

Heritability and number of genes controlling slow-mildewing resistance in Chinese wheat cultivar Lumai 21

Ni Xiaowen¹, Chen Xinmin¹, XIA Xianchun¹, He Zhonghu¹, and Morten Lillemo²

¹Institute of Crop Science, Chinese Academy of Agricultural Sciences, Beijing 100081; ² Department of Plant and Environmental Sciences, Norwegian University of Life Sciences, 5003, Ås, Norway

Corresponding author: Chen Xinmin, e-mail: chenxm@mail.caas.net.cn

SUMMARY

The Chinese winter wheat cultivar Lumai 21 showed slow resistance to powdery mildew. To estimate the number of genes and its heritability, 200 lines of F_{2,3} and F_{2,4} populations derived from the cross between Lumai 21 and Jingshuang 16 (susceptible) and their parents were planted at Beijing and Anyang, Henan for disease evaluation in 2005-06 and 2006-07 cropping seasons. The resistance was analyzed based on both quantitative and qualitative genetic models. At least 3-4 resistance genes were detected in the 2 populations. The broad-sense heritability of resistance was 0.53–0.78. Transgressive segregation indicated that Jingshuang 16 might hold 1 minor gene for resistance, and Lumai 21 contained at least 2-3 genes, accordingly.

INTRODUCTION

Powdery mildew, caused by *Blumeria graminis* f.sp.*tritici*, is one of the most important wheat diseases worldwide. It has caused tremendous yield loss in the Chinese wheat production since 1990. Utilization of resistant cultivars is an economical and environmentally safe way to control the disease. However, the durability of resistance is very short in the varieties with race-specific resistance gene, since it can easily be overcome by changes of pathogen population [1]. Slow mildewing, also called adult plant resistance or partial resistance, has showed durable resistance [2]. Lumai 21, bred by the Institute of Yantai Agricultural Science was released in 1991 and reached a maximum acreage of 107 million ha in 1997. This variety had a total growing area of more than 400 million ha up to 2006. It showed good resistance to powdery mildew for 17 years and was identified with slow mildewing [3]. The objective of this study was to determine the inheritance of resistance to powdery mildew in Lumai 21

MATERIALS AND METHODS

Parents and 200 lines of F_{2,3} and F_{2,4} from the cross between the slow mildewing cultivar Lumai 21 and susceptible variety Jingshuang 16 (also as susceptible check) were planted in Beijing and Anyang of Henan to evaluate the disease in 2005-06 and 2006-07 cropping seasons. Artificial inoculation with the isolate E20 of *B.graminis* f. sp. *tritici* was performed in Beijing at the two cropping seasons and relied on natural epidemic of disease in Anyang. One hundred and ninety five lines were planted in Anyang due to shortage of seeds in the

first cropping season. The field trials were conducted in a randomized complete block design with two to three replications at each location. Fifty seeds of each parent and of each F_{2,3} and F_{2,4} lines were planted in 1.5-m long rows and 30cm apart in October 2005 and 2006, respectively. To facilitate mildew increase and dissemination, spreader rows of the susceptible cv. Jingshuang 16 were planted every 10 rows and also surrounding the whole experiment. Before disease assessment, 10 randomly selected plants from each row were tagged. Powdery mildew severity was scored after flowering on penultimate leaves of tagged plants, based on the actual percentage of leaf area covered by powdery mildew, and then every 7 days until leaf senescence. The last score constituted the maximum disease severity (MDS). The average severity of 10 plants was the severity value of each line. The area under the disease progress curve (AUDPC) was calculated according to Bjarko and Line [4]. The mean value of MDS and AUDPC of 10 plants from each line was used for the final analysis. Number of genes controlling slow resistance to powdery mildew was estimated by both qualitative and quantitative methods. For qualitative analyses, the F_{2,3} and F_{2,4} lines were grouped into resistant and intermediate-susceptible types. A line was considered resistant when it had a similar disease score as the resistant parent Lumai 21± one standard deviation. The goodness of fit to the expected segregation ratios for three, four, and five independent genes was tested by chi-square analysis. A quantitative estimate for the minimum number of genes controlling slow-mildewing resistance was made according to Wright's method [5]. Analyses of variance (ANOVA) were conducted to estimate the σ_p^2 , σ_g^2 and σ_E^2 of F_{2,3} and F_{2,4} lines using SAS 8.1 (SAS Institute, Cary, NC), where $\sigma_g^2 = (\sigma_L^2 - \sigma_E^2) / n$, $\sigma_p^2 = \sigma_g^2 + \sigma_E^2$, and broad-sense heritability (h^2) was calculated by the formula $h^2 = \sigma_g^2 / \sigma_p^2$. Where σ_p^2 = phenotypic variance, σ_g^2 = genetic variance, σ_L^2 = variance of F_{2,3} and F_{2,4} lines, σ_E^2 = variance of environment, and n = number of replications.

RESULTS AND DISCUSSION

Powdery mildew development was good at both locations and years, and the severity of susceptible parent Jingshuang 16 were over 70% at the last score. Distribution of frequencies for MDS and AUDPC of F_{2,3} and F_{2,4} lines showed a continuous distribution close to normality at both locations and both years (data not shown). Although Anyang location was under natural disease condition and Beijing was artificially inoculated,

both locations had similar tendency of disease development. Several lines exhibited higher levels of resistance than Lumai 21 in both locations and both years (Table 1). Transgressive segregation indicated that the susceptible parent Jingshuang 16 also provided some resistance genes in F_{2:3} and F_{2:4} populations. Broad-sense heritability of MDS and AUDPC were between 0.51 and 0.78 (Table 1), and they were higher in Beijing under

artificial inoculation than in Anyang under natural infection. However, since a highly significant correlation was found between AUDPC and MDS (R=0.91-0.93 P<0.001) and because the investigation of AUDPC is time-consuming, it is more practical to use MDS for powdery mildew screening with a single scoring at an appropriate time.

Table 1 Score and heritability of powdery mildew in parents and generations F_{2:3} and F_{2:4}

Gene-ration	Location	Disease index ¹	Parent ²			No. of lines	Offspring		Broad-sense Heritability
			P1	P2	Mean		Range	Mean	
F _{2:3}	Beijing	MDS	6.0	85.0	45.5	200	1.7–90.0	53.6	0.68
F _{2:3}	Beijing	AUDPC	42.5	950.5	496.3	200	31.1–1115.5	625.9	0.78
F _{2:3}	Anyang	MDS	3.0	78.0	40.5	195	1.0–85.0	48.2	0.53
F _{2:3}	Anyang	AUDPC	10.5	385.0	194.3	195	8.5–455.0	261.3	0.61
F _{2:4}	Beijing	MDS	1.5	80.0	40.8	200	0.4–86.0	42.0	0.72
F _{2:4}	Anyang	MDS	1.0	71.0	37.5	200	0.3–76.3	39.0	0.51

¹MDS: maximum disease severity; AUDPC: area under the disease progress curve. ²P1: female parent Lumai 21; P2: male parent Jingshuang 16.

Table 2 Estimated gene numbers based on the segregation of F_{2:3} and F_{2:4} lines in two cropping seasons across two environments

Gene-ration	Location	Disease Index	No. of Genes ¹	No. of lines		3 genes		4 genes		5 genes	
				R	S+I	χ ²	P value	χ ²	P value	χ ²	P value
F _{2:3}	Beijing	MDS	4.21	5	195	2.59	0.05-0.1	0.06	0.75-0.9	6.91	0.005-0.01
F _{2:3}	Beijing	AUDPC	4.35	5	195	2.59	0.05-0.1	0.06	0.75-0.9	6.91	0.005-0.01
F _{2:3}	Anyang	MDS	3.78	5	190	2.88	0.05-0.1	0.32	0.25-0.5	15.2	<0.005
F _{2:3}	Anyang	AUDPC	4.03	6	189	1.48	0.1-0.25	0.67	0.25-0.5	12.6	<0.005
F _{2:4}	Beijing	MDS	4.13	6	194	6.89	0.005-0.01	0.11	0.5-0.75	1.68	0.1-0.25
F _{2:4}	Anyang	MDS	3.49	6	194	6.89	0.005-0.01	0.11	0.5-0.75	1.68	0.1-0.25

¹ Number of genes estimated based on Wright's formula. In F_{2:3}, the expected segregation values for chi-square test are 0.053:0.947, 0.020:0.980, and 0.007:0.993 for 3, 4, and 5 independent genes, respectively; while in F_{2:4}, they are 0.084:0.916, 0.037:0.963, and 0.016:0.984 for 3, 4 and 5 independent genes, respectively.

The result of gene number estimates, both quantitative and qualitative, are given in Table 2. The N values were from 3.49 to 4.35 in two sites and two generations based on Wright's method, indicating three to four genes conferred powdery mildew resistance in the two populations. According to chi-square method, the number of genes is from three to four in F_{2:3} populations and more possibility for three, while they are four to five in F_{2:4} populations and more possible for four. Based on the two kinds of genetic analyses, it appears most likely that at least three to four genes segregated for powdery mildew resistance in the two populations. Since Transgressive segregation was observed, the susceptible parent Jingshuang 16 might have also provided resistance genes in F_{2:3} and F_{2:4} populations, and if we assume that it contributed one minor resistance gene, Lumai 21 would have contributed at least two to three genes for powdery mildew resistance in the two

populations. The gene number estimates for slow resistance to powdery mildew reported in the present study are in good agreement with other genetic studies, in which two to three genes with moderate to high heritability, control slow-mildewing resistance [6, 7, 8]. Lumai 21 is a line with high yield and good agronomical characteristics that could be a suitable parent in wheat breeding.

ACKNOWLEDGEMENTS

This work was supported by research program (2006BAD01A02-1) from the Ministry of Agriculture.

REFERENCES

1. Heun M. 1987. Virulence frequencies influenced by host resistance in the host-pathogen system wheat powdery mildew. *J. Phytopathology*, 118: 363-366.
2. McDonald B A, Linde C. The population genetics of plant pathogens and breeding strategies for durable resistance. *Euphytica*, 2002, 124: 163-180
3. Wang Z L, Li L H, He Z H, Duan X Y, Zhou Y L, Chen X M, Lillemo M, Singh R P, Wang H, Xia, X C. Seedling and adult plant resistance to powder mildew in Chinese bread wheat cultivars and lines. *Plant Dis*, 2005, 89: 457-463
4. Bjarko M E, Line R F. Heritability and number of genes controlling leaf rust resistance in four cultivars of wheat. *Phytopathology*, 1988, 78: 457-461
5. Wright S. Evolution and the genetics of populations. In: *Genetic and Biometric Foundations*, Vol. 1., University of Chicago Press, 1968. p 469
6. Das M K, Griffey C A. Heritability and number of genes governing adult-plant resistance to powdery mildew in Houser and Redcoat winter wheats. *Phytopathology*, 1994, 84: 406-409
7. Griffey C A, Das M K. Inheritance of adult plant resistant to powdery mildew in Knox 62 and Massey winter wheats. *Plant Dis*, 1996, 83: 424-428
8. Lillemo M, Skinnes H. Genetic Analysis of a partial resistance to powdery mildew in bread wheat line Saar. *Plant Dis*, 2005, 90: 225-228