Genetic determinants of wheat resistance to common root rot (spot blotch) and *Fusarium* crown rot

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Abstract

Due to the field soil changes, high density planting, and straw-returning methods, wheat common root rot (spot blotch) and *Fusarium* crown rot (FCR) have become severe threatens to global wheat productions. Only a few wheat genotypes show moderate resistance to these root and crown rot fungal diseases, and the genetic determinants of wheat resistance to these two devastating diseases have been poorly understood. This review summarizes the recent progress of genetic studies on wheat resistance to common root rot and *Fusarium* crown rot. Wheat germplasms with relative higher resistance are highlighted and genetic loci controlling the resistance to each of the disease are summarized.

Keywords: wheat, resistance, QTLs, common rot root, spot blotch, *Fusarium* crown rot

1. Introduction

Various long-term environmental changes have greatly shaped the epidemics of different crop diseases. For instance, the higher temperatures associated with the trends of global warming may increase the severity of many plant diseases (Cohen and Leach, 2020). The bursts of wheat stem base rot diseases, including common root rot and *Fusarium* crown rot, are highly correlated with the crop rotations. The large-scale application of wheat-maize rotation in the

North China wheat cultivation area has dramatically changed the organic carbon, fertilization state, and nitrogen balance of the soil (Zhao et al., 2006; Wang et al., 2015). The disease suppressive capacity of the soil microbiome is also largely dependent on crop rotational diversity (Peralta et al., 2018).

Wheat common root rot is caused by *Bipolaris sorokiniana* infection (**Fig. 1A**, teleomorph *Cochliobolus sativus*) in the root and stem base of wheat and barley. Severe infections of this fungal pathogen in the root and crown of seedlings may lead to fatal damage of plants. The same pathogen can also induce phenotypes of leaf spot (spot blotch, Helminthosporium leaf blight, or foliar blight, **Fig. 1B**), seedling wilt, head blight, and black point in *Triticeae* crops (Kumar et al., 2002). The average yield loss caused by *B. sorokiniana* ranges from 15% to 20%, but under favorable heat and drought conditions this disease can decrease wheat production by 70% and reduce seed quality (Sharma and Duveiller, 2007). This fungal pathogen accumulates several toxins including prehelminthosporol, helminthosporol, helminthosporic acid, sorokinianin, and bipolaroxin to kill or weaken plant cells (Kumar et al., 2002; Gupta et al., 2018). However, the potential negative impact of *B. sorokiniana*-infected wheat grains (black point) on food safety has not been investigated in detail. The fungus *B. sorokiniana* has a very wide host range, and can infect wheat, barley, maize, rice, and many other grass species (Gupta et al., 2018). Earlier studies suggested that multiple-year *Triticeae* crop rotations of wheat and barley greatly promote the severity of common root rot caused by *B. sorokiniana* (Conner et al., 1996). Maize crops and returned straws may also serve as infection hosts of this fungus, so common root rot and spot blotch have been more frequently observed in wheat cultivation areas in North China, where large-scale wheat-maize rotation and the use of straw returning have been applied.

Fusarium crown rot (FCR) is caused by infection of *Fusarium pseudograminearum* (**Fig. 1C**), or several other *Fusarium* pathogens including *F. culmorum*, *F. avenaceum*, and *F. graminearum*. These fungal species infect the coleoptile, leaf sheath, and stem base of wheat seedlings, generating browning and decay phenotypes. *Fusarium* pathogens are globally wide-spread in arid and semi-arid wheat planting areas (Kazan and Gardiner, 2018). The estimated yield loss of winter wheat due to FCR infections in the Northwest Pacific region of the United States reached 35% (Smiley et al., 2005). Moreover, when FCR-infected plants are

co-infected with *Fusarium* Head Blight (FHB), wheat seeds are likely to be contaminated by fungal toxins such as deoxynivalenol (DON) and nivalenol (NIV), which greatly threaten the health of human and livestock (Monds et al., 2005; Obanor and Chakraborty, 2014). Maize also can sever as host to various *Fusarium* pathogens, and the fungi from infected plants can remain active in returned straw debris as long as five years (Burgess et al., 2001). For these reasons, FCR is a growing threat to wheat cultivation in wheat-maize rotation regions in North China.

These two diseases share several similar phenotypes such as stem base rot, head blight, and seed contamination, but they can be distinguished by their phenotypic features. For common root rot caused by *B. sorokiniana*, the infected wheat plants can be easily pulled out, the stem base and root system feel wet, and black and brown striped spots can be observed in both the stem base and lower leaves (**Fig. 1B**). For FCR caused by *F. pseudograminearum*, the stem base of the infected wheat plant is dry and fragile, and can be easily broken apart, and dark and red brown rot can be observed in the stem base (**Fig. 1D**).

2. Progress in dissecting the genetic determinants of wheat resistance to common root rot (spot blotch)

Breeding of wheat resistant cultivars remains the most efficient and economical way to control common root rot (spot blotch). However, there are insufficient germplasm resources with resistance to common root rot to meet the needs for global wheat breeding applications and there have been few studies to identify the genetic loci that control resistance to common root rot (Gupta et al., 2018). Early effort focused on the introgression of common root rot resistant loci from wheat relative *Thinopyrum ponticum* species (Li et al., 2004). Requiring complex quantitative trait loci (QTL), wheat breeding programs for common root rot resistance have faced many obstacles (Joshi et al., 2004). Using bi-parental populations and linkage mapping, four genetic loci with major resistant effect were identified and designated as *Sb* genes. *Sb1* was discovered in the bread wheat line “Saar”, was mapped to chromosome 7DS, and is associated with the wheat leaf rust resistance gene *Lr34* (Lillemo et al., 2013). The *Lr34/Yr18/Pm38* gene encodes a ATP-binding cassette (ABC) transporter that confers broad-spectrum resistance to multiple foliar fungal diseases, including leaf rust, stripe rust,

and powdery mildew (Krattinger et al., 2009). Another minor QTL that corresponded with *Lr46* on chromosome 1BL was also identified from "Saar". The *Lr46* gene is associated with adult plant slow rusting resistance toward leaf rust and is also associated with stripe rust resistance gene *Yr29* (William et al., 2003). The *Sb2* gene was identified in bread wheat cultivar "Yangmai 6" and was mapped to chromosome 5BL between simple sequence repeat (SSR) markers of *Xgwm639* and *Xgwm1043* (Kumar et al., 2015). The *Sb2* gene was later reported to be linked with the *Tsn1* gene, which confers host-selective sensitivity to the fungal toxin ToxA produced by *Pyrenophora tritici-repentis* (Kumar et al., 2016). The *Sb3* gene was discovered in the winter wheat line "621-7-1" as providing immune response to *B. sorokiniana*. Using bulked segregant analysis (BSA), *Sb3* was mapped to chromosome 3BS, flanking SSR markers of *Xbarc133* and *Xbarc147* (Lu et al., 2016). The *Sb4* gene was recently identified from two highly resistant wheat lines "Zhongyu1211" and "GY17". Using RNA-based BSA and single-nucleotide polymorphism (SNP) mapping, *Sb4* was delimitated in a 1.19 cM genetic interval region of chromosome 4BL, which contains 21 predicted genes in the corresponding "Chinese Spring" genome (Zhang et al., 2020). Future efforts to clone these designated *Sb* genes with major resistant effect may help elucidate the mechanism of wheat resistance toward this devastating fungal pathogen.

Several other major QTLs have been discovered and preliminarily mapped using bi-parental populations. For instance, in an earlier investigation, two resistant QTLs derived from "Yangmai 6" were mapped to chromosomes 5B and 7D using microsatellite markers (Kumar et al., 2005). Three QTLs on chromosomes 5B, 6A, and 6D were designated based on analysis of SSR markers from resistant genotype "G162" (Sharma et al., 2007). Four QTLs controlling resistance of wheat cultivar "Yangmai 6" to *B. sorokiniana* were mapped to chromosomes 2AL, 2BS, 5BL, and 6DL (Kumar et al., 2009). A total of seven QTLs providing resistance to *B. sorokiniana* infections were designated in wheat lines "Ning 8201" and "Chirya 3" (Kumar et al., 2010). Three QTLs on chromosomes 1BS, 3BS, and 5AS explained 8.5%, 17.6%, and 12.3%, respectively of resistant effect in "SYN1", a CIMMYT (International Maize and Wheat Improvement Center) synthetic-derived bread wheat line (Zhu et al., 2014). From a Brazilian resistant cultivar "BH 1146", two QTLs on chromosomes 7BL and 7DL were mapped using microsatellite markers (Singh et al., 2016). A prominent

resistant QTL near the *Vrn-A1* locus on chromosome 5AL was found in "BARTAI" and "WUYA" CIMMYT breeding lines (Singh et al., 2018). QTLs in *Vrn-A1* and *Sb2/Tsn1* loci were also detected in two other CIMMYT breeding lines, "CASCABEL" and "KATH" (He et al., 2020).

Genome-wide association study (GWAS) can also be used to map QTLs. Using 832 polymorphic Diversity Arrays Technology (DArT) markers, four QTLs resistant to spot blotch were mapped to chromosomes 1A, 3B, 7B, and 7D after analysis of 566 spring wheat germplasms (Adhikari et al., 2012). With recent progress in drafting the physical genome of hexaploid wheat (Appels et al., 2018), high-throughput SNP toolkits are now available for GWAS on various complex traits of wheat (Sun et al., 2020). A total of 528 spring wheat genotypes from different geographic regions were tested for spot blotch resistance and eleven associated SNP markers were found by 9K SNP assay (Gurung et al., 2014). A phenotypic screening of 11 parental genotypes and 55 F_2 lines identified "19HRWSN6" as a resistant source. Subsequent simple linear regression analysis revealed SSR markers on chromosomes 5B, 6A, and 7D associated with the resistance to *B. sorokiniana* (Tembo et al., 2017). Another study evaluated the responses of 294 hard winter wheat genotypes to *B. sorokiniana* and performed GWAS by 15K SNP assay. A total of ten wheat genotypes with relatively high resistance were identified and six major resistant QTLs were designated to collectively explain 30% of the phenotypic variation (Ayana et al., 2018). A total of 159 spring wheat genotypes were screened for common root rot resistance and twenty-four QTLs were identified, with a major one on chromosome 7B that explained 14% of the phenotypic variation of spot blotch severity (Jamil et al., 2018). Another study profiled the resistant phenotype of 287 spring wheat germplasms and performed GWAS using 90K SNP array. A total of eight genetic loci associated with incubation period, lesion number, and disease score of *B. sorokiniana* infection were detected (Ahirwar et al., 2018). A recent study phenotyped 301 Afghan wheat germplasms and found that approximately 15% exhibited lower disease scores than the resistant control. Subsequent a GWAS approach identified twenty-five marker-trait associations on more than twelve chromosomes, including previously identified *Vrn-A1* and *Sb2/Tsn1* loci (Bainsla et al., 2020). Another 141 spring wheat lines were collected for GWAS on spot blotch resistance. A total of 23 genomic regions were identified,

including several stable regions on chromosomes 2B, 5B and 7D, and a novel region on chromosome 3D (Tomar et al., 2020).

We have summarized the previously reported wheat germplasms with relatively higher resistance to *B. sorokiniana* **(Table 1)**. These wheat materials may serve as valuable resources for the genetic improvement of wheat resistance to common root rot (spot blotch). We have also summarized detailed information of previously designated resistant QTLs **(Table 1)** and drafted their genomic distribution using the released genome of hexaploid wheat **(Fig. 2)**.

3. Genetic loci controlling wheat resistance to *Fusarium* crown rot

Since the causal agent of *Fusarim* head blight (FHB), *Fusarium graminearum*, can also induce the phenotype of *Fusarium* crown rot in certain regions (Akinsanmi et al., 2006; Zhou et al., 2019), it is likely that FHB-resistant germplasms and genetic loci can be exploited to improve FCR resistance. For instance, the recently cloned FHB resistance gene *Fhb7* encodes a glutathione S-transferase (GST) and provides broad resistance to *Fusarium* diseases, including FCR induced by *F. pseudograminearum*, by detoxifying trichothecenes through de-epoxidation (Wang et al., 2020). However, an earlier investigation found no significant correlation of resistant phenotype or genetic loci conferring resistance to FHB and FCR in the same wheat genotypes (Li et al., 2010). A recent large-scale phenotyping of 205 Chinese wheat cultivars for resistance to both FHB and FCR also found no correlation (Shi et al., 2020). Great efforts have also been towards identification of FCR-resistant barley germplasms and genetic loci that control the FCR-resistance in barley (Liu and Ogbonnaya, 2015). Since recent review papers have already summarized QTLs conferring FHB resistance and susceptibility in wheat in detail (Buerstmayr et al., 2020; Fabre et al., 2020), here we have mainly focused on studies reporting wheat resistance to FCR induced by *F. pseudograminearum* and *F. culmorum*.

A series of genetic studies was performed to reveal a major QTL on chromosome 3BL (*Qcrs.cpi-3B*). This resistant locus, *Qcrs.cpi-3B*, was derived from wheat genotype “CSCR6” belonging to the taxon *Triticum spelta* (Ma et al., 2010). In a wheat recombinant inbred line population of “Lang/CSCR6”, a QTL on chromosome 4B derived from “Lang” was responsible for the soil-free FCR resistance (Yang et al., 2010). Another significant QTL on

chromosome 6B, but not *Qcrs.cpi-3B*, was identified and responsible for the FCR resistance during an introgression process for durum wheat using "CSCR6" as the donor parent (Ma et al., 2012b). Near-isogenic lines for the *Qcrs.cpi-3B* locus were developed for both genetic research and breeding (Ma et al., 2012a), and subsequent transcriptome and allele specificity analysis revealed differentially expressed genes associated with this locus (Ma et al., 2014). Fine mapping of this QTL shortened the genetic interval to 0.7 cM, containing a total of 63 coding genes in the reference wheat genome (Zheng et al., 2015). Future map-based cloning and validation of the functional gene in this large-effect QTL may provide valuable clues for us to understand the molecular bases of wheat resistance to FCR.

Other resistant QTLs have been identified using bi-parental populations. Early investigation discovered a resistant locus near the dwarfing gene *Rht1* on chromosome 4B from the wheat cultivar "Kukri" (Wallwork et al., 2004). Inherited from a wheat line "W21MMT70" with partial resistance to FCR, two QTLs were identified and mapped to chromosomes 2D and 5D (Bovill et al., 2006). A major QTL on chromosome 1DL (*QCr.usq-1D1*) and several minor QTLs were identified in wheat line "2-49 (Gluyas Early/Gala)" using SSR markers (Collard et al., 2005; Collard et al., 2006). An initial FCR resistance screening of 32 wheat genotypes revealed "2-49", "Aso zairai 11", and "Ernie" as resistant sources. A QTL derived from "Ernie" was mapped to chromosome 3BL close to markers *wPt-1151* and *wPt-1834* (Li et al., 2010). Another study reported that an Australian spring wheat cultivar "Sunco" showed partial resistance to FCR induced by *F. pseudograminearum*. Using bi-parental QTL mapping, a major QTL was identified on chromosome 3BL, between SSR markers *Xgwm247* and *Xgwm299* (Poole et al., 2012). These resistant sources of "W21MMT70", "2-49", and "Sunto" were then employed for QTL pyramiding (Bovill et al., 2010). Four FCR-resistant QTLs were designated and their resistant alleles were derived from the bread wheat commercial variety "EGA Wylie". Major QTLs on chromosomes 5DS and 2DL were consistently detected in all three populations and two minor QTLs were mapped to chromosome 4BS (Zheng et al., 2014). QTL mapping was also performed to find genetic loci controlling partial resistance to FCR in the four wheat germplasms "2-49", "Sunco", "IRN497", and "CPI133817". FCR resistance was evaluated in both seedlings and adult plants and a total of six QTLs among these resistant wheat sources

were detected (Martin et al., 2015).

A GWAS approach was used to screen 2,514 wheat genotypes for FCR resistance and identified two major QTLs on chromosome 3BL explaining between 35% and 49% of the phenotypic variation using DArT and SSR markers (Liu et al., 2018). A set of 126 spring bread wheat lines from CIMMYT were phenotyped against FCR induced by *F. culmorum* and further genotyped using DArT markers, which resulted in the identification of three major QTLs on chromosomes 3B and 2D (Erginbasorakci et al., 2018). The use of GWAS for FCR resistance has greatly benefited from advanced high-throughput sequencing techniques and the released hexaploid wheat genome. A total of 234 Chinese wheat cultivars were evaluated for FCR resistance in four greenhouse experiments, with GWAS conducted using a high-density 660K SNP assay. This revealed a major QTL on chromosome 6A, which was subsequently validated using a bi-parental population of "UC1110/PI610750" (Yang et al., 2019). A recent GWAS approach phenotyped 358 Chinese germplasms for FCR resistance and less than 10% of germplasms showed a lower disease index. The wheat 55K SNP assay was applied for the association analysis, resulting in detection of significant QTLs on chromosomes 1BS, 1DS, 5DS, 5DL, and 7BL (Jin et al., 2020). Another GWAS was performed to evaluate FCR resistance of 161 wheat accessions under growth room and greenhouse conditions using *F. culmorum* as the pathogen. Using a 90K SNP array, a total of fifteen QTLs for FCR resistance were detected with one major QTL on chromosome 3BS near the FHB resistance *Fhb1* locus (Pariyar et al., 2020). A marker-assisted recurrent selection approach was performed on two populations to pyramid minor FCR-resistant QTLs. Using 9K SNP array, a total of 23 marker-trait associations were identified by GWAS (Rahman et al., 2020).

In **Table 2**, we have summarized wheat germplasms resistant to FCR induced by either *F. pseudograminearum* or *F. culmorum*. Reported QTLs controlling FCR resistance are also highlighted (**Table 2**), with their genomic distributions annotated using the wheat genome database (**Fig. 3**).

4. Conclusion and future perspectives

We have briefly introduced two rot diseases that are commonly observed in the stem base of

the wheat plant (**Fig. 1**). Both of diseases have become major threats to wheat productions in wheat-maize rotation areas with large-scale application of straw returning. Wheat breeding is still the most efficient way to control these two devastating fungal diseases. However, as summarized in this review (**Tables 1 and 2**), there are few wheat germplasms with relative high resistance to either *B. sorokiniana* or *F. pseudograminearum*. Large-scale screening of wheat germplasms that are resistant to these diseases is still urgently needed for effective wheat breeding applications.

Genetic improvement of wheat resistance to these two diseases will also be facilitated by exploring novel QTLs that control resistance and dissecting functional genes within these QTLs. In this review, we list previously reported resistant QTLs (**Tables 1 and 2**) and present their genomic distributions based on the updated wheat genome (**Figs. 2 and 3**). For identified QTLs conferring resistance to *B. sorokiniana*, may be associations with certain resistant loci responsible for wheat resistance to other foliar fungal diseases, such as *Lr34/Yr18/Pm38*, *Lr46/Yr29*, and *Tsn1*. Wheat leaves might restrain the infection of different foliar fungal diseases by utilization of similar molecular approaches mediated by these resistant genes. More wheat germplasms with broad-spectrum resistant loci should be evaluated for their potential resistance to spot blotch or common root rot induced by *B. sorokiniana.* For QTLs controlling resistance to *Fusarium* crown rot, ones that also have resistance to FHB may be more valuable, since the major causal agents of these diseases (*F. pseudograminearum*, *F. culmorum*, and *F. graminearum*) very likely co-exist in a cultivation environment. Studies of FCR-resistant wheat germplasms that investigate the genetic determinants of FCR-resistance can build on the work performed to investigate resistance to FHB. Progress in wheat genome research and increased availability of high-density SNP toolkits will facilitate the use of GWAS on collected wheat germplasms to efficiently identify resistant germplasms and genetic loci.

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Table 1. Genetic bases of wheat resistance to common root rot (spot blotch).

QTL name	Associated markers or SNPs	Resistant wheat germplasms	Reference
Sb1/Lr34*	7DS: ***Xgwm295***, csLV34		
Qsb	7DS: *wPt-7654*, ***gdm88***	Saar	(Lillemo et al., 2013)
Qsb/Lr46/Yr29*	1BL: ***wmc719***, *hbe248*, *ncw1-V*		
Sb2/Tsn1*	5BL: ***Xgwm499***, *Xgwm639*, *Xgwm1043*	YS116, CASCABEL	(Kumar et al., 2015; Kumar et al., 2016; Bainsla et al., 2020; He et al., 2020)
Sb3*	3BS: ***Xbarc147***, *XWGGC3957*, *XWGGC4320*	621-7-1	(Lu et al., 2016)
Sb4*	4B: ***TraesCS4B01G295400.1***	Zhongyu1211, GY17	(Zhang et al., 2020)
Qsb	5B: ***Xgwm544*** 7D: ***Xgwm437***	Yangmai 6	(Kumar et al., 2005)
Qsb	5B: ***Xgwm67***	G162	(Sharma et al., 2007)
QSb.bhu-2A	2AL: *Xbarc353*, ***Xgwm445***		
QSb.bhu-2B	2BS: ***Xgwm148***, *Xgwm374*	Yangmai 6	(Kumar et al., 2009)
QSb.bhu-5B	5BL: *Xgwm067*, ***Xgwm371***		
QSb.bhu-6D	6DL: ***Xbarc175***, *Xgwm732*		
QSb.bhu-2A	2AS: ***Xgwm425***, *Xbarc159*		
QSb.bhu-2B	2BS: ***Xgwm148***, *Xbarc91*	Ning 8201	(Kumar et al., 2010)
QSb.bhu-5B	5BL: ***Xgwm067***, *Xgwm213*		
QSb.bhu-7D	7DS:***Xgwm111***, *Xgwm1168*		
QSb.bhu-2B	2BS: ***Xgwm148***, *Xgwm129*		
QSb.bhu-2D	2DS: ***Xgwm455***, *Xgwm815*		
QSb.bhu-3B	3BS: *Xgwm533*, *Xgwm1037*	Chirya 3	(Kumar et al., 2010)
QSb.bhu-7B	7BS: *Xgwm263*, *Xgwm255*		
QSb.bhu-7D	7DS: ***Xgwm111***, *Xswm008*		
QSb.cim-1B	1B: ***Xwmc128***, *Xgwm374*		
QSb.cim-3B	3B: ***990937|F|0***, *1123330|F|0*	SYN1, Mayoor, Tksn1081/Ae. squarrosa (222)	(Zhu et al., 2014)
QSb.cim-5A	5A: ***1086218|F|0***, *982608|F|0*		
QSb.iiwbr-7B	7BL: *wmc758*, ***wmc335***	BH 1146	(Singh et al., 2016)
QSb.iiwbr-7D	7DL: *wmc653*, ***barc121***		
Qsb/Vrn-A1*	5AL: ***Vrn-A1***	BARTAI, WUYA, CASCABEL, KATH	(Singh et al., 2018; Bainsla et al., 2020; He et al., 2020)
Qsb	1A: *wPt-730148*, ***wPt-668214*** 3B: ***wPt-1159, wPt-5769*** 7B: *wPt-2838*	Chirya 7, Forma Vinda de Varmland (PI 192569), IWA8600074 (PI 623098), Trigo (PI 477878), Soprimo (PI 479890), CI 10112 (PI 78814), Florentino (PI 565255), AW	(Adhikari et al., 2012)

	7D: *wPt-664459*	6635A/86 (PI 572693), IWA8611737 (PI 625572), NW56A (PI 429667)	
Qsb	1B: ***wsnp_Ex_c24700_33953160*** 5A: ***wsnp_Ex_c15342_23592740, wsnp_Ku_c17951_27138894*** 5B: ***wsnp_Ex_rep_c70120_69069789, wsnp_Ku_c20701_30355248*** 6B: ***wsnp_Ex_c15785_24157360*** 7B: ***wsnp_Ex_c52527_56097039***	PI25989, PI384237, PI384239, PI479802, PI479890, PI576639, PI245377, PI366685, PI481715, PI624517, PI481574, PI91235, PI350795, PI565213	(Gurung et al., 2014)
Qsb	5B: ***Xgwm544*** 6A: ***Xwgm570*** 7D: ***Xgwm437***	19HRWSN6, 30SAWSN5	(Tembo et al., 2017)
QSb.sdsu-2D.1 *QSb.sdsu-3A.1* *QSb.sdsu-4A.1* *QSb.sdsu-4B.1* *QSb.sdsu-5A.1* *QSb.sdsu-7B.1*	2D: ***Kukri_c31121_1460*** 3A: ***Excalibur_c46082_440*** 4A: *IWA8475* 4B: ***Excalibur_rep_c79414_306*** 5A: ***Kukri_rep_c104877_2166*** 7B: *TA005844-0160*	Duster, Colt, Custer, Intrada, MT0495, NE99495, OK04525, OK05122, OK05723W, Venango	(Ayana et al., 2018)
Qsb	1A: ***S1A_582293281*** 2A: ***S2A_16824871*** 3A: ***S3A_378506623*** 4B: ***S4B_554842477*** 5A: ***S5A_50162259*** 5B: ***S5B_513590441, S5B_504309131, S5B_528990456*** 6B: ***S6B_9296088, S6B_673978653*** 7A: ***S7A_483878120*** 7B: ***S7B_749474154***	Chirya.3, Aust-53, Pak-13, SB12-6704, 7HTWSN-4516, 7HTWSN-4513, Aust-8, SB12-6703, Aust-66, SB12-6720, Aust-12, 7HTWSN-4522, 7HTWSN-4526, 7HTWSN-4412, 7HTWSN-4405, 7HTWSN-4517, H.Sat-8, Aust-59, Aust-29, 7HTWSN-4406, 7HTWSN-4510	(Jamil et al., 2018)
Qsb	1B: ***BobWhite_c17559_105*** 4A: ***BobWhite_c20322_153, BobWhite_c17524_242*** 5B: ***Tdurum_contig25513_123****, tplb0027f13_1493* 6A: ***wsnp_Ra_c2270_4383252*** 6B: ***BS00092845_51*** 7A: ***Ku_c15750_761***	N. A.	(Ahirwar et al., 2018)
Qsb	1B: ***TraesCS1B01G416200*** 5A: ***TraesCS5A01G391400, TraesCS5A01G369700***	0KATIA, DE9, OK82282//BOW/NKT/3/F4105, PSN/BOW//ROEK/3/MILAN, KAUZ 2*/OPATA//KAUZ, ALTAR84/AE.SQ//2*, CNDO/R143//ENTE/MEXI-2/3/…, PAMIR-94 x, NING9415, RENESANSA, VORONA/CUPE	(Bainsla et al., 2020)
Qsb	1A: ***TraesCS1A01G018700*** 1B: ***TraesCS1B01G424000, TraesCS1B01G423900***	N. A.	(Tomar et al., 2020)

	1D: ***TraesCS1D01G012500, TraesCS1D01G012900*** 2B: ***TraesCS2B01G505200, TraesCS2B01G552700, TraesCS2B01G12400, TraesCS2B01G30100*** 3A: ***TraesCS3A01G107400, TraesCS3A01G103000*** 3B: ***TraesCS3B01G520100*** 3D: ***TraesCS3D01G537500*** 5A: ***TraesCS5A01G402700, TraesCS5A01G457100*** 5B: ***TraesCS5B01G066200, TraesCS5B01G224500, TraesCS5B01G521500*** 6A: ***TraesCS6A01G061900*** 7A: ***TraesCS7A01G504700, TraesCS7A01G530700*** 7B: ***TraesCS7B01G002400, TraesCS7B01G003000, TraesCS7B01G169400*** 7D: ***TraesCS7D01G067000, TraesCS7D01G081100, TraesCS7D01G221000***

Genomic distribution of all these summarized resistant loci were drafted using associated markers and SNPs (bold labeled) that can be found in "Chinese Spring" wheat genome database. QTLs with major effect or linked with designated genes were labeled with asterisk (*) and highlighted in **Fig. 2**.

Table 2. Genetic loci controlling wheat resistance to *Fusarium* crown rot.

QTL name	Associated markers or SNPs	Resistant wheat germplasms	Reference
Qcrs.cpi-3B*	3BL: ***Xgwm0181***, *wPt-10505, wPt-2277*	CSCR6 (*T. spelta*), Lang, Kennedy	(Ma et al., 2010; Yang et al., 2010; Ma et al., 2012a; Ma et al., 2012b; Ma et al., 2014; Zheng et al., 2015)
Qcsr.cpi-4B	4BS: ***wPt-5334***, *wPt-4918*, ***Xbarc199***		
Qcr	5A: ***Xwmc110***		
Qcr	6B: ***Xwmc494, Xgwm193, Xwmc397, Xbarc198, Xbarc178***		
Qcr	2BS: *Xgdm086*, ***Xbarc200***	W21MMT70, Mendos	(Bovill et al., 2006)
	2D: *Xwmc018, Xwmc190*		
	5D: ***Xbarc205***, *barc143*		
Qcr	1AL: ***Xwmc120***, *Xwmc312*	Kukri, 2-49 (Gluyas Early/Gala), Janz	(Wallwork et al., 2004; Collard et al., 2005; Collard et al., 2006)
QCr.usq-1D.1	1DS: *Xcfd19*		
QCr.usq-2B.1	2BS: *Xbarc349.1*, ***Xgwm388***		
Qcr/Rht1*	4BL: *Xgwm165*, ***Xgwm251***		
Qcr	7BS: ***Xgwm400***, *Xwmc476*		
QCr.usq-1D.1	1DS: ***wPt-3738***, *Xcfd19, wPt-9380*	2-49, W21MMT70, Sunto	(Bovill et al., 2010)
QCr.usq-2B.2	2B: *wPt-5374*, ***wPt-0434***		
QCr.usq-3B.1	3BL: ***wPt-7301***, *wPt-0365*		
QCr.usq-4B.1	4BS: ***wPt-4535***, *Xgwm251*		
Qcr	7AS: ***wPt-4748***, *wPt-8418*		
Qcr	3B: ***wPt-1834***, *wPt-1151*	2-49, Aso zairai 11, Ernie	(Li et al., 2010)
Qcrs.wsu-3BL	3BL: *Xgwm247*, ***Xgwm299***	Sunco, Macon, Otis	(Poole et al., 2012)
Qcr	3BS: ***wPt-5390***, *Xwmc777*		
Qcr	7AS: ***wPt-3702***		
Qcrs.cpi-2D	2DL: ***1131013|F|0***, *1246993|F|0*	EGA Wylie	(Zheng et al., 2014)
Qcrs.cpi-4B.1	4BS: ***100004319|F|0***, *2324159|F|0*		
Qcrs.cpi-4B.2	4BS: ***1108472|F|0***, *1093616|F|0*		
Qcrs.cpi-5D	5DS: ***1215315|F|0***, *1237596|F|0*		
Qcr	1AS: ***Xbarc148***, *Xgwm164*	2-49, Sunco, IRN497, CPI133817	(Martin et al., 2015)
	1BS: *Xcfd65*, ***Xgwm11***		
	1DL: *Xcfd19*, ***Xwmc216***		
	2A: ***Xgwm95,*** *Xcfa2043*		
	2B: ***Xgwm630***, *Xcfa2278*		
	2DS: ***Xgwm484***, *Xgwm102*		
	3AL: ***Xcfa2134,*** *Xcfa2262*		
	3BL: ***Xgwm299,*** *wPt-0021*, ***Xwmc236***, *wPt-0365*		

	4BS: *Xwmc467, Xgwm165* 4BS: ***Xbarc193***, *Xwmc349* 6DL: *Xcfd188,* ***Xcfd47*** 6DL: *Xbarc196,* ***Xbarc273***		
Qcr	2DS: ***wPt-669517*** 3BS: *wPt-2193, wPt-22988, wPt-732330,* ***wPt-2766***	2-49, Sunco, Altay-2000	(Erginbasorakci et al., 2018)
QFCR.heau-2A *QFCR.heau-2D* ***Qcr-6AL**** *QFCR.heau-6A* *Qcr-6B* *Qcr-6D*	2AS: *Xwms382, wPt-7462, wPt-3757* 2DS: ***Xcfd53*** 6AL: ***AX-111106634,*** *AX-94534539* 6AS: ***Xbarc3***, *Xwmc754* 6B: ***SNP position 534,514,143*** 6D: ***SNP position 354,819,336***	Xunmai 118, Kaimai 26, Yanke 316, Xuke 732, Zhonglemai 9, Jinmai 1, Shenzhou 209, Fannong 1, Jiyanmai 7, UC1110, PI610750	(Yang et al., 2019)
Qcr *Qcr* *Qcr* *Qcr* *Qcr* ***Qcr-5DL**** *Qcr* *Qcr*	1BS: ***Affx-88612017, Affx-109495423*** 1DS: ***Affx-92108178, Affx-109205872*** 2AL: ***Affx-111557509*** 5DS: ***Affx-88597504, Affx-110248324*** 5AL: ***Affx-109253960*** 5DL: ***Affx-110484766, Affx-110079634*** 6BS: ***Affx-110282972*** 7BL: ***Affx-109846651, Affx-109540847***	Henong 982, Shiyou 17, Bao 6818, Quanmai 890, 04 Zhong 36, Junda 129, Xu 10054, Fanmai 5, Lian 0809, Shixin 733, Shi05-6678, Han 06-5170, Luomai 8, Zhongyuanzhixing, Yangao 21, Xumai 33	(Jin et al., 2020)
Qcr *Qcr* ***Qcr/Fhb1**** *Qcr* *Qcr* *Qcr* *Qcr* *Qcr* *Qcr* *Qcr* *Qcr*	2AL: *Kukri_c57491_156* 3AS: ***wsnp_Ra_c16278_24893033, CAP8_c1393_327*** 3BS: ***CAP12_rep_c3868_270*** 3DL: ***wsnp_Ex_c14027_21925404*** 4BS: ***wsnp_Ku_c12399_20037334*** 4BL: ***RAC875_rep_c72961_977*** 5BS: ***wsnp_Ku_c17875_27051169, Excalibur_c23304_353*** 5DS: ***RAC875_rep_c111521_246*** 5DL: ***Excalibur_c2795_1518*** 6BS: *RAC875_c17297_341* 6BL: ***BobWhite_c19298_97*** 6DS: *BS00021881_51*	VICTORYA, Katea, KOLLEGA, DORADE-5/3/BOW"S"/GEN//SHAHI, 2180*K/2163//?/3/W1062A*HVA114/W3416, L 4224 K 12, NE04424, TX69A509.2//BBY/FOX/3/GRK//NO64/PEX/4/CER/5/KAUZ//ALTA R 84/AOS, ID800994.W/MO88	(Pariyar et al., 2020)
Qcr	1A: ***BobWhite_c1027_1127, wsnp_Ku_c183_358844*** 1B: ***BS00070139_51, Tdurum_contig13117_1316*** 1D: ***wsnp_Ex_c3372_6195001*** 2D: ***BS00062567_51*** 3B: ***BS00072994_51, BS00079029_51, IACX11310***	AUS29529/2/2.49/Cunningham//Kennedy/3/Sunco, CSCR16/2/2.49/Cunningham//Kennedy/3/Sunco/2*Pastor	(Rahman et al., 2020)

	4A: ***BS00035307_51*** 4B: ***Ku_c3385_521*** 5B: ***BS00032003_51, BobWhite_c6094_447*** 6B: ***RAC875_c60007_199*** 7A: ***BobWhite_c33300_159, wsnp_JD_c1219_1766041*** 7B: ***wsnp_be352570B_Ta_2_1***		
N. A.	N. A.	Cunmai633, LS4607, Pubing01, Hongyun2**,** Jimai216, Fengyunmai5, Huaihe15076, Luofeng2419, Yanfeng168, Zhengmai22, Zhoumai38, Zhoumai37, Lemai185, Xinmai38, Xinong733, Xinmai45, Guohemai12, Xinong625, Zhengmai162	(Shi et al., 2020)

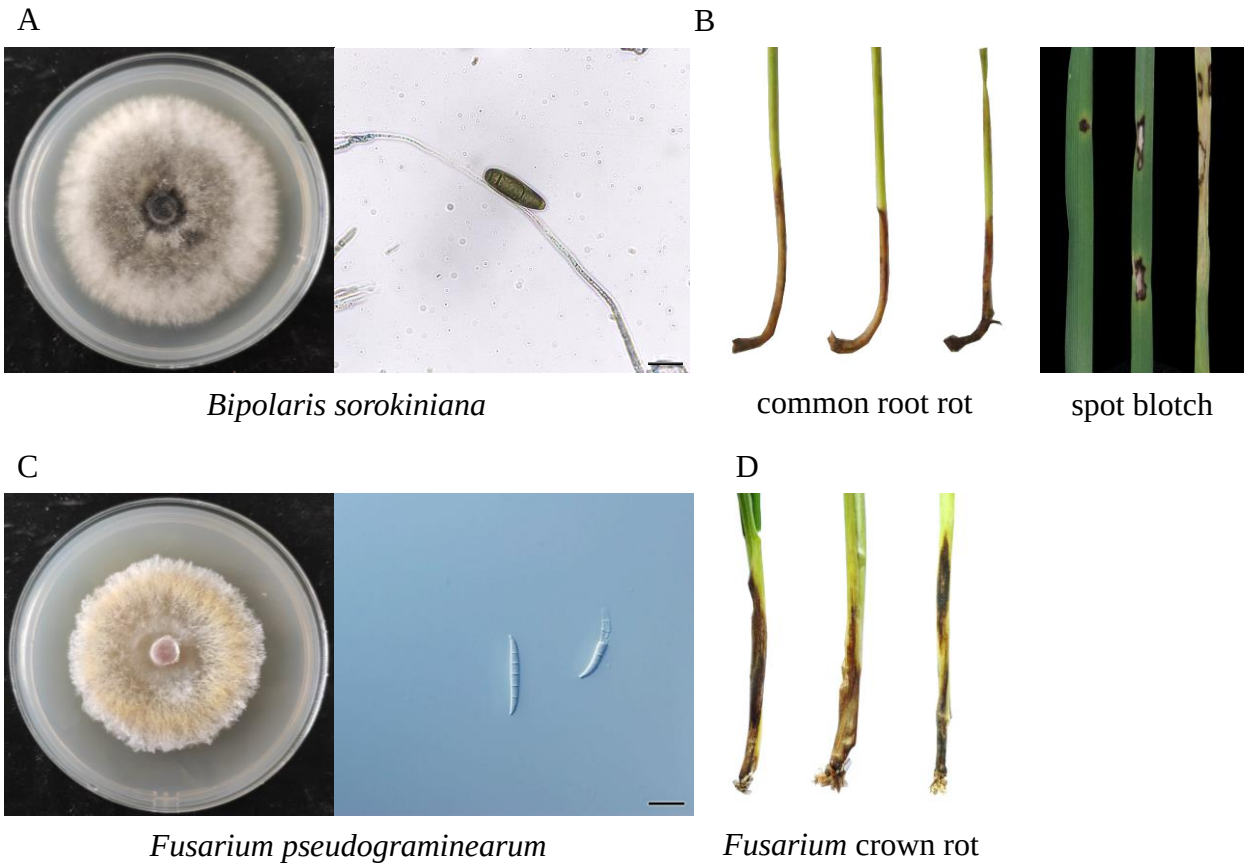
Genomic distribution of all these summarized resistant loci were drafted using associated markers and SNPs (bold labeled) that can be found in "Chinese Spring" wheat genome database. QTLs with major effect or linked with designated genes were labeled with asterisk (*) and highlighted in **Fig. 3**.

Figure legends

Fig. 1 Pathogenic profiles of *Bipolaris sorokiniana* and *Fusarium pseudograminearum.* (A) *B. sorokiniana* cultivated on PDA (potato dextrose agar) medium. Spores were directly collected from *B. sorokiniana* cultures on PDA medium. **(B)** Common root rot and spot blotch caused by *B. sorokiniana*. Infected wheat plants can be easily pulled out, the stem base and root system feel wet, and black and brown striped spots can be observed in both the stem base and lower leaves. **(C)** *F. pseudograminearum* cultivated on PDA medium. Spores of *F. pseudograminearum* can be induced on CMC (carboxymethyl cellulose sodium) medium. **(D)** *Fusarium* crown rot caused by *F. pseudograminearum*. The stem base of an infected wheat plant is dry and fragile, so can be easily broken apart. Additionally, dark and red brown rot can be observed in the stem base. Scale bar = 20 μm.

Fig. 2 Genetic determinants of wheat resistance to common root rot (spot blotch). Molecular markers, SNPs, and genes associated with common root rot or spot blotch resistant QTLs were collected from previous publications and searched against the JBrowse-1.12.3-release of common wheat "Chinese Spring" genome available from the "Triticeae Multi-omics Center (http://202.194.139.32/)". Physical positions were used to generate a distribution map of all the collected QTLs using Mapchart v2.32 software. QTLs with major effect or linked with designated genes are highlighted in red. Detailed information for these QTLs can be found in **Table 1**.

Fig. 3 Genetic loci controlling wheat resistance to *Fusarium* crown rot. Molecular markers, SNPs, and genes associated with FCR-resistant QTLs were collected from previous publications and searched against the JBrowse-1.12.3-release of common wheat "Chinese Spring" genome available from the "Triticeae Multi-omics Center (http://202.194.139.32/)". Physical positions were used to generate a distribution map of all the collected QTLs using Mapchart v2.32 software. QTLs with major effect or linked with designated genes are highlighted in red. Detailed information for these QTLs can be found in **Table 2**.



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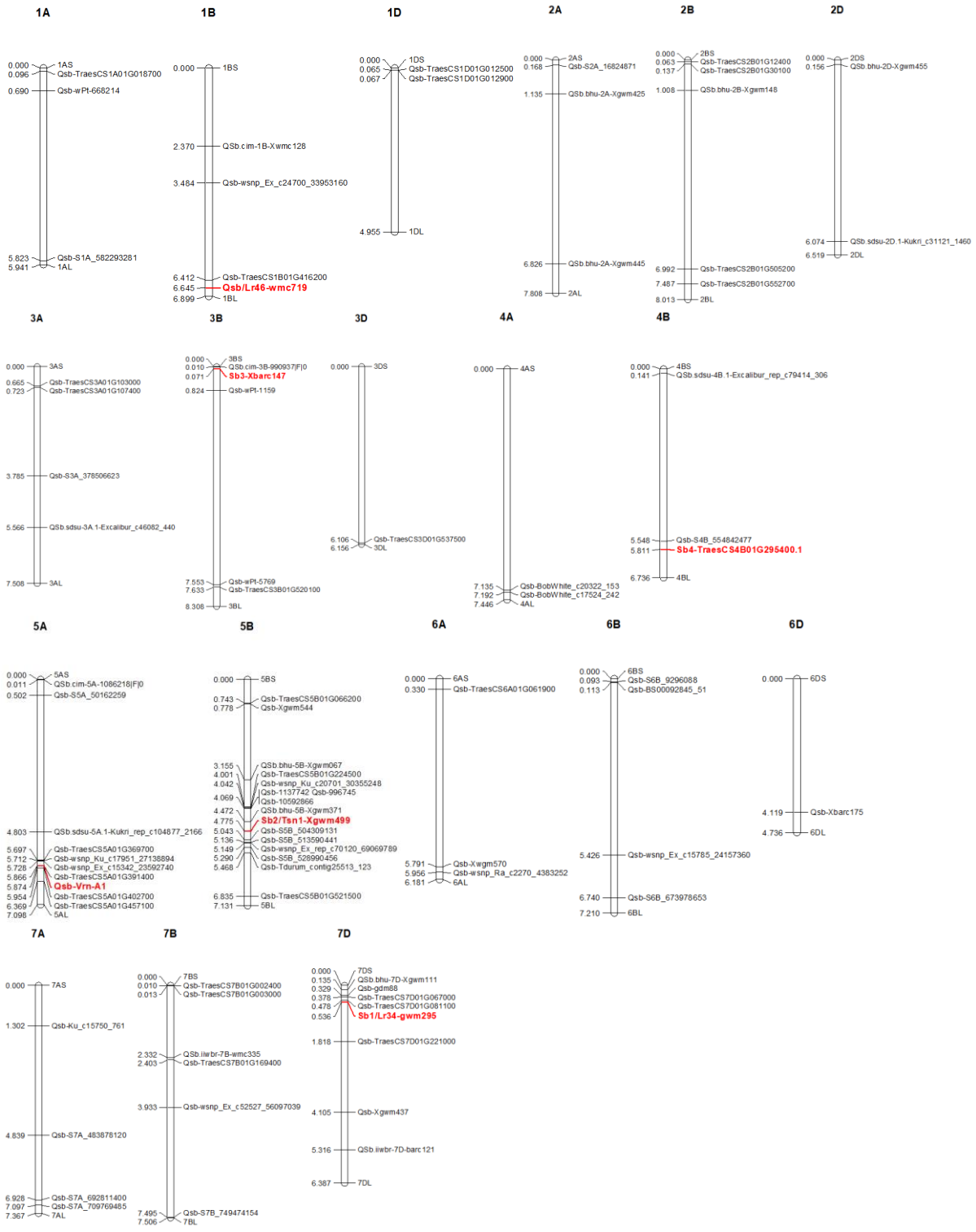

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