Genome-Wide Association Study and QTL Mapping Revealed Genetic Loci for *Fusarium* Head Blight Resistance in Chinese Hexaploid Wheat

Chaonan Shi

Henan Agricultural University

Junyou Geng

Henan Agricultural University

Yan Ren

Henan Agricultural University

Yueting Zheng

Henan Agricultural University

Chunyi Liu

Henan Agricultural University

Huiting Hu

Henan Agricultural University

Xialei Lv

Henan Agricultural University

He Pei

Henan Agricultural University

Feng Chen (✉ chf0088@163.com)

Henan Agricultural University <https://orcid.org/0000-0002-7327-2969>

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Abstract

Fusarium Head Blight (FHB) greatly affects wheat yield worldwide and could also downgrade quality. In this study, a total of 406 wheat cultivars were investigated for FHB disease index (DI). Results indicated that the FHB DI of 95% of the tested cultivars showed more than scale 2. Genome-wide association study (GWAS) identified 321 SNPs significantly related to FHB resistance on all chromosomes with 2.71–13.32% phenotypic variance explained (PVE). An important genetic locus *gFHB-5A* (329,828,930 to 595,372,995 bp) was identified to modulate FHB resistance in two panels, and the incidence of resistant alleles at the *gFHB-5A1a* locus were increasing with time in the tested cultivars. Two loci of *gFHB-2B* (9,740,162 to 68,200,954 bp) with PVE values of 2.73–3.57% and *gFHB-7A* (515,126,041 to 537,557,064 bp) with PVE values of 2.71–7.04% were identified to be significantly associated with FHB resistance. Linkage analysis in a bi-parental population identified the QTL *FHB.hau-4D* flanked by markers *wPt-8836* and *wPt-4572* with PVE value of 8.9%. This study identified new genetic loci to control FHB resistance and provided useful information of marker-assist selection in wheat FHB resistance breeding.

Introduction

Fusarium head blight (FHB), mainly caused by *Fusarium graminearum* (*syn. Gibberella zeae*), is an important fungal disease of wheat (*Triticum aestivum* L.) (Desjardins & Hohn, 1997; McMullen et al., 2012). Wheat infected by the pathogen showed shriveled kernels with low weight, and red episperm and endosperm. *F. graminearum* generally produce several kinds of mycotoxins, such as deoxynivalenol (DON) and other trichothecene toxins. Infected wheat kernels with excessive accumulation of DON pose a severe threat to human and animal health (Cheng et al., 2012; Nganji et al., 2004).

It is well known that breeding resistant cultivars is one of the most economical and effective methods to manage disease, therefore it is particularly important to identify and develop FHB-resistant germplasm by combination of molecular marker-assisted selection with backcross selection and multi-point identification (Bai et al., 2012; Lan et al., 2009; Lan et al., 2010a; Lan et al., 2010b; Lu et al., 2019). Previously, two Chinese early historical accessions ‘Sumai 3’ and ‘Wangshuibai’ have been identified as highly resistant to FHB (Bai et al., 1999; Chen et al., 1998). Up to date, a large number of FHB resistance QTL has been reported on all wheat chromosomes and molecular markers have been developed for most of the mapped QTL (Buerstmayr et al., 2002; Buerstmayr et al., 2003; Steiner et al., 2017). Among them, a major gene *Fhb1* in Sumai 3 was widely reported (Anderson et al., 2001; Bai et al., 1999; Buerstmayr et al., 2002; Cuthbert et al., 2006; Lv et al., 2014). With the development of molecular markers, *Fhb1* was further fine mapped to a 1.1-Mb region (Schweiger et al., 2016). Through map-based cloning, a pore-forming toxin-like (PFT) gene encoding a chimeric lectin was regarded as *Fhb1* to modulate FHB resistance (Rawat et al., 2016). However, two independent studies showed that *TaHRC* encoding a putative histidine-rich calcium-binding protein was the key gene within the interval of *Fhb1* locus to regulate FHB resistance (Li et al., 2019; Su et al., 2019). Recently, a *Fhb7* gene from wheatgrass encoding GST protein showed a new solution for FHB resistance (Wang et al. 2020). It suggests that identification of new loci that confers FHB resistance is necessary in order to pyramid several loci/genes for developing FHB-resistant germplasm.

Common wheat ($2n=42$) is an allohexaploid (AABBDD) with a large genome size (16 Gb) and high sequence duplication between subgenomes as well as abundance of repetitive elements (about 85% of the genome) (IWGSC, 2014). With the rapid development of sequencing technologies in recent years, the wheat genome has been sequenced and provides a solid platform for cloning genes in wheat. The development of wheat high-density SNP arrays (Sun et al. 2020) including the Wheat 90K iSelect assay (Wang et al., 2014), 660K (Zhou et al., 2018), and 35K Wheat Breeder’s assay (Allen et al., 2017) made it feasible to rapidly excavate genetic loci underlying complex traits in the whole wheat genome by genome-wide association studies (GWAS). In hexaploid wheat, GWAS has been widely used to identify many important genetic loci for regulating kinds of traits, including black point (Liu et al., 2017; Lv et al. 2020), herbicide resistance (Shi et al. 2020), *Fusarium* crown rot (Yang et al., 2019), and quality traits (Chen et al., 2019), etc.

In the present study, Chinese cultivars mainly from the Huanghuai valley and a bi-parental population were used to identify genetic loci of FHB resistance by GWAS and linkage mapping. These results could provide valuable information for marker-assisted selection through pyramiding more FHB -resistance loci in wheat breeding programs.

Materials And Methods

Plant materials

A total of 406 Chinese wheat accessions were divided into two panels in this study. Panel I contained 243 wheat cultivars or advanced lines from the Huanghuai valley, the most important and largest wheat production area with 60 to 70% of both total harvested area and total wheat production in China, and they all were developed after 2014 as we previously described in Yang et al., (2019) (Table S1). Panel II contained 163 Chinese landraces and historical wheat cultivars (Chen et al., 2019; Sun et al., 2017) (Table S1). A bi-parental recombinant inbred line (RIL) population named UP derived from a cross between the susceptible parent UC1110 (DI=3) and the resistant parent PI610750 (DI=1) (Lowe et al., 2011) with 187 lines was used for QTL mapping. This RIL population was kindly provided by Jorge Dubcovsky from UC Davis. All the test accessions were planted in Jianyang (N27.30°, E118.2°) in 2016-2017 cropping season with two replications, and ‘Xinmai 18’ as a susceptible control was planted every 10 accessions.

Evaluation of wheat FHB index

Identification of FHB resistance was carried out at the anthesis stage, and disease symptoms were recorded on the 20th and 30th after the first spray inoculation, following the inoculation method of Steiner et al. (2004). A 1-4 scale was used to represent different disease index (DI) according to the severity of the FHB of the whole line (Feng, 2018). An average DI of 10 spikes was used for each test variety, where

1 = infected spikelets account for 1/4 of the total;

2 = infected spikelets account for 1/2 of the total;

3 = infected spikelets account for 3/4 of the total;

4 = infected spikelets account for more than 3/4 of the total or the whole ear died.

Genotyping and genome-wide association study

and Panel II were genotyped using the wheat 660K (Yang *et al.*, 2019) and 90K SNP arrays (Wang *et al.*, 2014), respectively. Quality control was performed for SNP markers with MAF > 0.05 and missing data < 20%. This pre-processing was examined by PLINK software (Purcell *et al.*, 2007).

GWAS was performed based on the mixed linear model (PCA+K) using GAPIT packages in R software (Lipka *et al.*, 2012; Lv *et al.*, 2020; the R Core Team, 2019). The corrected *P*-values of 1.0e-3 and 3.16e-3 were used as the appropriate significance threshold for and II, respectively. Haplotype analysis was performed using Haploview 4.2 software (Barrett *et al.*, 2005).

QTL mapping

The bi-parental RIL population UP was previously constructed by Lowe *et al.* (2011) and the genetic linkage map was composed of 1,494 probes (DArT, SSR, and ESTs). Based on polymorphism, 558 markers were extracted for QTL mapping. Genetic linkage map was constructed using IciMapping V4.1 (<http://www.isbreeding.net/>), and measurement of linkage algorithm and criteria were completed by nnTwoOpt and SAD (Li *et al.*, 2011), and Kosambi was used for genetic function. Phenotypic variance explained (PVE) was used to evaluate QTL effect (Ren *et al.*, 2017) based on ICIM-ADD model. Default was used for parameter designs. LOD scores for detecting significant QTL were calculated at the *P* = 0.05 level from 1,000 permutations, and the threshold of LOD score was set to 2.5.

Statistical analysis

SPSS ver. 20.0 software and least significant range (LSR) multiple comparisons were used to calculate average DI of cultivars with different haplotypes and compare their significant difference.

Results

FHB resistance distribution in Chinese wheat cultivars

Investigation of FHB in 406 wheat cultivars indicated that the mean disease index (DI) of and II was 2.47 (± 0.52) and 2.33 (± 0.59) (Fig. 1a), and more than 95% wheat cultivars showed FHB DI of more than 2 (Fig. 1b). In Panel I, 0.5%, 37%, 57% and 5.5% of the accessions showed FHB DI of 0 to 1, 1 to 2, 2 to 3, and 3 to 4, respectively. In Panel II, 4%, 45%, 45%, and 6% of them showed FHB DI of 0 to 1, 1 to 2, 2 to 3, and 3 to 4, respectively. The correlation coefficients between the two replicates in Panel I and Panel II are 0.73 and 0.68, respectively. Results showed that cultivars with FHB DI of 1 to 3 were predominant, and there were small number of accessions with good FHB resistance. For example, 8 cultivars (Shengyuan 928, Jimai 36, Jimai 32, Youmai 2, Yunong 201, Changnong 339-5-1, Dan 98-4025, and Beijing 841) showed FHB DI of 1 in multiple replications, and they could be used as FHB-resistant germplasms in wheat breeding.

Genome-wide association study of wheat FHB resistance

After filtering, a total of 395,782 SNPs in Panel I and 41,561 SNPs in Panel II were used for GWAS analysis (Fig. 2). A total of 321 significant SNPs with PVE of 2.71% to 13.32% were detected and they mainly distributed on chromosomes 2B, 5A, and 7A (Table S2).

In Panel I, 204 SNPs with PVE of 2.71% to 9.76% were significant in two replications. Among them, 55, 24, and 49 significant SNPs mainly distributed on 2B (9,740,162 to 68,200,954 bp), 5A (329,828,930 to 595,372,995 bp), and 7A (515,126,041 to 537,557,064 bp) (Fig. 2a; Fig. 3a; Table S1). In Panel II, 117 SNPs were significant in two replications with the PVE of 5.74% to 13.32%, and 36 of them mainly distributed on 5A (329,828,930 to 595,372,995 bp) (Fig. 2; Fig. 3b; Table S1). Combination of Panels I and II showed that there was an important genetic locus on 5A named as *gFHB-5A* (329,828,930 to 595,372,995 bp) for FHB resistance in two panels, and 60 significant SNPs located in this interval (Table 1; Table S1). Furthermore, *gFHB-2B* (9,740,162 to 68,200,954 bp) and *gFHB-7A* (515,126,041 to 537,557,064 bp) were also the important FHB resistance loci in Panel I since there were 55 and 49 significant SNPs in intervals of *gFHB-2B* and *gFHB-7A*, respectively (Fig. 2a; Table S1). In addition, some other FHB resistance loci on 3A (1,326,004 to 12,813,934 bp), 3D (2,597,023 to 92,375,516), 5D (426,001,273 to 544,697,840 bp), and 7B (675,149,692 to 714,012,248) were also detected in two panels.

Haplotype analysis of *gFHB-5A* in Chinese cultivars

Haplotype analysis (Fig. 4a, Table 2) in Panel I showed that 21 significant SNPs on 5A (577,926,236 to 588,752,395 bp) developed a 10.83-Mb block (*PL_Hap_5A1*) with two haplotypes (Table 2). Phenotypic comparison indicated that cultivars with *PL_Hap_5A1a* (2.37 to 2.4) showed a stronger FHB resistance than those with *PL_Hap_5A1b* (2.75 to 3.45) (Fig. 4c). In Panel II, 17 significant SNPs on 5A (331,401,559 to 422,077,781 bp) formed a 90.68-Mb block (*PII_Hap_5A1*) and 7 significant SNPs (591,155,558 to 595,372,995 bp) on 5A formed a 4.22-Mb block (*PII_Hap_5A2*) (Fig. 4b). *PII_Hap_5A1* contained two haplotypes (Table 2). Phenotypic comparison indicated that cultivars with *PII_Hap_5A1a* (2 to 2.24) showed a stronger FHB resistance than those with *PII_Hap_5A1b* (2.55 to 2.64) (Fig. 4d). *PII_Hap_5A2* contained two haplotypes, and phenotypic comparison indicated that cultivars with *PII_Hap_5A2a* (2.06 to 2.1) showed a stronger FHB resistance than those with *PII_Hap_5A2b* (2.5 to 2.52) (Fig. 4e). Therefore, cultivars with *PL_Hap_5Aa*, *PII_Hap_5A1a*, and *PII_Hap_5A2a* showed relatively stronger resistance to FHB. Pyramiding analysis indicated that cultivars with *PII_Hap_5A1a/PII_Hap_5A2a* (1.7 to 2) showed significantly stronger FHB disease resistance than those with *PII_Hap_5A1b/PII_Hap_5A2b* (2.8 to 2.9) in Panel II (Fig. 4f, Table 2). *PII_Hap_5A1a*, *PII_Hap_5A1a*, and *PII_Hap_5A2a* could be used for marker-assisted selection in FHB resistance breeding.

Resistance alleles at the *gFHB-5A* locus were becoming popular in China

All cultivars from Panel I were developed after 2014, and 67.5% of them possessed FHB-resistant allele *PL_Hap_5A1a*, indicating that it has been widely used in the Huanghuai valley (Fig. 5a). Based on released years of cultivars, Panel II could be divided into three periods, i.e. before 1980s, 1980s to 2000s, and after 2000s. Percentages of FHB-resistant allele *PL_Hap_5A1a* are 11.1%, 11.4%, and 14.8% in the cultivars of before 1980s, 1980s to 2000s, and after 2000s, respectively (Fig. 5b). Moreover, percentages of FHB-resistant *PL_Hap_5A2a* are 11.1%, 16.5%, and 22.2% in the cultivars of before 1980s, 1980s to 2000s, and after 2000s, respectively (Fig. 5b). It suggested that FHB-resistant alleles at the *gFHB-5A* locus were becoming prevalent with time in Chinese cultivars.

Linkage mapping for FHB resistance in UP population

Linkage analysis in the RIL population UP showed that a QTL (*FHB.hau-4D*) on chromosome 4D was detected for FHB resistance and it was flanked by markers *wPt-8836* and *wPt-4572* with PVE of 8.89%. Wheat lines with *FHB.hau-4D_AA* (2.37) showed a stronger FHB resistance than those with *FHB.hau-4D_BB* (2.73) (Fig. 6). Analysis of physical position in the Chinese Spring genome database indicated that *FHB.hau-4D* was mapped to be around 9431967-bp position on 4D.

Discussion

As one of most serious diseases that threaten wheat yield and quality, FHB has been widely studied for screening resistance germplasm and QTL mapping. Among all of the FHB-resistant QTL detected in wheat and its relative, *Fhb1-Fhb7* have formally been designated as the important genetic loci or genes. Of all 7 loci, both of *Fhb1* on 3BS and *Fhb2* on 6B were derived from 'Sumai 3' (Cuthbert et al., 2006; Cuthbert et al., 2007), and *Fhb4* is from 'Wangshuibai' (Xue et al., 2010), and *Fhb5* on 5A is from 'Wangshuibai' and 'Sumai 3' (Xue et al., 2011), and both *Fhb6* and *Fhb7* are from wild relatives of wheat (i.e. *Leymus racemosus*, *Elymus tsukushiensis* and *Thinopyrum ponticum*) (Cainong et al., 2015; Guo et al., 2015; Qi et al., 2008).

Fhb1 locus on 3BS as the most stable and largest effect QTL was first reported in Sumai3 and its derivative Ning7840 (Bai et al., 1999; Rawat et al., 2016; Waldron et al., 1999). *Fhb1* gene was first reported to be a pore-forming toxin-like (PFT) gene encoding a chimeric lectin with two agglutinin domains and an ETX/MTX2 toxin domain (Rawat et al., 2016). But more recently, two different groups simultaneously reported a new gene *TaHRC* as *Fhb1*, and it encodes a putative histidine-rich calcium-binding protein. *TaHRC* conferred FHB susceptibility and the deletion spanning its start codon can result in FHB resistance (Li et al., 2019; Su et al., 2019). Identification of candidate genes at other loci are still ongoing. Here, 10 significant SNPs were identified on 3B by GWAS, and *Fhb1* gene and *Qfhb.hwwg-3BSc* (Somers et al., 2003) were in the interval we identified in the present study.

The genetic locus on 5A (*gFHB-5A*) in the present study was identified in both Panels I and II. This locus contained 60 significant SNPs in the region of 329,828,930 to 595,372,995 bp on 5A. Compared to previous studies, *Fhb5* on 5A was the important resistance locus from Wangshuibai and Sumai 3 (Brar et al., 2019; Lin et al., 2006). Steiner et al. (2019) identified two QTL on 5A in Sumai3. The major QTL *Qfhs.ifa-5Ac* was mapped to be close to centromere and the smaller QTL *Qfhs.ifa-5AS* (70.7-195.3 Mb) was mapped to the distal of chromosome 5AS with distance of 140 Mb from *Qfhs.ifa-5Ac* (245.9-290 Mb). However, Zhang et al. (2012) identified that *Qfhb.hwwg-5A* was flanked with *Xbarc141* and *Xgwm186* (468.9-471.7 Mb) in Baishanyuehuang, and *Qfhb.hwwg-5A* were also mapped between *Xgwm129* and *Xbarc197* (492,893,704 to 492,893,948 bp) in Frontana and *QFhbp-jaas.5AL-1* was linked to BS00069175_51 (485,598,111 to 485,598,143 bp) (Steiner et al., 2004; Yi et al., 2018). Therefore, there is at least one important genetic locus on 5A to control FHB resistance. The *gFHB-5A* we identified in the present study is possibly the same with the *Qfhb.hwwg-5A* or *QFhbp-jaas.5AL-1* though they are from different wheat cultivars.

In Panel I, FHB-resistant loci on 2B and 7A were identified by GWAS. On 2B, significant SNPs mainly clustered into 9,740,162 to 68,200,954 bp with PVE of 2.71-4.17%, but it was only detected in Panel I. QTL on 2B was previously reported in different wheat accessions containing Ning7840 (Zhou et al., 2002) and Ernie (Liu et al., 2007). A QTL on 2BS was closely linked to *Xgwm210* in Renan and in Patterson/Goldfield population (Gervais et al., 2003; Gilsinger et al., 2005). The QTL *Qfhs.nc-2B.1* flanking with *IWB31987* and *IWB40514* (46.5-68.2 Mb) was identified in the Truman/MO 94-317 population, and this QTL was possibly the same with the *gFHB-2B* we identified in the present study. On 7A, significant SNPs mainly clustered in the interval of 515,126,041 to 537,557,064 bp, and it has long physical distance from the previous QTL (*XsdauK66* ~ *Xcfa2240*) on 7A identified in *Thinopyrum ponticum*. Moreover, *FHB.hau-4D* (wPt-8836 ~ wPt-4572) was identified by linkage mapping in UP population. Previous studies identified some FHB-resistant QTL on chromosome 4D in the population of ChineseSpring/SM3-7ADS and Spark/Arina (Draeger et al., 2007; Ma et al., 2006; Srinivasachary et al., 2008) but it need more work to confirm if they are the same locus. Although some resistance loci were excavated in this study, some loci were not very stable since they were only significant in one replicates possibly because of environmental influence. However, one stable resistance loci identified in this study could be considered to use for marker-assisted selection in improvement of FHB resistance in common wheat.

Abbreviations

FHB: *Fusarium* Head Blight

GWAS: Genome wide association study

DI: Disease index

SNP: Single nucleotide polymorphisms

PVE: Phenotypic variance explained

DON: Deoxynivalenol

PFT: Pore-forming toxin-like

MAF: Minor allele frequency

QTL: Quantitative trait locus

RIL: Recombinant inbred line

Declarations

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Author Contributions

FC conceived and designed the experiments; CS, JG, YR, YZ, CL, HH, XL, and HP performed phenotype observation, GWAS analysis and QTL mapping; CS and FC wrote the paper.

Competing interests

The authors declare that they have no competing interests.

Data Availability Statement

Not applicable.

Ethics approval and consent to participate

Not applicable.

Consent for publication

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Tables

Table 1 FHB resistance loci identified by GWAS in two panels.

Chrom.	Left marker		Right marker		Range (Mb)	Number of interval/total significant SNPs	PVE (%)	Panel and replicate
	Name	Position	Name	Position				
1A	AX-110577325	2936928	AX-110544824	63341634	60.40	32/35	2.74-9.02	Panel_IL_R2, Panel_IL_R1
1B	Kukri_c51864_225	582906917	BS00076330_51	626724382	43.82	4/6	5.79-10.00	Panel_IL_R1, Panel_IL_R2
1D	AX-95008362	228239	AX-109306242	5444062	5.22	6/7	2.74-3.44	Panel_IL_R2
2A	AX-111074099	703111613	AX-111137145	703559811	0.45	10/11	7.04-9.36	Panel_IL_R1
2B	AX-109423210	9740162	AX-89562766	68200954	58.46	55/60	2.71-4.17	Panel_IL_R2
2D	D_contig17313_245	9344681	Kukri_c27335_619	35053295	25.71	3/7	8.21-9.26	Panel_IL_R1, Panel_IL_R2
3A	Tdurum_contig76679_307	1326004	BS00110922_51	12813934	11.49	8/8	5.83-9.46	Panel_IL_R1, Panel_IL_R1, Panel_IL_R2
3B	AX-111764570	120960387	IACX5725	206195428	85.24	6/10	2.71-5.85	Panel_IL_R2, Panel_IL_R2
3D	Excalibur_c18826_452	2597023	AX-109337862	92375516	89.78	4/5	2.86-9.81	Panel_IL_R2, Panel_IL_R1, Panel_IL_R2
4A	AX-111132886	723385268			-	1/1	7.01	Panel_IL_R1
4B	Excalibur_c32467_676	534735432	Ex_c9296_858	589178585	54.44	4/4	5.83-6.65	Panel_IL_R2
4D	CAP8_c9110_427	121181674	Excalibur_c38704_1423	503621477	382.44	3/3	5.80-9.20	Panel_IL_R1, Panel_IL_R1, Panel_IL_R2
5A	wsnp_Ex_c16317_24795290	329828930	BobWhite_c15758_79	595372995	265.54	60/60	3.04-13.32	Panel_IL_R1, Panel_IL_R2, Panel_IL_R1, Panel_IL_R2
5B	Kukri_c35537_464	484637902	BS00022231_51	653655623	169.02	9/9	9.26-9.66	Panel_IL_R1, Panel_IL_R1
5D	GENE-3318_556	426001273	wsnp_JD_c825_1223424	544697840	118.70	6/6	2.90-10.67	Panel_IL_R2, Panel_IL_R1, Panel_IL_R2
6A	AX-94666339	616878	AX-95658689	31355419	30.74	5/6	2.89-11.68	Panel_IL_R2, Panel_IL_R1
6B	wsnp_Ex_c9750_16105678	613297099	Ku_c10647_585	618668841	5.37	4/8	5.75-5.75	Panel_IL_R2
6D	AX-94426737	13568801	AX-109353709	23486224	9.92	3/4	2.80-10.04	Panel_IL_R1, Panel_IL_R2, Panel_IL_R1
7A	AX-86179404	515126041	AX-110051209	537557064	22.43	49/57	2.71-7.04	Panel_IL_R1, Panel_IL_R2
7B	Tdurum_contig47317_100	675149692	AX-94482459	714012248	38.86	8/9	3.17-9.38	Panel_IL_R1, Panel_IL_R2, Panel_IL_R1, Panel_IL_R2
7D	AX-110419815	110637	AX-110963548	53290532	53.18	3/5	2.89-10.58	Panel_IL_R1, Panel_IL_R2, Panel_IL_R1

Table 2 Haplotype analysis and phenotypic comparison at the *gFHB-5A* locus in two panels

Population	Block	Position	Haplotype	Alleles
Panel I	Hap_5A1	577.93Mb	<i>PI_Hap_5A1a</i>	<i>TT/GG/CC/CC/CC/GG/GG/AA/TT/GG/GG/AA/CC/AA/AA/AA/CC/TT/TT/TT/GG</i>
		588.75Mb	<i>PI_Hap_5A1b</i>	<i>GG/AA/TT/TT/GG/AA/CC/GG/CC/AA/AA/GG/GG/GG/GG/GG/TT/CC/CC/AA/AA</i>
Panel II	Hap_5A1	331.4Mb	<i>PIL_Hap_5A1a</i>	<i>CC/GG/GG/GT/GG/GG/TT/AA/TT/AA/TT/AG/CC/AA/TT/CC/TT</i>
		422.08Mb	<i>PIL_Hap_5A1b</i>	<i>AC/GT/AG/GT/AG/AG/TT/AA/CC/GG/CC/AA/CT/GG/CT/AC/CT</i>
	Hap_5A2	591.16Mb	<i>PIL_Hap_5A2a</i>	<i>AA/TT/CC/GG/AG/AG/TT</i>
		595.37Mb	<i>PIL_Hap_5A2b</i>	<i>AG/CT/CT/AG/AA/GG/CC</i>
	-	-	<i>PIL_Hap_5A1a+PIL_Hap_5A2a</i>	-
	-	-	<i>PIL_Hap_5A1b+PIL_Hap_5A2b</i>	-

Different small and capital letters after numbers means the significance $P < 0.05$ and $P < 0.01$, respectively.

Figures

Figure 1

Phenotype distribution of wheat FHB resistance in two panels. (a) The mean value of FHB disease index of two panels. (b) The FHB disease index distribution of two panels.

Figure 2

(a-d) Manhattan plots of two replicates of Panel I and Panel II. (e-f) Q-Q plots of two replicates of Panel I and Panel II.

Figure 3

Number distribution of significant SNPs in Panel I (a), Panel II (b) and all varieties (c).

Figure 4

Haplotype analysis of significant SNPs at gFHB-5A locus. (a) LD heatmap of the significant SNPs in the region of gFHB-5A locus in Panel I and on block were showed in this region. (b) LD heatmap of the significant SNPs in the region of gFHB-5A locus in Panel II and two blocks were showed in this region. (c) Cultivars with haplotype of PI_Hap_5A1a showed stronger FHB resistance than PI_Hap_5A1b. (d) Cultivars with haplotype of PII_Hap_5A1a showed stronger FHB resistance than PII_Hap_5A1b. (e) Cultivars with haplotype of PII_Hap_5A2a showed stronger FHB resistance than PII_Hap_5A2b. (f) Cultivars with polymerized haplotype of PII_Hap_5A1a and PII_Hap_5A2a showed stronger FHB resistance than PII_Hap_5A1b and PII_Hap_5A2b.

Figure 5

(a) Percentages of superior alleles at the gFHB-5A locus in Panel I. (b) Percentages of superior alleles at the gFHB-5A locus in Panel II that cultivars released before 1980s, 1980s-2000s and after 2000s.

Figure 6

(a) LOD contours for FHB QTL on chromosomes 4D identified in the UC1110 × PI610750 population. (b) FHB resistance comparison of different alleles at the FHB.hau-4D locus. (c) QTL for FHB resistance in the UC1110 × PI610750 population.

Supplementary Files

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