chloroform-glacial acetic acid, 6:3:1) pre-prepared, and numbered according sequence. After fixation 4h the smear begins. The appropriate plant inflorescences are selected, and then anthers are taken out and placed in a slide. A drop of aceto carmine stain is applied over the crushed anthers and allows 2-5 min to stain the nuclei of the cells. The anthers are cut with a blade, squeezed out of pollen mother cells, covered gently with coverslip, and observed under microscope. Monosomic plants are selected by the hysteresis characteristic of univalent chromosome in cell meiosis stage. Based on the characteristics of monosomics lagging in meiosis the monomer plants are determined. In metaphase, diploid chromosomes set in the equatorial and haploid chromosomes delayed in bipolar; during anaphase, diploid chromosomes separate and move to poles, while haploid chromosomes are in the middle.

Infection assessment and statistical analysis: Through the methods of classical genetics, allelic analysis, and monosomic analysis, wheat yellow rust resistant genes are analyzed in seedling. After soaking seeds to hasten germination with 1% H₂O₂ (V/V), each cultivar was grown in standard peat soil in 10 cm square pots containing 10 plants. Seedlings at the two leaf stage (when the first leaf was fully expanded) were inoculated *Pst* isolates with sweeping seedling (Stubbs, 1988). After inoculation, the seedlings were placed in a dew chamber at 10°C and 100% of relative humidity for 24 h and then transferred to a greenhouse maintained with 16 h light/8 h dark photoperiod at 14-18°C. Infection type (IT) was recorded 15-17 days after inoculation when rust was fully developed on the susceptible check MX169. Based on the traditional 6 scale of infection types (IT), 11 classes were used in this study (Yang *et al.*, 2008). 0 represented highly resistant with no symptom, 0⁻ represented small necrosis but no sporulation, 0⁺ represented larger or necrosis without sporulation, 1 represented some necrosis with only a trace of to slight sporulation, 1⁺ for chlorosis and necrosis associated with limited uredium development, 2 represented chlorosis flecks or necrosis with moderately sporulation, 2⁺ for chlorosis and necrosis among abundant intermediate sporulation, 3⁻ for chlorosis and necrosis among increased uredium development, 3 represented extensive sporulation production with some chlorosis, 3⁺ represented chlorosis flecks or necrosis with moderately sporulation, and 4 represented susceptible. The division standard to resistant and susceptible was adapted according to infection type levels and infection type number in the parents, F₁, F₂, and BC₁ generation to determine resistant or susceptible type (Liu, 1988). Chi-squared (χ^2) and corresponding probability (P) values were used to evaluate the goodness of fit of the observed and expected segregation ratios of F₂ and BC₁ populations.

RESULTS

Analysis of major resistance genes to 2E16 in Early Premium:

The reciprocal crosses, Early Premium×MX169 and MX169×Early Premium, including the F₁, F₂, BC₁ and parents, were inoculated by 2E16 to determine the resistance gene and inheritance characteristics in Early Premium. Result revealed that Early Premium, MX169, BC₁ generation in reciprocal crosses were all susceptible, while segragation was found in F₂ (Table II). According to the infection type levels and numbers in the parents, F₁, F₂ and BC₁ generation, infection type 0-2⁺ was as resistant plants, as well as 3⁻4 as susceptible plants. In the positive cross F₂ plants in all 222, there were 2 resistant plants and 219 susceptible plants. Chi-squared (χ^2) tests of the resistant and susceptible plants fitted a ratio of 1:63 (χ^2 {1:63} =0.63<3.84, P=0.25-0.50), indicating that the resistance in Early Premium to 2E16 was controlled by three recessive complementary genes. In the reverse cross F₂ plants in all 185, there were 5 resistant plants and 180 susceptible plants. Chi-squared (χ^2) tests of the resistant and susceptible plants fitted a ratio of 1:63 (χ^2 {1:63} =1.56<3.84, P=0.10-0.25), confirming the result that the resistance in Early Premium to 2E16 was controlled by three recessive complementary genes. The two results are consistent, indicating the resistance was inherited by nucleus.

Comparison of resistance gene in Early Premium to 2E16 with known major gene: Through allelic analysis we can determine the relationship of tested genes and known genes. The known gene carrier lines Lovrin13, Moro, Shuyun11, VPM1, Kangyin 655 and Selkirk, which were resistance to 2E16, were crossed and self-pollinated with Early Premium to obtain F₂ population, respectively. The identification and statistical analysis of F₂ seedling in the greenhouse were scored (Table III). It can be confirmed that F₂ were segregation in all crosses of resistant and susceptible plants (Table III). It showed that major resistance genes to 2E16 in Early Premium were different from the *Yr9*, *Yr10*, *Yr17*, *Yr27*, *YrSu*, *YrKy1* and *YrKy2* contained in known gene carrier lines.

Analysis of major resistance genes to CYR18 in Early Premium: To determine the resistant gene and inheritance trait of Early Premium to CYR18, reciprocal crosses, Early Premium × MX169 and MX169 × Early Premium, including F₂ and BC₁ generations were inoculated by CYR18 in seedling stage in the greenhouse. According to infection type levels and numbers in the parents, F₂ and BC₁ generation, infection type 0-2 was as resistant plants, 3⁻4 as susceptible plants (Table IV). The experiment confirmed that Early Premium was resistant, while MX169 and backcross generation (BC₁) of reciprocal crosses were susceptible. In the positive cross F₂ in all 194 plants, there were 5 resistant plants and 189 susceptible plants. Chi-squared (χ^2) tests of the resistant and susceptible plants fitted a ratio of 1:63 (χ^2 {1:63} =1.30<3.84, P=0.25-0.50), indicating that the resistance to CYR18 in Early Premium

was controlled by three recessive complementary genes. In the reverse cross F₂ in all 207 plants, there were 4 resistant plants and 203 susceptible plants. Chi-squared (χ^2) tests of the resistant and susceptible plants fitted a ratio of 1:63 (χ^2 {1:63} =0.19<3.84, P=0.05-0.10), confirming that the resistance to CYR18 in Early Premium was controlled by three recessive complementary genes. The two results are consistent, indicating the resistance was inherited by nucleus.

Comparison of resistance gene in Early Premium to CYR18 with known major gene:

The known gene carrier lines (Compair, Lovrin13, Moro, VPM1 & Kangyin 655) resistance to CYR18 were crossed and self-pollinated with Early Premium to obtain F₂ population, respectively. The identification and statistical analysis of F₂ seedling in the greenhouse were scored. It can be confirmed that F₂ were segregation in all crosses of resistant and susceptible plants (Table V). It showed that major resistance genes to CYR18 in Early Premium were different from the *Yr8*, *Yr9*, *Yr10*, *Yr17*, *Yr19*, *YrKy1* and *YrKy2* contained in known gene carrier lines.

Monosomic analysis of Early Premium for resistance to 2E16:

The resistance identification of different monosomic crosses and the parents, *T. spelta* and Early Premium were scored in Table VI. The observations results showed that it was obvious resistant in Early Premium, with infection 0;-2, while the monosomic crosses and the disomic cross were segragation of resistance and susceptible. According to resistant and susceptible segragation, infection type 0-3 was divided as resistant plants and 3⁺-4 as susceptible plants. Among F₂ plants in the 21 monosomic lines, except for three haploid 3B, 4D and 5D, 18 haploid as well as total of 18 haploid F₂ population resistant and susceptible segregation ratios fitted a ratio of 37R:27S, moreover F₂ plants in the three haploid did badly agree with expectation 37R:27S and Chi-squared (χ^2) test was greatly significant difference. It shows that Early Premium was controlled by three recessive overlapping or independent genes resistance to 2E16, which was located on chromosomes 3B, 4D and 5D, respectively.

DISCUSSION

The germplasm resources of the wheat cultivars bred in China were derived from the United States such as Early Premium, Romania such as Lovrin 10 and Lovrin 13, former Soviet such as Кавказ and Прецгорная, Italy such as St series of varieties, Chile such as Orofen, Germany such as Neuzhucht, and Mexico such as Tanori F71 and Potam S70 (Dong & Zheng, 1999); And recent year new germplasm resources are introduced into our country from the Americas and Europe through international academic exchange and cooperation and so on. These resources which possess excellent genetic genes and derivatives ability enriched wheat genetic resources in China.

Table I: Wheat cultivars or lines and pedigree

Cultivars name	Contained gene	Pedigree*
Early Premium	+	--
Compair	<i>Yr8, Yr19</i>	CS*3/ <i>Ae. Comosa</i> // <i>Ae. speltoides</i> /3/*CS
Moro	<i>Yr10, YrMor</i>	P.I.178383/Omar
VPM1	<i>Yr17</i>	<i>Ae. ventricosa</i> / <i>T. persicum</i> //3*Marne
Suwon 11	<i>YrSu</i>	--(Korea)
Selkirk	<i>Yr27</i>	Mcmurachy/Exchange//3 × Redman
Ming Xian 169	no	Chinese Shanxi native wheat variety

Note: + unknown gene * pedigree mainly origin from <http://www.ars-grin.gov>; -- unknown

Table II: Inheritance analysis of the cross MX169 and Early Premium for yellow rust resistance to 2E16

Parents and crosses	Generation	Infection type								Expected ratio	χ^2	P			
		0	0;	0;+	1	1+	2	2+	3				3	3+	4
MX169	P ₁											30			
Early Premium	P ₂		5	7	5										
MX169/Early Premium	F ₂	2						1	1			218	1:63	0.06	0.75-0.90
	BC ₁											40	0:1		
Early Premium/MX169	F ₁											3	0:1		
	F ₂	5						1		3		176	1:63	3.40	0.05-0.10
	BC ₁											16	0:1		

Table III: Seedling resistance of the cross Early Premium with known gene carrier lines for F₂ inoculated with race 2E16

Parents and crosses		Infection type		F ₂ population		Segregation or not
P ₁	P ₂	P ₁	P ₂	Resistant plants	Susceptible plants	
Early Premium	Lovrin13	0;-1	0-0;	161	75	Segregation
Early Premium	Moro	0;-1	0-0;	175	52	Segregation
Early Premium	Shuyun11	0;-1	0-0;	5	195	Segregation
Early Premium	VPM1	0;-1	0-0;	21	158	Segregation
Early Premium	Kangyin655	0;-1	0-0;	103	106	Segregation
Early Premium	Selkirk	0;-1	0;-1	23	167	Segregation

Table IV: Inheritance analysis of the cross MX169 and Early Premium for yellow rust resistance to CYR18

Parents and crosses	Generation	Infection type								Expected ratio	χ^2	P		
		0	0;	0;+	1	1+	2	2+	3				3	3+
MX169	P ₁											30		
Early Premium	P ₂	4			4		5							
MX169/Early Premium	F ₂			1		2	2		4	28	157	1:63	1.30	0.25-0.50
	BC ₁										46	0:1		
EarlyPremium/MX169	F ₂				1	1	2	8	5	51	139	1:63	0.19	0.50-0.75
	BC ₁										15	0:1		

Early Premium with the pedigree unknown was introduced from the United States. Wang *et al.* (1994) previously researched and found that Early Premium contains unknown gene(s) or gene combination. The resistance gene(s) in Early Premium was analyzed by classic genetics for populations of reciprocal crosses, MX169 × Early Premium and Early Premium × MX169. Results showed that its resistance to CYR18 and 2E16 are all controlled by three recessive complementary genes, and the results of reciprocal crosses were the same, indicating that the resistance is inherited by nucleus. Allelic analysis revealed that three pairs of major resistance genes to CYR18 in Early Premium were different from the *Yr8*, *Yr9*, *Yr10*, *Yr17*, *Yr19*, *YrKy1* and *YrKy2*, and to 2E16 were different from the *Yr9*, *Yr10*, *Yr17*, *Yr27*, *YrKy1*, *YrKy2* and *YrSu*.

Seedling resistance identification showed that there were three recessive complementary genes in Early Premium controlling resistance to 2E16. In addition, the resistance to 2E16 in Early Premium was controlled by three recessive overlapping or independent genes. The monosomic cross of *T. spelta* and Early Premium population showed the genes were located on chromosomes 3B, 4D and 5D, respectively. The two aspects results about the genetic analysis can affirm that there are three recessive complementary genes controlling the resistance to 2E16 in Early Premium. So more detailed studies should be continued about whether the genes are unknown gene or not.

According to available data, the present major resistance genes, which have named and tentatively entitled on chromosomes 3B, 4D and 5D, respectively are namely, *Yr30*, *YrSte* on chromosomes 3B; *YrJh2*, *Yr28*, *Yr22* on 4D;

Table V: Seedling resistance of the cross Early Premium with known gene carrier lines for F₂ inoculated with race CYR18

Parents and crosses		Infection type		F ₂ population		Segregation or not
P ₁	P ₂	P ₁	P ₂	Resistant plants	Susceptible plants	
Early Premium	Compare	0;-1	0;-1	54	202	Segregation
Early Premium	Lovrin 13	0;-1	0-0;	167	44	Segregation
Early Premium	More	0;-1	0-0;	178	46	Segregation
Early Premium	VPM1	0;-1	0-0;	18	220	Segregation
Early Premium	Kangyin 655	0;-1	0-0;	102	97	Segregation

Table VI: Segregation of F₂ populations in haploid and diploid of the crosses *T. spelta* with Early Premium

Line	The total number of plants	Segregation		χ^2 value (37R:27S)	P value
		Resistant plants	Susceptible plants		
1A	202	107	95	1.94	0.10-0.25
2A	227	137	90	0.61	0.25-0.50
3A	190	100	90	2.07	0.10-0.25
4A	221	125	96	0.14	0.50-0.75
5A	220	122	98	0.50	0.25-0.50
6A	221	141	80	3.23	0.05-0.10
7A	185	102	83	0.55	0.25-0.50
1B	223	120	103	1.45	0.10-0.25
2B	230	125	105	1.14	0.25-0.50
3B	201	81	120	25.27**	<0.05
4B	223	116	107	3.05	0.05-0.10
5B	188	100	88	1.65	0.10-0.25
6B	159	81	78	3.06	0.05-0.10
7B	210	131	79	1.80	0.10-0.25
1D	227	129	98	0.09	0.75-0.90
2D	243	137	106	0.21	0.50-0.75
3D	215	132	83	1.13	0.25-0.50
4D	229	108	121	10.56**	<0.05
5D	203	83	120	23.90**	<0.05
6D	227	141	86	1.73	0.10-0.25
7D	72	38	34	0.74	0.25-0.50
The total number of plants (exc. 3B,4D,5D)	3683	2084	1599	2.27	0.10-0.25

Yr40, *YrDa2* on 5D (<http://www.ars.usda.gov/Main/docs.htm?docid=10342>). However, *Yr30* and *Yr46* are adult plant resistance genes (Singh *et al.*, 2005; Herrera-Foessel *et al.*, 2011); *Yr28* was obtained from *T. tauschii* W-219; *Yr40* was derived from *Aegilops geniculata* (Kuraparthi *et al.*, 2007). Therefore, the resistance genes in Early Premium are different from the three genes.

Jinghe 8811 (carring gene *YrJh2*) conferred resistance to CYR31 (Zhang *et al.*, 2001), while Early Premium did not confer resistance to CYR31. Therefore, it is impossible to contain *YrJh2* in Early Premium. Allelic analysis showed that Lee was susceptible to CYR18 and 2E16, but Early Premium was resistance to the two races. Therefore, Early Premium didn't carry *Yr22* (in Lee). As for the differences and relations between the three resistance genes in Early Premium with *YrDa2* (Daws) and *YrSte* (Stephens) needs more studies in the future. For the lines with known gene(s) is limited, this study fail to determine differences and relations of the resistance genes in Early Premium to CYR18 and 2E16. Consequently, more detailed studies should be continued on resistance genes in Early Premium. Therefore, the preliminary results show that Early Premium contains at least three pair resistance genes to yellow rust, temporarily named *YrEP1*, *YrEP2* and *YrEP3*.

Acknowledgement: This research was supported by the National Basic Research and Development Program (2011CB100403), the R & D Special Fund for Public Welfare Industry (agriculture) (200903035) and the National Natural Science Fund for Young Scholars of China (30700521). The authors are grateful to the various cooperators for supplying rust isolates and cultivars.

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(Received 27 January 2011; Accepted 28 April 2011)