

GENETIC MAPPING AND QTL ANALYSIS ASSOCIATED WITH FUSARIUM HEAD BLIGHT RESISTANCE AT DIFFERENT DEVELOPMENTAL STAGES IN WHEAT (*TRITICUM AESTIVUM* L.) USING RECOMBINANT INBRED LINES

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Abstract

In order to reveal the genetic basis of wheat FHB resistance of a recombinant inbred line (RIL) population including 160 plants derived from a cross between PI277012 and Yumai 18 was used in this study. Wheat recombinant inbred line (RIL) population was inoculated in a suspension of *Fusarium graminearum* spores and cultured indoor, during which pathogenesis was observed. Disease index and area under the disease progress curve (AUDPC) were calculated based on the data that collected on days 5, 10, and 15, in order to analyze the dynamic changes in wheat FHB resistance at different developmental stages. During the first stage, no QTL (quantitative trait locus) was found, and disease resistance index of each strain was increased steadily and uniformly. We speculated that when *Fusarium graminearum* started to expand in wheat spikelet, the wheat plant has yet to respond to the infection, resulting in a minor difference in disease resistance between the different lines. During the second stage, four QTLs were found, which were mapped to chromosomes 1A, 1D, 3B and 4D, respectively, with QTLs located between wmc777 and barc147 on chromosome 3B, which was consistent with previous results. We speculated that the large differences in resistance index in this stage were attributed to the reaction of the FHB-resistant QTLs, leading to differences among different wheat strains. During the third stage, three QTLs were located on chromosomes 1A, 5A and 7A, respectively. Compared with the traditional field cultured inoculation, field topsoil inoculation or natural breeding, the indoor experiment was highly controlled, facilitating annual comparison independent of variations in climate.

Key words: Wheat, *Fusarium* head blight, *Fusarium graminearum*, QTL.

Introduction

Wheat *Fusarium* head blight (FHB) is primarily induced by *Fusarium graminearum*. It affects food yield and safety, reducing seed germination and grain weight (Agostinelli *et al.*, 2012; Han *et al.*, 2016). It also generates toxins, which are harmful to humans and animals (He *et al.*, 2013). Wheat FHB is mainly found in humid and semi-humid areas, comprising middle-to-lower reaches of Yangtze River, Jianghuai and Huanghuai wheat areas in China. In 2012, wheat FHB was reported epidemic in a large area of Shandong and other northern regions of China (Zeng *et al.*, 2013; Zhang *et al.*, 2009). Although urgent measures to prevent and treat infection are needed, resistant wheat species are scarce and inadequate. Currently, Sumai 3 and its derivatives as well as Wangshuibai that has no genetic relationship with Sumai 3, are considered highly FHB-resistant, and are widely used in wheat breeding (Ma *et al.*, 2005; Bai *et al.*, 1996; Zhou *et al.*, 2004a). In addition, Japan's Shinchunaga, Brazil's Frantana and other species have been effectively used in wheat breeding with FHB resistance worldwide (Steiner *et al.*, 2017). However, the resistant varieties exhibit poor agronomic traits. Therefore, wheat species manifesting high resistance and high yield are unavailable (Zhuang *et al.*, 2003; Lu & Yao, 2001; Liu, 2001).

Wheat FHB is a quantitative trait controlled by multiple genes, with several types of resistance. Mesterházy (1995) classified wheat FHB resistance into five categories: type I refers to initial pathogen infection triggering FHB resistance, type II refers to expansion of FHB resistance, type III involves induction of grain resistance, type IV denotes tolerance to FHB, and type V represents deoxynivalenol (DON) resistance. Types I, II and V are extensively studied. In this study, we investigated type II resistance.

Currently, the widely recognized wheat resistance loci include Fhb1 (QJhs.ndsu-3BS) located on chromosome 3B, and Fhb2 on chromosome 6B of Sumai 3 (Waldron *et al.*, 1999; Cuthbert *et al.*, 2007). Cuthbert (2006) *et al.*, localized the Fhb1 between SSR-labeled Xgwm533 and Xgwm493, while Fhb2 was located between Xgwm133 and Xgwm644. The studies (Cuthbert *et al.*, 2007) also investigated wheat FHB resistance in BW278/AC recombinant inbred line (RIL) populations, and mapped the resistant QTL on chromosome 6BS, labeled Xgwm133 and Xgwm644, and designated as Fhb2. Liu *et al.*, (2009) published more than 50 studies investigating QTLs of FHB resistance and mapped resistant QTLs covering the full-length wheat genome. Most of the QTLs were associated with type II resistance, while QTLs on 3A, 5AS, 7A, 1B, 3BS, 4B, 6BS and 2D were located on two or more chromosomal regions. Buerstmayr (2009) also reported that the current resistance genes against FHB almost covered the full-length genome of wheat using diverse detection methods and experimental species. Therefore, new perspectives and ideas may be required to map FHB resistance genes. In this experiment, we analyzed the dynamic changes in QTLs associated with disease resistance based on disease index at different stages after inoculation and comprehensive FHB resistance based on AUDPC calculated with disease index.

In addition, traditional field inoculation is limited by contaminated environment and difficult experimental conditions. Therefore, we partially modified the traditional method to conduct the indoor experiment under controlled conditions. This experiment was repeated after 2 years, during which wheat samples were acquired during the flowering period, inoculated, cultured and observed in the laboratory.

Materials and Methods

Plant materials: The recombinant inbred lines (RIL) with 160 lines derived from PI277012 and Yumai 18 was used in this study. PI277012 has a pedigree of “Extremo Sur/Argelino// T. timopheevi” is a excellent source for FHB resistance (Chu *et al.*, 2011). Yumai 18 has a pedigree of “Zhengzhou761/Yanshi4” with thin and short root system, high tiller number and good grain quality which has been grown in Northern China for many years. The F1 generation was derived via hybridization of male parent PI277012 and female parent Yumai 18, and F2 generation was obtained by inbreeding F1 generation. The subsequent generations (until F9) were derived as single seed descent (SSD), resulting in 160 offspring strains.

The *Fusarium graminearum* strains were isolated from diseased spikes of FHB wheat in Shandong Province.

Identification of FHB resistance: Wheat RIL populations and the parents were planted in Jiaozhou Experimental Station of Qingdao Agricultural University during 2015 and 2016. Five plants were sampled to detect FHB resistance for the RILs and the parents. Spore suspension liquid was used for inoculation by using single flower instillation in the laboratory, and incubated to observe disease incidence.

Spore suspension: *Fusarium graminearum* strains stored in 4°C sterile until transferred to PDA (Potato Dextrose Agar) medium for activation. After culturing for 8 days, the strains were cut into 3×3mm cakes. Seven to eight fragments were selected and transferred into 100mL green bean culture fluid. Five bottles of strains were inoculated, placed into a 150rpm shaker and cultured at 25°C. Five days later, the green bean culture fluid was filtered to obtain conidial suspension liquid, which was preserved in a 4°C refrigerator after adjusting its concentration to 5×10⁵ cells/mL using sterile water.

Inoculation using single flower instillation: Blossoming wheat spikes ready for pollination were selected and transplanted to a test tube rack. The rack was placed in a dish containing water. Subsequently, 10μL of conidial suspension was injected into a small flower in the spikelet located in the middle of the wheat spike using a micro syringe. The inoculated spikelet was sprayed using a watering can, enveloped with a lock bag, and transferred into a 25°C incubator. Five spikes of each strain were inoculated, and the lock bags were removed after 24 h.

Statistical method for disease conditions: On days 5, 10 and 15 after inoculation (denote first, second and third stages, respectively), the incidence and expansion of wheat FHB were observed and recorded. Disease incidence was graded based on a 0–9 classification method.

Based on disease classification, the disease index of each strain was determined by:

$$\text{Disease index (\%)} = \sum ax/nT \times 100$$

a refers to disease classification, **X** denotes the number of diseased spikes, **n** represents the highest disease classification and **T** represents the number of samples.

The experiment was conducted over two consecutive years, and the average disease index was adopted. The area under the disease progress curve (AUDPC) of each species was calculated using three values of disease index. The AUDPC and disease index were selected to compare the disease resistance between different strains. Ultimately, the correlation coefficients were obtained using the software of EXCEL.

Construction of genetic linkage map: DNA was extracted using DNA extraction kit (Plant Genomic DNA Extraction Kit, Tiangen Biotech (Beijing) Co., Ltd.).

A total of 1222 SSR (Simple Sequence Repeats) primers were used. The primer sequences were acquired from the network and synthesized at Sangon Biotech Co., Ltd. The reagents were manufactured by Tiangen Biotech (Beijing) Co., Ltd. and Sangon Biotech Co., Ltd.

The total PCR reaction volume of 25μL comprised 12 μL ddH₂O, 2μL Primer Pair, 10μL PCR Master Mix and 1μL DNA (20ng/L). The PCR reaction conditions were: 94°C for 5min, 94°C for 1min, 52°C for 45s and 72°C for 1min, 35 cycles, and 94°C for 1min, 72°C for 10min and 4°C. Following addition of 2μL loading buffer to 4μL of the amplification products, the denaturation step was carried out at 95°C for 5min and immediately transferred into an ice-water mixture for subsequent experiments.

The amplification products were analyzed in a 6% polyacrylamide gel, followed by silver staining.

Subsequently, QTL IciMapping 4.0 software was used to construct the genetic linkage map.

QTL analysis: QTL IciMapping 4.0 software was used to analyze resistance indices during the three stages after inoculating of the RIL populations. The final AUDPC values were analyzed to obtain the additive QTLs and epistatic QTLs for wheat FHB resistance, using a 1.0cM scan frequency. Figures were drawn using ICIM-ADD and ICIM-EPI methods. The LOD threshold was 2.5, resulting in additive QTL (A) and additive × additive epistatic QTL (AA). A positive effect size was defined by the enhancing allele of the QTL derived from the male parent PI277012. A negative effect size suggested that the enhancing allele at the QTL was derived from the female parent Yumai 18.

Results and Analysis

Dynamic expansion of FHB resistance in wheat populations: Wheat populations were divided into 10 groups according to intervals of disease index variation at each stage after inoculation. Approximately, 50 differences in variation amplitude were observed between the lines during the first and third stages. The differences between lines at the second stage were the maximum (80) and were within the limit of variation amplitude of the disease index (0–100). A smaller increase in disease index

suggested higher resistance of wheat. During the first stage, the incidence was more concentrated and uniform, with 90% of the lines showing a disease index variation ranging from 10 to 40. Thus, the resistance to expansion was not dose-dependent among the lines. During the second stage, differences were observed among the lines, with a smaller increase suggesting adequate resistance, while a larger increase indicated weak resistance. There were 48 lines showing a disease index variation ranging from 50 to 60, accounting for 33%, and only two lines exhibited a disease index variation ranging from 80 to 90. Lines with a disease index variation in other ranges were distributed more evenly, with each group comprising nearly 10 to 30 lines. During the third stage, most of the lines failed to show substantial variation in disease index, while 89% of the lines showed a variation less than 30 (Table 1 and Fig. 1).

Resistance of each line in the wheat RIL populations was determined by calculating the AUDPC value. A low value suggested better resistance. Populations were divided into eight groups with an AUDPC interval of 10, which represented a right-skewed distribution (Fig. 2). There were 45 lines with an AUDPC value ranging from 155 to 165, while six lines in the group exhibited optimal performance, i.e., with a minimum AUDPC value.

Increases in the disease index and AUDPC values in the top 15 and bottom 15 at each stage were compared (Table 2). The results showed that the two lines in the top 15 in ascending order (of increasing disease resistance) were also in the top 15 disease indices in ascending order during the first stage. Four lines were also in the top 15 of increased disease index in ascending order during the second stage. Three lines ranked 1, 3 and 4 in the AUDPC list. However, the top 15 AUDPC values in ascending order did not represent the same lines as the top 15 of increased disease index during the third stage.

Meanwhile, the top 15 AUDPC values in descending order (of weak disease resistance) included nine lines, which were also in the top 15 disease indices in the descending order during the first stage. However, they did not include lines, which were also in the list of disease indices in the descending order in the other two stages.

In the second stage, wheat lines with strong spread resistance showed maximum correlation between variation in disease resistance index and the lines with a small AUDPC value. Lines with a weak spread resistance do not exhibit excellent performance during the second stage. The persistent weak expansion during the first stage resulted in a maximum correlation between wheat with poor resistance and disease index variation at the first stage.

Wheat genetic map: Among the 1222 SSR primers, 308 polymorphic primers in the parents were used to construct the wheat genetic map covering all the 21 wheat chromosomes, spanning the full length of 2592.4cM. The average distance between the molecular markers was 8.42cM. The markers in each chromosome ranged from 10 to 24, with an average of 15 markers each. Thus, this genetic map met the criteria for QTL mapping.

Dynamic QTL analysis of wheat FHB resistance:

Based on the disease indices and the calculated AUDPC values during the three stages after inoculation, nine additive effects and nine pairs of epistatic loci were located. The nine additive loci were mapped to chromosomes 1A, 1D, 2B, 3B, 4D, 5A, 5D and 7A, of which two loci were mapped to 1A, and one locus to each of the other chromosomes (Fig. 3).

Dynamic analysis of QTLs during the First Stage: No additive or epistatic loci were detected.

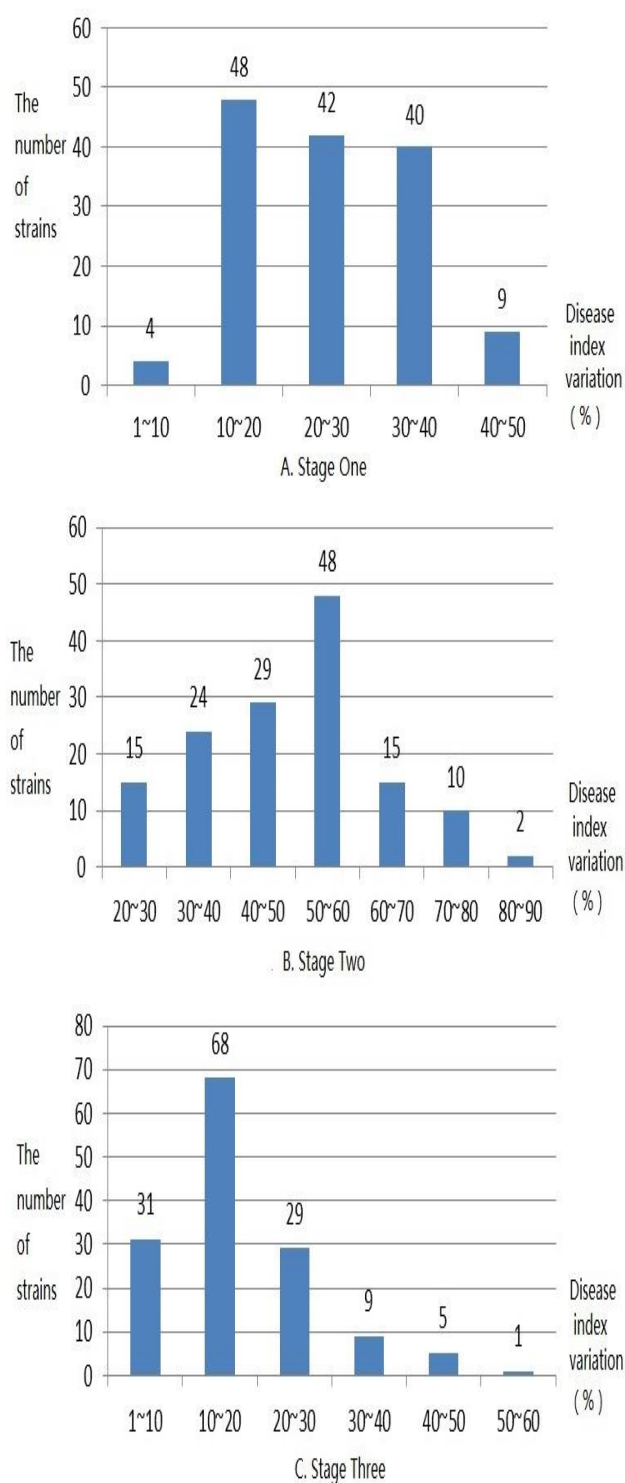


Fig. 1. Disease index variation at different stages.

Table 1. Disease index variation at different stages.

	Variation in average disease index (%)	Variation range of disease index (%)	Kurtosis	Skewness	Standard deviation
1 st stage	26.43	4.44~48.88	-2.97	-0.54	20.46
2 nd stage	49.43	20~48.88	1.13	0.95	15.02
3 rd stage	17.81	2.22~51.11	1.37	1.24	24.99

Table 2. Disease index increase and AUDPC ranking.

	Top 15 in ascending order (No.)	Top 15 in descending order (No.)
Order of variation during the first stage	21, 22, 14, 18, 84, 15, 42, 50, 130, 55, 36, 71, 90, 126, 127	109, 9, 74, 16, 88, 29, 54, 4, 139, 128, 38, 34, 1, 155, 135, 99, 85, 49
Order of variation during the second stage	33, 35, 38, 93, 34, 56, 88, 80, 43, 77, 85, 96, 61, 29, 74	18, 15, 14, 71, 22, 21, 107, 126, 84, 159, 68, 67, 150, 92, 36
Order of variation during the third stage	3, 146, 4, 9, 15, 131, 1, 7, 2, 8, 11, 74, 118, 54, 59, 109, 128	56, 43, 122, 96, 77, 48, 35, 46, 117, 73, 116, 110, 94, 80, 30
Order of AUDPC value	56, 5, 77, 96, 123, 43, 122, 125, 127, 117, 48, 24, 83, 55, 75, 93	54, 128, 16, 146, 155, 9, 49, 139, 158, 109, 97, 102, 99, 156, 131, 1

Table 3. Additive effects of QTLs for FHB resistance based on disease index at the second stage.

QTL	Position (cM)	Left marker	Right marker	LOD value	Additive effect
qFHB5-1A	11	wmc611	barc213	3.36	-21.07
qFHB5-3B	23	wmc777	barc147	4.60	-13.14
qFHB5-1D	23	cf19	wmc339	3.70	-12.82
qFHB5-4D	103	gwm285	wmc622	3.21	-4.10

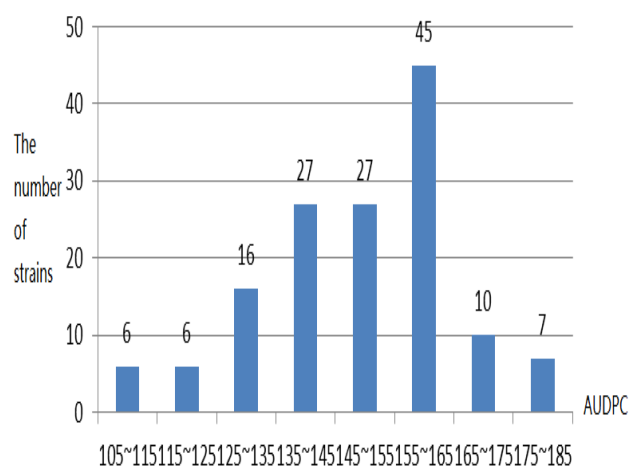


Fig. 2. 2AUDPC of wheat RIL population.

Dynamic analysis of QTLs during the second stage:

At this stage, 4 loci with additive effects and 5 pairs of loci with epistatic effects were detected (Tables 3 and 4). The qFHB5-1A, qFHB5-3B, qFHB5-1D and qFHB5-4D additive loci were, respectively, located on chromosomes 1A, 3B, 1D and 4D, with LOD values ranging from 3-5, and the additive effects were negative. Thus, all the enhancing alleles were derived from the female parent Yumai 18.

Five pairs of epistatic loci were located on chromosomes 1B and 2B, 1B and 4D, 1B and 5B, 2B and 5D, 4D and 5D, respectively. The LOD values ranged from 5 to 8, with negative epistatic effects of qFHB5-1B.1 and qFHB5-2B.2, qFHB5-1B.2 and qFHB5-4D.2, qFHB5-4D.1 and qFHB5-5D, suggesting that the parental

epistatic effect was greater than the recombinant epistatic effect. Meanwhile, the epistatic effects of qFHB5-1B.3 and qFHB5-2B.1 were positive, which suggested that the recombinant epistatic effect was greater than the parental epistatic effect.

Dynamic analysis of QTLs at the third stage: A total of three additive loci and 4 pairs of epistatic loci were detected (Tables 5 and 6). The qFHB7-1A, qFHB7-5A and qFHB7-7A were located on chromosomes 1A, 5A and 7A, respectively. They showed a negative additive effect and a LOD value range of 4-10, which indicated that the enhancing alleles were derived from the female parent Yumai 18.

Four pairs of epistatic loci were located on chromosomes 2A and 1B, 1B and 6B, 1B and 4D, 3D and 5D, respectively. The LOD values were about 5 and 6, and the epistatic effects of qFHB7-1B and qFHB7-6B were negative, which suggested that their recombinant epistatic effect was greater than their parental epistatic effect. By contrast, the parental epistatic effect of qFHB7-2A and qFHB7-1B.2, qFHB7-1B.1 and qFHB7-4D, as well as qFHB7-3D and qFHB7-5D, was greater than their recombinant epistatic effect.

QTLs with comprehensive resistance: Based on the analysis of AUDPC values, two additive QTLs were located, but no epistatic locus was detected (Table 7). The two additive loci were mapped to chromosomes 2B and 5D, with LOD values of 2.59 and 3.15, respectively, of which qFHB5-2B was derived from the female parent Yumai 18 and qFHB5-5D was derived from the male parent PI277012.

Table 4. Epistatic QTLs for FHB resistance based on disease index at the second stage.

QTL1	Position 1 (cM)	Left marker	Right marker	QTL2	Position 2 (cM)	Left marker	Right marker	LOD value	Additive effect
qFHB5-1B.1	70	gwm344	gwm498	qFHB5-2B.2	215	barc159	cfa2201	5.16	-8.37
qFHB5-1B.2	90	gwm344	gwm498	qFHB5-4D.2	125	gwm285	wmc622	5.07	-6.65
qFHB5-1B.3	95	gwm344	gwm498	qFHB5-5B	75	gwm234	gwm335	5.95	9.66
qFHB5-2B.1	75	barc55	wmc245	qFHB5-5D	50	gwm292	cf29	7.36	12.06
qFHB5-4D.1	110	gwm285	wmc622	qFHB5-5D	35	gdm63	gwm292	5.89	-9.21

Table 6. Epistatic QTLs for FHB resistance based on disease index at the third stage.

QTL1	Position1 (cM)	Left marker1	Right marker1	QTL2	Position2 (cM)	Left marker2	Right marker2	LOD value	Additive effect
qFHB7-2A	140	wmc170	Cfa2164	qFHB7-1B.2	70	gwm344	gwm498	6.13	3.31
qFHB7-1B.1	20	gwm312	wmc120	qFHB7-6B	20	wmc487	bj242562	5.08	-3.03
qFHB7-1B.1	20	gwm312	wmc120	qFHB7-4D	135	gwm285	wmc622	5.29	3.06
qFHB7-3D	90	gwm497	gwm3	qFHB7-5D	15	cfa2141	gdm63	5.02	3.65

Table 5. Additive QTLs for FHB resistance based on disease index at the third stage.

QTL	Position (cM)	Left marker	Right marker	LOD value	Additive effect
qFHB7-1A	67	barc120	barc158	4.10	-5.43
qFHB7-5A	12	barc141	gwm617	10.49	-6.61
qFHB7-7A	9	barc180	barc70	6.28	-6.80

Table 7 Additive QTLs for comprehensive FHB resistance based on AUDPC.

QTL	Position (cM)	Left marker	Right marker	LOD value	Additive effect
qFHBA-2B	130	bem99	wmc317	2.59	-15.21
qFHBA-5D	31	gdm63	gwm292	3.16	17.21

Discussion

Disease resistance and QTLs at three stages were comprehensively analyzed. No QTL was found during the first stage, and increase in disease resistance of each strain was more focused and uniform. *Fusarium graminearum* may expand in the wheat spikelet leading to insignificant differences in disease resistance among the various strains. During the second stage, four additive QTLs were mapped to chromosomes 1A, 1D, 3B and 4D. Zhang (2012) utilized FHB-resistant winter wheat Heyneto map a resistant QTL on 4DL, which accounted for 23.4% of the phenotypic variation. In addition, to Fhb1 mapped on chromosome 3B, QTLs have been mapped to chromosome 3B in other studies (Gao *et al.*, 2005; Yang *et al.*, 2005; Cuthbert *et al.*, 2006). For example, Zhou (2003) reported that the major QTL of Wangshuibai was located between barc147 and gwm493, within a region of 11.5cM, which accounted for a mere 15.1% of the FHB resistance. In this experiment, we also detected QTLs between wmc777 and barc147 during the second stage, with a spacing of 27.8cM, where the LOD value of the QTL was 4.60 and the additive effect was -13.14, which suggested origin in the female parent Yumai 18. Multiple loci were associated with FHB resistance on chromosome 3B. A comprehensive analysis of the resistance index increase during the second stage revealed that the QTLs controlling the FHB resistance accounted

for large differences in resistance indices resulting in variation among wheat strains. During the third stage, three QTLs were located on chromosomes 1A, 5A and 7A. Liu (2007) utilized resistant winter Ernie to successfully locate four expanding QTLs to explain 43.3% of phenotypic variation, including locus on the 5A. Jayatilake (2011) used the disomic substitution line on chromosome 7A of spring-Sumai 3 to successfully map a novel resistance QTL Fhb7AC near the centromere of chromosome 7A, to explain 22% of the phenotypic variation in spread resistance. Zhou (2004b) analyzed QTLs associated with FHB resistance in Wangshuibai/Alondra recombinant inbred line, and detected QTLs for FHB resistance on chromosomes 4A, 5A, 7A and 4D.

We compared AUDPC values with resistance indices during the second stage, and selected four strains (No. 56, 77, 93 and 96) with FHB resistance in wheat RIL populations. The AUDPC values and resistance indices at the second stage of these 4 strains ranked in the top 15 in the descending order, suggesting to study QTLs for FHB resistance.

Compared with conventional outdoor inoculation, topsoil inoculation or natural breeding indoor single flower instillation is associated with many advantages. First, indoor inoculation enables control of environmental conditions, for accurate annual comparisons. Second, outdoor experiment increases the risk of land pollution, which adversely affects experimental field and the surrounding food farming. Therefore, indoor experiment is a safe and suitable method. Finally, indoor inoculation is easy to operate and compare, as well as convenient and accurate. Although the indoor single flower instillation is labor-intensive compared with topsoil inoculation or natural breeding, the results are highly accurate. We have optimized the technique to reduce the workload, and is currently under consideration for a patent.

Wheat FHB resistance is a QTL controlled by multiple genes, and the reaction mechanisms and patterns of QTL are still unclear. Breeding of disease-resistant varieties requires supplemental efforts. Therefore, new ideas, directions and continuous exploration are needed for breeding of FHB-resistant wheat varieties.

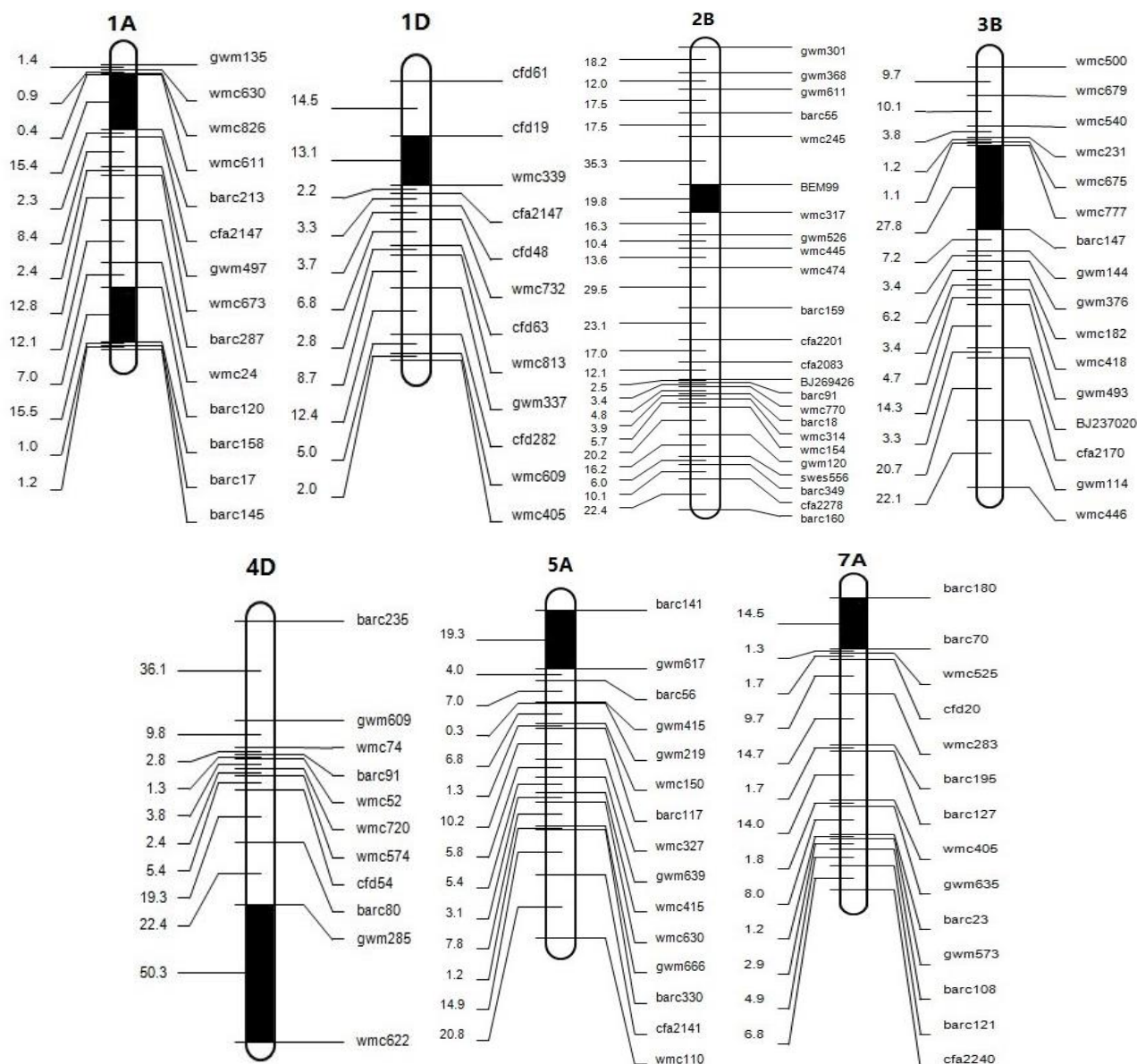


Fig. 3. QTLs for FHB resistance in wheat RIL population.

Conclusion

QTLs associated with FHB resistance in wheat are hysteretic compared with the spread of *Fusarium graminearum*. QTLs may vary in different stages. During the second stage, four QTLs were mapped to chromosomes 1A, 1D, 3B and 4D. During the third stage, three QTLs were mapped to chromosomes 1A, 5A and 7A. QTL analysis of wheat FHB resistance at different stages after infection is essential for in-depth understanding of the underlying mechanisms as well as breeding of wheat species with FHB resistance and appropriate agronomic traits.

Acknowledgements

This project was supported by the foundation of "Molecular design breeding strategy in wheat" of Shandong province (Grant No. 2016LZGC023), and the foundation of modern agricultural industrial system of Shandong province (Grant No. SDAIT-01-05).

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(Received for publication 18 August 2017)