Molecular mapping of quantitative trait loci (QTL) for Fusarium head blight (FHB) resistance and agronomic traits in US hard winter wheat

by

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B.S., Shandong Agricultural University, 2010
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AN ABSTRACT OF A DISSERTATION

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Abstract

Fusarium head blight (FHB) is one of the devastating fungal diseases in wheat and results in dramatic losses in grain yield and quality. Use of genetic resistance is an effective approach to FHB control, but highly FHB resistant germplasms usually have many undesirable agronomic traits. To dissect the relationship between FHB resistance and yield component traits, we developed two recombinant inbred line (RIL) populations for identification of native FHB resistance quantitative trait loci (QTL) in US hard winter wheat by genotyping the populations with single nucleotide polymorphism (SNP) markers generated from genotyping-by-sequencing (GBS) and phenotyping the FHB and agronomic traits in both greenhouse and field experiments. In the G97252W x G97380A RIL population, one major native QTL (*QFhb.hwwg-2DS*) was mapped on chromosome arm 2DS for FHB resistance even after removing the confounding effects from heading date (HD) and plant height (HT). *QFhb.hwwg-2DS* coincided with the major QTL for HT, HD, spike length (SL), kernel number per spike (KNS), spikelet number per spike (SNS), thousand grain weight (TGW), and grain size. Additional QTL for spike and grain traits were identified on chromosome arms 2AL, 2DS, 3AL and 4BS. G97252W contributed FHB resistance and high SNS alleles at *QFhb.hwwg-2DS*, as well as high KNS alleles at the QTL on 2AL and 2DS, and high TGW and large grain size alleles at the QTL on 3AL, whereas G97380A contributed high TGW and large grain size alleles at the QTL on 2AL and 2DS, respectively, and the high KNS allele at the 4BS QTL. In the JagR1097 x Jagger RIL population, three QTL for FHB resistance were detected on chromosome arms 4AL, 4DL and 6AL, even after removing the effects from HD and HT. *QFhb-4AL* from Jagger showed a major effect that had 11.80% of the phenotypic variation for FHB resistance and was coincided with the major QTL for HT, HD, SL and SNS. QTL clusters were identified on chromosome arms 2BS, 2DL, 3AS, 3DL, 4BS, 5AS, 5DL and

7AL for different agronomic traits. Jagger contributed FHB resistance and high SNS alleles at *QFhb-4AL* as well as high SNS alleles at the QTL on 5DL. whereas JagR1097 contributed FHB resistance alleles at the QTL on 4DL and 6AL, and high TGW alleles at the QTL on 3AS and 4BS, and the high SNS allele at the QTL on 2BS and 7AL. Pyramiding those FHB resistance QTL with positive alleles for spike and grain traits from different chromosomes may simultaneously improve FHB resistance and grain yield in new cultivars.

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7AL for different agronomic traits. Jagger contributed FHB resistance and high SNS alleles at *QFhb-4AL* as well as high SNS alleles at the QTL on 5DL. whereas JagR1097 contributed FHB resistance alleles at the QTL on 4DL and 6AL, and high TGW alleles at the QTL on 3AS and 4BS, and the high SNS allele at the QTL on 2BS and 7AL. Pyramiding those FHB resistance QTL with positive alleles for spike and grain traits from different chromosomes may simultaneously improve FHB resistance and grain yield in new cultivars.

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Chapter 1 - Literature Review

1.1 Wheat production and improvement

Wheat (*Triticum aestivum*) is the second largest staple crop worldwide based on its planting acreage and total production volume. In 2022, the total global production of wheat was about 778 million metric tons. The top four wheat production region or countries are European Union, China, India, and Russia. The U.S. is the fifth-largest wheat producing country in the world with about 45.7 million acres in 2022 and Kansas was the largest wheat-producing state harvesting 364 million bushels of wheat in 2021 (KSU, 2023 https://www.statista.com/topics/1668/wheat/#topicOverview). The world population is estimated to be nine billion by 2050, therefore the crop production needs to be doubled to meet the huge demand for food from the rapidly growing world population at that time (Ray et al. 2013). During the past decades, the amount of arable land is being reduced due to desertification, soil erosion, salinization, climate change and unsustainable human activities (Godfray et al. 2010). Therefore, the increasing in crop production can only be achieved in the same or even less arable land currently available, which means the crop yield must increase at least 2.4% annually in next two decades. For wheat, 38% of yield increase is needed to achieve this goal. However, the current annual wheat yield increase is only 0.9%, which is far less than the expected 2.4% (Ray et al. 2013). Thus, further genetic gains in wheat yield are mandated to fill up the gap. Many pathogens can cause devastating diseases in wheat and result in dramatic losses in wheat yield and quality (Ning et al. 2017). Genetic resistance is an effective method to control crop diseases, but high levels of resistance are often accompanied with yield penalties (Brown 2002). So, it is urgent to breed novel wheat varieties with high levels of resistance to multiple diseases and yield potential simultaneously. To achieve this goal, dissection of genetic architecture of important agronomic

traits is required to understand their molecular mechanisms underlying trade-offs between disease resistance and yield in order to facilitate their manipulation in wheat breeding (Ning et al. 2017).

1.2 Wheat Fusarium head blight (FHB)

Wheat suffers from FHB, also called scab, in warm and humid wheat growing regions worldwide, which is one of the devastating fungal diseases that reduces both grain yield and quality. In 1982, an FHB epidemic caused approximate 4% of the total wheat yield reduction which is transferred to 100 million bushels of wheat yield loss across the U.S. (McMullen et al. 1997). In Kansas the 2021 FHB epidemic reduced about 3.5% or 13.3 million bushels of wheat (KSU, 2021 https://agriculture.ks.gov/divisions-programs/plant-protect-weed-control/reports-and-publications). More than 17 *Fusarium* species can cause FHB (Parry et al. 1995; Xu and Nicholson 2009; Becher et al. 2013), but the predominant causal *Fusarium* species may vary with biogeographical regions. *F. graminearum* and *F. pseudograminearum* are the major causal pathogens of FHB in Australia (Miedaner et al. 2008; Obanor et al. 2013). *F. culmorum, F. graminearum, F. avenaceum,* and *F. poae* are the pathogen species for wheat FHB in Europe (Parry et al. 1995; Waalwijk et al. 2003; Xu and Nicholson 2009). *F. graminearum* and *F. asiaticum* are major causal pathogens of FHB in Asia (Qu et al. 2007). Among them, *F. graminearum* species complex (FGSC) is the most prominent causal agents in temperate and warm regions of North and South America (Goswami and Kistler 2004; Yerkovich et al. 2020). Many molecules synthesized in pathogen are critical virulence factors for *Fusarium* pathogenicity and aggressiveness. Deoxynivalenol (DON) is of the virulence factors associated with FHB infection and development. It interacts with rRNA residuals in peptidyl transferase centre to inhibit protein synthesis in the eukaryote ribosome (Garreau de Loubresse et al. 2014). The virulence level

diminishes if DON biosynthesis is disrupted and also varies with the trichothecene chemotypes from different Fusarium species (Maier et al. 2006).

1.3 Source of inoculum and life cycle

F. graminearum infects a wide range of small-grain cereals including wheat, barley, rye, oat (Bottalico and Perrone 2002; Leonard and Bushnell 2003). Additionally, FHB pathogens can also be isolated from rice, maize, soybean and weeds (Desjardins et al. 2000; Logrieco et al. 2002; Pereyra and Dill-Macky 2008; Chiotta et al. 2021). Maize, wheat and barley residues on soil surface usually act as the primary source of inoculum that produces ascospores to initiate the FHB infection in fields (Dill-Macky and Jones 2000; Pereyra and Dill-Macky 2008).

F. graminearum initially enters and grows on living tissues in the intercellular spaces without visible symptoms, but subsequently kills the host cells and lives on dead tissues (Ma et al. 2013). *F. graminearum* could survive up to 36 months on crop residues over-wintering and then produce ascospores to start a new disease cycle under favorable environment conditions (Pereyra and Dill-Macky 2008; Ma et al. 2013).

Flowering stage is the most vulnerable time for *F. graminearum* to infect wheat spikes. Airborne spores are dispersed by wind to spikes (Bai and Shaner 2004). There are three successive stages during infection process: initial colonization (surface colonization), main infection (penetration) and final infection (sporulation) stage (Boenisch and Schäfer 2011). Fungus enters host by landing into a cavity of cereal spikelet or attaching to spikelet tissue and then penetrating epidermal cuticle and cell walls to infect spikelets. Spores germinate by forming germ tubes and dense hyphae within 6-24 hours after inoculation (hai) when temperature and moisture are favorable. Within 24-36 hai, fungus generates short infection hyphae indicating direct penetration. From 1 to 7 days after inoculation, hyphal networks are subsequently formed on caryopses, paleas,

lemma, and then grow inter- and intracellularly into glumes, rachis. In rachis, hyphae spread into adjacent spikelets by growing upward and downward through vascular bundles and cortical parenchyma tissues (Mary Wanjiru et al. 2002; Boenisch and Schäfer 2011). During the main infection stage, runner hyphae form complex branches and produce lobate appressoria, foot structures and large infection cushions. Hyphae produce trichothecenes within infected tissues and disease symptoms develops rapidly in the infected spikes. FHB pathogens secrete cell wall-degrading enzymes leading to the degradation of cytoplasm and host cells at this stage. In the early infection stage, the glumes in infected spikelets show dark-brown and water-soaked spots, gradually, entire spikelet becomes blighted in the susceptible cultivars (Yoshida et al. 2007; Boenisch and Schäfer 2011). In the final infection stage, aerial hyphae and sporodochia are formed. After 16-18 days of inoculation, the hyphae cover substomatal cavities without growing through stomata. The association between pathogen and stomata and silica cells induces the sexual development. Perithecial initials act as overwintering structures finally. The husk tissues get entirely necrotic and chlorotic with the entire spike becoming blighted. Infected heads are unable to produce grain or only shriveled grain with structurally damaged starch and proteins (Boenisch and Schäfer 2011; Becher et al. 2013).

1.4 FHB management

FHB epidemics occurred when susceptible varieties, FHB pathogens and warm and wet environments are available. The incidence and severity of FHB are strongly influenced by environments and variety resistance levels. It’s hard to control environment factors or pathogen populations to prevent disease epidemics. Most management strategies aim to disrupt the disease cycle or minimize disease severity and mycotoxin contamination in grains (Gilbert and Tekauz 2011). These strategies use host resistance, chemical control, biological control, or integrated

agronomic practices such as crop rotation and deep plowing to reduce the amount of fungus-infected residue on the soil surface and decrease the amount of primary inoculum overwintered from those residues. Research shows that soybean-wheat rotation has the lower FHB incidence and mycotoxin contamination than maize-wheat or wheat-wheat rotation (Dill-Macky 2008). It is very important to grow non-host species in crop rotation to break the disease cycle. Maize and susceptible cereals should be avoided to be the pre-crop for wheat. Conservation tillage or no-tillage practices is widely spread and applied worldwide to reduce soil erosion, increase organic nutrient concentration and yield, but leaves a large amount of crop residues unburied on the soil surface (Lori et al. 2009). Crop residues is a major reservoir for pathogen over-winter to produce inoculum in the following spring season. Tillage operation reduced FHB incidence and DON content by 80% and 45%, respectively, relative to conservation tillage (Alföldi et al. 2000). Fungicide is to use chemicals to control FHB and DON. Demethylation inhibitor (DMI) class is the most effective fungicide which is widely applied for FHB and DON control in cereal crops. DMI fungicides can increase yield and test weight by 13.8% to 15%, respectively (Paul et al. 2008, 2010). However, the efficacy of fungicides can be varied when applied under different field conditions. Fungicide alone is often insufficient for controlling FHB and mycotoxin contamination to desirable levels in infected cereal grains (Paul et al. 2007). Fungicides are usually more effective when applied between start of flowering to one week after anthesis (D'Angelo et al. 2014). However, the uneven flowering time and unfavorable weather conditions make it very difficult for timely application (Freije and Wise 2015). Another challenge is the emerging fungicide resistance in *F. graminearum* due to the over-use of DMI fungicides, as discovered in New York state in 2014 (Spolti et al. 2014). Biological control has been extensively investigated for the management of FHB and DON contamination in cereals. Several fungal and bacterial antagonists have been

identified as candidate biocontrol agents against *Fusarium* species to reduce FHB severity and mycotoxin production in grains, such as *Cryptococcus spp*., *Clonostachys spp*., *Bacillus spp*., *Pseudomonas spp.*, and *Steptomyces spp.* (Legrand et al. 2017). However, biological control faces the similar challenge and limitations as fungicides and there is no approved and commercialized biocontrol product in the market (Becher et al. 2013; Legrand et al. 2017). To date, the use of integrated management strategies combined with host plant resistance show the highest potential to control FHB (Shude et al. 2020).

1.5 Host plant resistance types and disease assessment

Genetic resistance is the most effective and stable way to control FHB epidemics. Host resistance to FHB is a quantitative trait and controlled by multiple genes. Species or race-specific host resistance against FHB and *Fusarium* species has not been found to date (van Eeuwijk et al. 1995). FHB resistance can be classified as active or passive patterns. Active resistance activates inside host plant defense mechanisms to suppress pathogen growth and spread after the initial infection (Mesterházy et al. 1999). Passive resistance is also called avoidance because of morphological features establishing unfavorable minor-environment for initial infection around spikes and make host plant to escape FHB disease development (Rudd et al. 2001; Gilsinger et al. 2005). Many morphological traits are associated with FHB resistance including plant height, heading date, anther extrusion, grain filling rate, spike compactness, awn length, peduncle length (Miedaner 1997; Rudd et al. 2001; Buerstmayr et al. 2020). To date, there are five types of active resistance have been described in wheat. Type I describes resistance to initial infection or penetration and is estimated by counting the number of spikelets showing primary infection (Mesterházy et al. 1999). Type II refers to resistance to spread of FHB symptoms within an infected head and is scored by the percentage of symptomatic spikelets per spike (PSS) (Mesterházy et al.

1999). Type III is the resistance to DON accumulation in infected kernels, which is evaluated by DON content in infected kernels (Miller et al. 1985; Mesterházy et al. 1999). Type IV involves resistance to kernel infection, also called *Fusarium* damage kernels (FDK). Type V is FHB tolerance as reflected by relative yield decline (Mesterhazy 1995; Mesterházy et al. 1999; Rudd et al. 2001; Fakhfakh et al. 2011). Three inoculation methods were used for FHB disease assessment in greenhouse and field: point or single floret inoculation, spray spore inoculation, and grain spawn inoculation. Point inoculation is typically applied to estimate type II resistance in greenhouse. Inoculum is delivered into a single central floret at flowering stage. PSS is recorded at 14 to 21 days after inoculation and used as variables to estimate type II resistance (Rudd et al. 2001). Spray spore inoculation is usually used to screen large amounts of breeding materials in a field. The conidial suspension is sprayed over flowered wheat spikes. FHB incidence, FHB severity, FHB disease index are collected at 10 to 21 days after inoculation and used as variables for resistance evaluation (Rudd et al. 2001). Generally, FHB incidence measures Type I resistance and FHB severity measures type II resistance in a field FHB trial (Rudd et al. 2001). Grain spawn is an alternative to spray inoculation. The grain spawn is made from infected wheat or corn kernels and spread in a field at boot stage of wheat growth with a second application two week later. The fungus develops in those infected kernels to form perithecia and ascospores released from the perithecia as initial inoculum to infect plants. Resistance assessment is similar to spray inoculation. Moreover, the percentage of infected spikelets per plot is visually scored to measure FHB severity (Buerstmayr et al. 2008). Each row can be harvested and threshed and the grains are used for FDK and DON measurement (Rudd et al. 2001).

1.6 Quantitative trait loci (QTL) for FHB resistance in wheat

FHB resistance inheritance is controlled by multiple genes with major or minor effects, and a continuous variation in resistance levels was observed in segregating populations. However, the complete immunity to *Fusarium* pathogens have not been discovered in wheat yet. In order to understand the molecular mechanism of resistance, it must dissect the genetic architecture of FHB resistance. Up to date, more than 500 FHB resistant QTL have been mapped on all the 21 chromosomes in wheat (Buerstmayr et al. 2020). A small portion of these QTL showed major and stable effects on resistance to FHB in diverse genetic backgrounds and environments. Eight major QTL from *Fhb1* to *Fhb8* have been officially reported and named. *QFhs.ndsu-3BS* was named as *Fhb1*, the first mapped QTL, on chromosome 3BS derived from Chinese cultivar 'Sumai 3'. *Fhb1* shows the largest effect among the QTL identified to date and explained up to 60% of the genetic variation for type II resistance (Bai et al. 1999a; Waldron et al. 1999). *Fhb2* was detected on chromosome 6BS flanked by *Xgwm133* and *Xgwm644* in 'Sumai 3' and explained 21% of the genetic variation of type II resistance (Yang et al. 2003; Cuthbert et al. 2007). Jia et al. (2018) finely mapped *Fhb2* into a 2.2 cM interval between marker *Xwgrb688* and *Xmag3017* in 'Wangshuibai'. *Fhb3* for type II resistance was transferred from alien species *Leymus racemosus* into wheat with a Robertsonian translocation T7AL.7Lr#1S on chromosome 7AL associated with three markers, *BE586744-STS*, *BE404728-STS* and *BE586111-STS* in several wheat-Leymus introgression lines (Qi et al. 2008). The *Qfhi.nau-4B* locus for type I resistance was named as *Fhb4* and mapped in a 1.7 cM interval between *Xhbg226* and *Xgwm149* on chromosome 4BL in 'Wangshuibai' (Xue et al. 2010). Jia et al. (2018) confined *Fhb4* into a 0.14 cM region flanked by *Xmag8990* and *Xmag8894*. The *Qfhi.nau-5A* locus for type I resistance was designated as *Fhb5* and detected in a 0.3 cM region flanked by *Xgwm304* and *Xgwm415* on chromosome 5AS in

'Wangshuibai', which was reported to reduce about 55% infection in resistant NILs (Xue et al. 2011). Jia et al. (2018) confined *Fhb5* into a 0.09 cM region flanked by *Xwgrb0222* and *Xwgrb1621*. *Fhb6* for type II resistance was transferred from *Elymus tsukushiensis* with a 1Ets#1S segment into the subterminal region of 1AS of bread wheat, which reduced about 28% of the FHB severity in homozygous resistant lines. Three cleaved amplified polymorphic sequence (CAPS) markers (*tplb0017E15*, *tplb0029J02* and *AK357509*) and one kompetitive allele specific polymerase chain reaction (KASP) marker (*wg1S_snp1*) were reported to tag *Fhb6* (Cainong et al. 2015). *Fhb7* for type II and type IV resistance was transferred from *Thinopyrum ponticum* into common wheat in a 7DS.7el2L Robertsonian translocation. *Fhb7* was identified in a region flanked by markers *XsdauK66* and *Xcfa2240*, explaining up to 32.5% of phenotypic variances for FHB resistance (Guo et al. 2015). *Fhb8* was detected for FDK and confined into a 1cM interval between *Xwgrb1500* and *Xwgrb1559* on wheat chromosome arm 7DL in wangshuibai (Wang et al. 2023).

Other several major QTL have also been finely mapped in recent studies. *Qfhs.ndsu-3AS* derived from *T.dicoccoides* was mapped into a 5.2 cM interval flanked by *Xwgc501* and *Xwgc510* on chromosome 3A in a durum wheat 'LDN', explained about 37% of the phenotypic variation for FHB resistance (Otto et al. 2002; Zhu et al. 2016). *QFhb.nau-2B* was mapped on chromosome 2B in common wheat cultivar 'Nanda 2419'. It was delimited to a 4.1 cM region between *Xwgrb1503* and *Xwgrb1373* for type II resistance and a 0.9 cM interval flanked by *Xwgrb1561* and *Xwgrb1410* for type I resistance (Li et al. 2019a).

1.7 Map-based cloning of FHB resistance genes in wheat

Up to date, three FHB resistance genes have been cloned from wheat via positional cloning strategy. These resistance genes respond to FHB infection through different mechanisms. *Fhb1* confers durable and stable type II resistance to FHB in wheat. Firstly, Rawat et al. identified a

pore-forming toxin-like (*PFT*) gene encoding a chimeric lectin protein as the candidate for *Fhb1* in a Chinese variety 'Sumai 3' (Rawat et al. 2016). However, *PFT* was also detected in many susceptible cultivars and some recombinant lines with the *PFT* allele alone showed high susceptibility to FHB, which indicates that *PFT* may not be *Fhb1* (Jia et al. 2018; He et al. 2018). Later, a histidine-rich calcium-binding-protein gene (*TaHRC*) was reported as the *Fhb1* candidate gene cloned from two Chinese varieties 'Ning7840' and 'Wangshuibai' by two independent studies in 2019, respectively (Li et al. 2019b; Su et al. 2019). Su et al. (2019) reported that *TaHRC* encodes a nuclear protein mediating susceptibility to FHB and a large sequence deletion in the start codon region of *TaHRC* results in FHB resistance due to the loss-of-function mutation. However, Li et al. (2019b) reported that the large deletion in *TaHRC* results in FHB resistance due to gain-of-function via generating a new start codon in the upstream region and translating into a new protein product. Recently, Chen et al. (2022) confirmed *TaHRC* as an FHB susceptibility gene by using a Barley stripe mosaic virus (BSMV) mediated gene editing approach and investigated the mechanism of *TaHRC* in triggering FHB susceptibility in wheat. They found that TaHRC interacts with a cation exchanger (CAX)-interacting protein 4 (TaCAXIP4) in the nuclei to suppress the calcium-mediated plant immune responses to facilitate susceptibility during FHB infection.

More recently, *Fhb7* has been cloned and predicted to encode a glutathione S-transferase (GST) that detoxifies pathogen-produced mycotoxins by conjugating a glutathione (GSH) unit onto the epoxide moieties of trichothecenes (Wang et al. 2020). *Fhb7* confers durable and stable FHB resistance through a different mechanism from *Fhb1* and has been transferred into several Chinese wheat cultivars without adverse effects on growth and yield potential.

QFhb.mgb-2A QTL is another resistance gene that has been map-based cloned from durum wheat (Gadaleta et al. 2019). It confers type I and II resistance to FHB and was firstly mapped on chromosome 2AS in a durum wheat RIL population derived from the cross between Sumai 3 and an FHB susceptible tetraploid wheat cultivar 'Saragolla' (Giancaspro et al. 2016). A wall-associated receptor-like kinase (*WAK2*) gene was isolated as the causal gene of *QFhb.mgb-2A* (Gadaleta et al. 2019). Guo et al. (2021) confirmed the function of *TaWAK2A-800* (a wheat wall associated kinase gene) as a positive regulator triggering wheat resistance to FHB infection presumably through chitin-induced pathway.

1.8 FHB Resistance Germplasm and Breeding

Variation in FHB resistance has been extensively observed among wheat genotypes. To date, approximately 7000 accessions of wheat and wheat relatives have been reported worldwide to show some degrees of FHB resistance (Ma et al. 2020). The exotic, alien and native resistance have been reported and were applied in breeding and FHB resistance improvement in crops.

1.8.1 Resistant sources in other countries relative to US.

Asian wheat varieties provide important genetic resistant resources. More than 60 Asian landraces including Wangshuibai, 'Haiyanzhong', 'Tanwanxiaomai', 'Huangcandou', 'Huangfangzhu', 'Fanshanxiaomai', 'Pinghuijianzimai', 'Baishanyuehuang', 'Nobeokabozu', 'NyuBai', 'Shinchunaga', 'Chokwang' have been reported to possess moderate or high FHB resistance (Yu et al. 2006; Li et al. 2016). Two Italian wheat cultivars, 'Mentana' and 'Funo', were introduced to China in the middle of the last century. Many resistant cultivars such as 'Nanda2419', 'Wannian 2', 'Wangmai 15', 'Emai 6', 'Wumai 1', 'Ewusan 3', 'Jingzhou 1', 'Jingzhou 47', 'Jingzhou 66', 'Yangmai' series cultivar, Sumai 3 were then released with the two Italian germplasm in their pedigree via pure-line breeding or crossing breeding (Zhu et al. 2019).

Sumai 3 (Funo/Taiwanxiaomai) is a Chinese cultivar released in 1970, which is the well-known resistance donor carrying Fhb1 (Bai et al. 2018). It has been widely applied in wheat breeding programs all over the world. Using Sumai 3 as a resistant parent, many important resistant cultivars such as 'Ningmai9', 'CM82036', Ning7840 were developed and then were used as new *Fhb1* donors in China. The new *Fhb1* donors display improved agronomic characters and adaptability (Bai et al. 2018; Zhu et al. 2019). Japan and Korea germplasm were also identified with high FHB resistance and widely applied in wheat breeding programs, such as 'Shinchunaga', 'Nobeokabouzu', 'Nyu Bai', 'Chokwang' (Ban 2000). Phylogenetic analysis indicates that *Fhb1* resistance allele can also be detected in Japanese cultivar 'Norin 129' (Niwa et al. 2018). European germplasm was considered as moderately resistant to FHB and inferior to Sumai 3. Certain European resistant resources were discovered in tetraploid wheat from Tunisia, Syria, Israel and Turkey (Buerstmayr et al. 2003; Huhn et al. 2012; Talas et al. 2012). Some resistant cultivars were released in Europe, such as 'Soissons', 'Spark', 'Vector', 'Fundulea', 'Renan', 'Dream', 'Petrus', 'Toras', 'Soliater', 'Arina', 'Apache', 'Arche' (Gosman et al. 2007; Buerstmayr et al. 2008; Becher et al. 2013).

There are plenty of genetic resistance variation in south America. In Argentina, some old germplasm, such as 'Klein Sin Rival', 'Klein Vencedor', 'Ardito', played a foundation role in improvement of FHB resistance and development of some moderately resistant cultivars including '38MA', 'Klein47', 'Klein Sinmarq', 'Klein Otto Wulf', 'Klein 66', 'Vencelel MA', 'Magnif Entreriano'. All above varieties were crossed with exotic or native germplasm to generate new commercial varieties, such as 'Pergamino Gaboto', 'Oncativo INTA', 'Tezanos Pintos Precoz', 'Vilela Sol', 'Klein Atlas' (https://link.springer.com/book/10.1007/978-94-007-7091-1) (Alconada Magliano and Chulze 2013). Argentina germplasm plays an important role in FHB

resistance improvement in Uruguay. Argentina variety '38MA' and Uruguay local cultivar 'Pelon 33C' contributed to the development of 'Litoral' varieties in 1930s and 'Estanzuela Dakuru' in 1960 (https://link.springer.com/book/10.1007/978-94-007-7091-1) (Alconada Magliano and Chulze 2013). Another important worldwide renowned resistant variety, 'Frontana' (Fronteira/Mentana) was developed by Brazilian breeders in 1943, which shows good resistance in field and also provide adult plant resistance to leaf rust and pre-harvest sprouting. Frontana and its derivatives are excellent source of stable resistance for more than 50 years.

CIMMYT varieties suffers serious FHB infections due to its semi-dwarf characters. In the early 1980s, CIMMYT started introduction of Chinese resistant germplasm into South America through international shuttle breeding program. Sumai 3 and its derivatives were used to improve the FHB resistance of semi-dwarf varieties. Some advanced lines with Sumai 3 intheir pedigrees showed good agronomic characters and FHB resistance, such as '6SRSN22', '7SRSN 05' (https://link.springer.com/book/10.1007/978-94-007-7091-1) (Alconada Magliano and Chulze 2013). 'Catbird' is an excellent variety developed by CIMMYT showing high yield potential and high FHB resistance without 'Sumai 3' in its pedigree and has been widely used as a parent for wheat breeding in CYMMIT breeding programs (https://link.springer.com/book/10.1007/978-94-007-7091-1) (Alconada Magliano and Chulze 2013). In Canada, some cultivars in Manitoba have'Sumai 3' in their pedigree, such as 'AAC Brandon', 'AC Carberry', 'Cardale', 'CDC VR', 'AAC Elie'. These cultivars possess much better FHB resistance than older varieties (Gilbert and Tekauz 2000).

1.8.2 Alien resistance sources

Wheat alien species are important genetic resources for FHB resistance. The short arm of *L. racemosus* chromosome 7Lr#1 carries *Fhb3* resistance allele and was introgressed into wheat

via a Robertsonian translocation T7AL.7Lr#1S (Qi et al. 2008). *Fhb6* locus was traced back to *E. tsukushiensis* and located on the chromosome 1Ets#1S. It has a major effect and reduced FHB severity up to 28% in the translocation lines (Cainong et al. 2015). The distal region of 7el2 long arm of *Thinopyrum ponticum* carries a major effect gene *Fhb7* and this gene was introgressed into wheat by US breeding program at Purdue University (Guo et al. 2015). Song et al. (2023b) identified a novel FHB resistance locus *FhbRc1* on the long arm of 7Sc in an alien translocation line of wheat derived from *Roegneria ciliaris (Trin.) Nevski*. More than 100 alien species fragments have also been extensively investigated and integrated into wheat breeding programs, such as *Roegneria kamoji, R. ciliaris, Th. Elongatum, Th. junceum, Th. intermedium, Elytrigia intermedia* (Oliver et al. 2005). The wheat close relatives such as *T. tauschii, T. spelta, T. macha, T. timopheevii, T. dicoccoides* also show high variation in FHB resistance (Ghimire et al. 2020). These alien sources can be incorporated into wheat varieties using cytogenetic coupled with back-crossing strategies to widen the resistance genetic diversity in wheat by developing alien fragment translocation, substitution, addition and recombination lines (Oliver et al. 2005; Bai et al. 2018).

1.8.3 Resistance sources in the USA

Frontana was the first exotic germplasm used as a parent to improve FHB resistance in the U.S., such as ‘Willet’ is the first commercial cultivar with Frontana in its pedigree (Zhu et al. 2019). Sumai 3 and other Asian cultivars and landraces have been then incorporated into wheat breeding programs in the US since 1990s. To date, more than 20 hard red spring wheat cultivars (Brick, Prevail, Focus) with the pedigrees of Sumai 3 have been released for production in Minnesota, North Dakota and South Dakota (Steiner et al. 2017; Zhu et al. 2019). Private seed companies have also contributed to development of commercial varieties by incorporating exotic sources into native backgrounds to improve FHB resistance, and ‘25R18’, ‘25R42’, ‘Impervo’,

‘Bigg Red’, ‘Freyr’, ‘25R51’, ‘Kelby Kuntz’, ‘SY Soren’, ‘SY Ingmar’ are the cultivars carrying resistance genes from Asian sources (Steiner et al. 2017; Zhu et al. 2019). Native resistant germplasm can be easier applied in wheat breeding than exotic sources due to their better agronomic performance and quality characters as well as extensive adaptability to local environments. After screened a set of SRWW cultivars from Eastern and Southern region of the U.S., ‘Jamestown’, ‘Massey’, ‘COKER 9474’, ‘COKER 9511’, ‘Foster’, ‘Patton’, ‘McCormick’, ‘Goldfield’, ‘Freedom’, ‘INW0411’, ‘INW0304’, ‘NC-Neuse’, ‘il94-1653’, ‘Cecil’, ‘Tribute’, ‘Roane’, ‘USG 3555’, ‘Ernie’, ‘Truman’, ‘Bes’s, ‘ny88046-8138’, ‘WestBred X00-1079’ were found to carry FHB resistance genes (Bai et al. 2018; Ghimire et al. 2020). Several HRWW varieties from the Great Plains were reported to have moderate FHB resistance including ‘Wesley’, ‘Hondo’, ‘Everest’, ‘Heyne’, ‘Lyman’, and ‘Overland’ (Bai et al. 2018). *Fhb1* was cloned and has been transferred to different HRWW backgrounds to facilitate the application of this major effect gene in HRWW breeding programs (Bai et al. 2018). *Fhb7* is another promising resistance gene for wheat breeding, which has not been extensively utilized. The diagnostic markers have been developed in order to deploy *Fhb7* in the U.S. wheat breeding programs (Zhao et al. 2022).

1.9 Relationships between FHB resistance and developmental traits.

Plant disease resistance genes or QTL usually interact antagonistically with genes regulating plant growth and development (Ning et al. 2017). Some developmental traits including plant height (HT), heading date (HD) and flowering time (FT) have been reported to be associated with FHB resistance. Previous mapping studies discovered that some developmental QTL coincided with FHB resistance QTL. Buerstmayr et al (2011) and Chu et al (2011) independently reported a QTL for FHB resistance, FDK and DON on 5AL chromosome coincided with the *Q*

gene, a domestication gene for free-thresh, which was also associated with plant height and flowering time. The *q* allele contributes positive effect to FHB resistance at this locus.

McCartney et al (2016) and Xu et al. (2020) reported one QTL for FHB resistance on 2DS chromosome flanked by *Xgwm261* and *AX-111561744*, which overlapped with the *Rht8* semi-dwarfing locus. The *Rht8* semi-dwarfing allele contributes to increased FHB susceptibility and decreased plant height. McCartney et al (2016) and Liu et al (2013) reported one QTL for FHB resistance on 2DS chromosome flanked by *Xgwm484*, which overlapped with QTL for HD and plant HT. Further analysis found that this QTL region also contains *Ppd-D1*, and the photoperiod sensitive allele *Ppd-D1b* was associated with increased FHB resistance, tall plant HT and long HD, thus *Ppd-D1b* might has pleiotropic effects on these traits (Liu et al. 2013; McCartney et al. 2016).

In some previous studies, one FHB resistance QTL on 4B chromosome was mapped in a region including a plant height QTL at the *Rht-B1* locus with the tall allele *Rht-B1a* associated with increased FHB resistance (Buerstmayr et al. 2012; Lu et al. 2013; Liu et al. 2013; Prat et al. 2017), indicating that the dwarfing allele *Rht-B1b* may contribute reduced plant HT and increased FHB susceptibility. However, Srinivasachary et al (2009) reported *Rht-B1b* showed decreased Type I resistance to FHB, but increased type II resistance to FHB. Other studies reported that plant HT showed significantly positive correlations with FHB resistance. The semi-dwarf allele *Rht-D1b* displayed pleiotropy in reducing plant HT and decreasing Type I resistance to FHB, but had no significant influence on type II resistance (Srinivasachary et al. 2009; Liu et al. 2013; He et al. 2016).

One QTL for FHB resistance was associated with plant HT and HD at *Vrn-A1* locus on 5AL chromosome (He et al. 2016). Another QTL was detected for FHB resistance on 5B

chromosome in ‘AGS 2000’ and coincided with *Vrn-B1* locus, in which FHB resistance was associated with short vernalization (Petersen et al. 2016).

Some of developmental traits may result in passive FHB resistance by creating micro-climate to influence the initiation of pathogen infection and spread under field conditions (Buerstmayr et al. 2009). However, not all the plant HT QTL are coincident with FHB resistance QTL, implying that the correlation between developmental traits and FHB resistance may not be simply resulted from disease escape (Buerstmayr et al. 2020). Some pleiotropic or tightly lined genes may be responsible for the association between FHB resistance and these developmental traits (Buerstmayr et al. 2020). Identification of causal resistance gene via positional cloning will facilitate clarification of the genetic relationships between FHB resistance and developmental traits.

1.10 Relationship between FHB resistance and yield component traits

Some researchers reported that spikelet number per spike (SNS) had a negative correlation with FHB susceptibility. Tessmann and Van (2019) found that SNS had a negative correlation with FHB severity and FDK except for FHB incidence and DON accumulation. Photoperiod sensitive allele *Ppd-D1b* displayed a positive effect to increase SNS and reduce FHB susceptibility. Lv et al. (2014) mapped one QTL on 5D chromosome for reduced FHB susceptibility and increased SNS, which coincided with vernalization gene *Vrn-D1*. However, there was study reported that SNS was positively related to FHB susceptibility due to G x E interaction under different environments. Chen et al. (2021) reported a QTL *QFhb-hnau.2DL* from ‘Yangmai 13’ contributing to the type I and type II resistance and reducing FHB severity in the natural infection environments but showed negative effect on SNS.

Previous studies reported that FHB resistance was associated with kernel number per spike (KNS). One QTL was mapped on chromosome 4B coinciding with the *Rht-B1* locus, which contributes to the increased FHB resistance, but decreased KNS. Another QTL was discovered on chromosome 5D co-locating with *Vrn-D1* locus, which showed effects on increasing FHB resistance and KNS (Lv et al. 2014). Hu et al. (2023) mapped one FHB resistance QTL on chromosome 2DL, which coincided with QTL for KNS while contributing to increased FHB resistance and KNS.

Some researchers investigated the relationship between major FHB resistance QTL and thousand kernel weight (TGW), but did not find any significant associations (Salameh et al. 2011; Li et al. 2019c; Zhang et al. 2021). Li et al (2019c) investigated the relationship between *Fhb1* and agronomic traits, the results indicated that there was no significant association between *Fhb1* and agronomic traits, such as kernel weight per spike and TGW. Researchers pyramided *Fhb1* and *Qfhs.ifa-5A* QTL into nine European winter wheat varieties by marker-assisted backcrossing. These two QTL significantly increased FHB resistance on these varieties without negative effects on TGW and grain yield (Salameh et al. 2011). *Fhb1*, *Fhb4* and *Fhb5* were simultaneously introgressed into several Chinese cultivars or breeding lines. All the introgression lines displayed significantly increased type I resistance and type II resistance while had no negative effect on TGW and yield (Zhang et al. 2021). However, some reports indicated that FHB resistance QTL reduced TGW. Hu et al. (2023) reported a QTL for FHB resistance on 2DL chromosome decreased TGW. Otherwise, other FHB resistance QTL showed positive effect on increasing TGW. Suzuki et al. (2012) reported that the 'Sumai 3' resistance allele at 4BS QTL associated with increased TGW. One QTL was mapped on chromosome 4B coinciding with the *Rht-B1* locus, which contributes to the increased FHB resistance and TGW (Lv et al. 2014).

Only several studies reported association between grain traits and FHB susceptibility. Castor (1980) reported that grain size and percentage of normal grains were significantly reduced in FHB infected panicles than normal panicles. An average of 23.5% of the grains from FHB panicles were reduced in size compared with only 4.6% of the grains from normal panicles. Jung et al. (2010) investigated the relationship between grain traits and FHB severity. They found that grain length (GL) showed a positive correlation with FHB susceptibility of PSS (Type II). Gong et al. (2020) discovered that there was significant change in grain width (GW) of the tested lines at the same developmental stages after FHB infection. However, there was no significant differences for GL under the impact of FHB infection. The newly developed substitution line DM96 was derived from a distant hybridization between M842-16 (an octoploid Tritileymus line) and D4286 (a Triticum durum line), which displayed increased resistance to FHB, longer GL and larger grain area (GA) (Zhao et al. 2019). However, the relationship between FHB resistance and grain traits have not been investigated deeply yet.

The immunity-related genes usually have influences on plant growth and grain yield by reducing the production of vegetative biomass (Ning et al. 2017). Yield components are usually useful variables to investigate the responses of different genotypes to FHB infection. The understanding of genetic relationships among FHB resistance and yield component traits makes a solid foundation for simultaneous improvement of FHB resistance and grain yield through wheat breeding.

Chapter 2 - Genetic architecture of QTL for FHB resistance and agronomic traits in a hard winter wheat population

2.1 Introduction

Wheat (*Triticum aestivum L.*) is an important cereal crop for human nutrition supply in the world. Continuous increase in wheat productivity is critical to meet the growing demand from a rapidly rising world population. Wheat FHB, mainly caused by *F. graminearum*, is a devastating disease that reduces not only grain yield but also grain quality, and therefore threatens global wheat production (Buerstmayr et al. 2009). Mycotoxins such as DON produced by the fungus during infection are detrimental to humans and livestock when the contaminated grain is used as food and feed (McMullen et al. 2012).

Growing resistant cultivars is one of the most effective strategies to reduce FHB damage. Wheat FHB resistance can be active, passive, or both (Buerstmayr et al. 2020). Active resistance is usually expressed physiologically or biochemically by activating internal host plant defense mechanisms to suppress pathogen growth and limit the spread of FHB symptoms within wheat spike tissues after initial infection (Mesterházy et al. 1999); however, passive resistance is mainly expressed as disease avoidance due to certain morphological traits that create favorable micro-environments to avoid or reduce fungal initial infection, resulting in low FHB infection in host plants (Mesterhazy 1995). Several morphological and developmental traits including plant HT, HD, anther extrusion, and spike compactness (SC) have been discovered associated with plant reactions to FHB (Miedaner 1997; Rudd et al. 2001; Gilsinger et al. 2005; Buerstmayr et al. 2020). In general, passive FHB avoidance due to morphological features is usually more vulnerable to changes in testing environments than active resistance.

Based on FHB infection, DON content, disease progress in wheat spikes and kernels, and grain yield losses, wheat FHB resistance has also been described as five types (Mesterházy et al. 1999). Type I is resistance to fungal initial infection. Type II is the resistance to spread of FHB symptoms within an infected spike (Schroeder et al. 1963). Miller et al. (1985) described Type III resistance as resistance to DON accumulation in infected kernels. Later, Type IV resistance was proposed as low FDK and Type V resistance as low yield loss or FHB tolerance (Mesterhazy 1995). To date, more than 50 QTL for Types I, II and III resistance have been reported on all 21 wheat chromosomes from various resistant sources (Bai et al. 2018), and some of them have been frequently associated with undesired developmental and yield traits (Buerstmayr et al. 2020). However, the genetic relationships between these traits and FHB resistance have not been well characterized. Unveiling the genetic relationships among these traits will provide useful guidelines for selecting wheat cultivars with not only a high level of FHB resistance but also desirable agronomic traits for high yield potential in wheat breeding programs.

Wheat KNS, SNS, and TGW are major grain yield components and have higher heritability than grain yield per se; thus, it is more effective to assess grain yield components, which will increase statistical power for detecting QTL for grain yield (Zhang et al. 2018a). The objectives of this study are to identify QTL for FHB resistance and related yield-related traits using a recombinant inbred line (RIL) population and to characterize the relationships among the QTL for those traits.

2.2 Materials and methods

2.2.1 Plant materials

A population of 132 F6:8 RILs was developed by single seed descent from a cross between two winter wheat lines G97252W and G97380A from Goertzen Seed Research, Inc in KS. The

cross was initially made at Oklahoma State University in the mid-2000s to map *Rht8*. G97252W was found to be moderately FHB resistant, whereas G97380A was highly FHB susceptible. These two wheat lines from Kansas showed significant differences (Table 2.1) in plant HT, HD, SC, KNS, SNS, TGW, SL, GW, GL, and GA.

2.2.2 Evaluation of FHB and agronomic traits in greenhouses.

Two parents and all the RILs were evaluated for type II FHB resistance in four greenhouse experiments in 2019 spring (FHB_GH2019S), fall (FHB_GH2019F) and winter (FHB_GH2019W), and 2020 spring (FHB_GH2020S) at Kansas State University using a randomized complete block design with two replications. Wheat seedlings were vernalized at 6 °C for 50 d and then were transplanted into 14 x 14 cm Dura pots containing Metro-Mix 360 soil mix (Hummert International, Earth City, MO). The greenhouse temperatures were set at 12 ± 2 °C for daytime and 15 ± 3 °C for night during the seedling stage and changed to 25 ± 3 °C (at day) and 20 ± 3 °C (night) three weeks after transplanting. The daylength was set for 12 h with supplemental light. Five plants per line were transplanted into each pot (replication) and fertilized with Miracle-Gro® (The Scotts Miracle-Gro Company, Marysville, OH) weekly for four weeks. A conidial spore suspension of *F. graminearum* was prepared by culturing the *F. graminearum* strain GZ3639 from Kansas in mungbean broth (Bai et al. 2000). The final inoculum concentration was adjusted to about 100,000 conidiospores mL-1 by counting them in a microscope. At the flowering stage, a 10-uL conidial suspension (1000 conidia/spike) was injected into a central spikelet of a spike using a syringe (Hamilton, Reno, NV). Five spikes were inoculated in each pot and moved into a moist chamber at 100% relative humidity and 20-25 °C to initiate fungal infection. After 48 h of incubation, the plants were moved back to the greenhouse benches for disease development. The number of infected spikelets and total number of spikelets per inoculated spike were determined

for each plant at 16 d after inoculation. FHB severity was estimated using PSS for QTL analysis. In all trials, plant HT was measured from the ground to the top of the spike of the main stem excluding awns before harvesting. HD was recorded when 50% of plants had 50% spikes emerging from the flag leaf sheaths (Feekes 10.1). SL was measured from the base to the top of a spike excluding awns and SNS was counted before harvest. SC was calculated by dividing the SNS by SL.

The RIL population was separately evaluated for KNS, TGW, GW, GL, and GA in two greenhouse experiments in spring (Yld_GH2015S) and fall (Yld_GH2015F) 2015 at Kansas State University. The greenhouse yield experiments were conducted using the same design as the greenhouse FHB experiments described above. Spikes from five primary tillers in each pot were collected after maturity and hand-threshed to estimate KNS, GW, GL, GA, and TGW using a Marvin seed analyzer (GTA Sensorik GmbH, Germany). A two-dimensional image of a seed sample was extracted and the outline of the shadow area was determined. Then, GW and GL were measured along the cross and vertical sections of each seed, respectively. GA was measured by calculating the pixels inside the shadow area. Mean values from two replications were used for QTL mapping.

2.2.3 Evaluation of FHB and agronomic traits in field trials

The FHB field trial was conducted in the Rocky Ford FHB nursery, Manhattan, KS in the 2019-2020 wheat growing season (FHB_RF2020S). About 30 seeds per line were sown in a 1 m long single-row plot using a randomized complete block design with two replications. The nursery was inoculated by scattering 4 grams of *F. graminearum*-infested corn kernels per plot on the soil surface twice with the first application before the boot stage (Feekes 8) and the second application two weeks later (Feekes 10.1). The nursery was misted using an overhead impact sprinkler system

for 3 min hourly from 7 pm to 6 am daily between flowering (Feekes 10.5.1) and milky ripe (Feekes 11.1) stages to facilitate FHB infection. We estimated the FHB severity by visually rating of PSS in field, which might include both type I and type II resistance because the two type of resistance usually mix together in field. When highly susceptible lines showed over 90% PSS on 50% spikes, PSS was estimated visually for all RILs and parents heading at the same time window within 2 days along with these susceptible lines. HT, HD, SL, SNS, and SC were measured in the field experiment before harvest using the same method as described for the greenhouse experiments. All wheat plots were hand-harvested after maturity and threshed using an Almaco thresher (Nevada, IA) with the air blower open slightly. The collected seeds were manually cleaned to keep as many infected kernels as possible. Samples from the field trial were visually estimated for FDK by comparing the grain samples with a set of controls at 5, 10, 20, 50, 80, and 100% FDK. FDK value was determined by two skilled evaluators and averaged for QTL analysis. Ten grams of grain from each line were randomly sampled and ground to a fine powder for DON assay using a gas chromatography-mass spectrometry (GC-MS) at the University of Minnesota.

The RIL population was evaluated for KNS, TGW, GW, GL and GA in two additional field trials in spring 2020 (Yld_AB2020S) and 2021 (Yld_AB2021S) at the Kansas State University Agronomy Farm in Ashland Bottoms, Manhattan, KS. In these field experiments, the RILs were arranged in a randomized complete block design with two replications. For each RIL, 50 seeds were sown as a single row plot of 1.22-m long. Field management followed local practices without irrigation. KNS, GW, GL, GA, TGW, SL, SNS, SC, HT, and HD traits were estimated using the same methods as described above for greenhouse experiments.

2.2.4 DNA extraction and SNP genotyping

Three pieces of 2.0 cm-long wheat leaf tissues were collected at the two-leaf stage from each RIL and parent into 1.3 mL 96-deep-well plates with a 3.2-mm stainless steel bead in each well. The tissues were dried in a freeze dryer (ThermoSavant, Holbrook, NY) for 48 h and ground into a fine powder by shaking the plates at 30 cycles per sec for 3 min in a Mixer Mill (MM300, Retsch, Germany). Genomic DNA was isolated using a modified cetyltrimethyl ammonium bromide (CTAB) protocol (Bai et al. 1999). Genomic DNA quality was checked by electrophoresis using a 1% agarose gel and quantified using a Quant-iT™ PicoGreen® dsDNA Assay Kit (Thermo Fisher, Waltham, MA) and a FLUOstar Omega microplate reader (BMG LABTECH, German). The genotyping-by-sequencing (GBS) libraries were constructed using MspI and PstI restriction enzymes following the protocol from Poland et al. (Poland et al. 2012) and sequenced for three runs in an Ion Proton sequencer (Thermo Fisher, Waltham, MA, USA). The SNPs were called using the reference based GBSv2 pipeline implemented in the 'Trait analysis by association evolution and linkage' (TASSEL) package (Glaubitz et al. 2014). Only SNPs called from more than 70% of the RILs with heterozygotes <10% and minor allele frequency >20% (MAF) were used for linkage map construction.

2.2.5 Linkage map construction and QTL analysis

Initially, 1,600 GBS SNPs were used for the first round of linkage map construction and QTL analysis. After the QTL in 2DS was identified, eight additional kompetitive allele specific polymorphic chain reaction (KASP) markers and 12 SSR markers in the 2DS QTL interval were added to the linkage map. The KASP primers were designed based on the flanking sequences for four SNPs identified from exome capture (https://wheat.triticeaetoolbox.org/) (Blake et al. 2016), three SNPs from the wheat 55K SNP array (Liu et al. 2018), and one SNP (*AX-111561744*) from

Xu et al. (2020). In addition, 12 SSR markers that were mapped in the 2DS QTL region in previous studies (Table 2.2) (Röder et al. 1998; Guyomarc'h et al. 2002; Somers et al. 2004; Chai et al. 2018) and one KASP marker for a photoperiod gene *Ppd-D1* (Table 2.2) (Rasheed et al. 2016) were added to the QTL region. Redundant GBS-SNP markers were removed using the bin function in QTL IciMapping v4.1 (Meng et al. 2015) by keeping the markers with the least missing data points in each bin. The genetic linkage map was constructed with a minimum logarithm of odds (LOD) value of 3.0 using the IciMapping v4.1 software. Recombination rates were converted to genetic distances in centiMorgans (cM) using the Kosambi function (Kosambi 2016). Linkage groups were assigned to corresponding chromosomes based on the physical positions of these markers in the International Wheat Genome Sequencing Consortium (IWGSC) RefSeq v2.1 (Zhu et al. 2021).

QTL mapping was conducted using the inclusive composite interval mapping of additive function (ICIM-ADD) in IciMapping v4.1. The LOD threshold for each trait was estimated by 1000-time permutations using the BLUP values to claim a significant QTL. The QTL effects were estimated as the phenotypic variation explained (PVE) by the QTL calculated by ICIM in IciMapping v4.1. Peak LOD values in the QTL regions were used to estimate the QTL positions. QTL for different traits that were located to the same region or overlapped within the confidence interval were considered the same QTL. QTL significant in at least two experiments were considered relatively stable QTL.

QTL were named following international nomenclature. All QTL names started with 'Q', followed by a trait designator, a dot, a laboratory designator (HWWG to represent USDA, Hard Winter Wheat Genetics Research Unit), a hyphen (-) and the symbol for the chromosome or chromosome arm on which the QTL resided. If more than one QTL for a certain trait were

identified in the same chromosome, a serial number (1, 2, 3, etc.) was added after the chromosome name to show their order in the chromosome from the short arm to the long arm.

2.2.6 Conversion of GBS-SNPs to KASP markers

The GBS-SNPs within the major QTL interval for FHB resistance were converted to KASP assays (https://biosearch-cdn.azureedge.net/assetsv6/kasp-explanation-fact-sheet.pdf). The PolyMarker software (http://www.polymarker.info/) was used to design genome-specific primers for the KASP markers. Two tail sequences (GAAGGTGACCAAGTTCATGCT and GAAGGTCGGAGTCAACGGATT) were added to the 5'-end of the two allele-specific-forward primers to match with the FAM- and HEX-fluorescence-dye-labeled sequences in the KASP reaction mix. KASP assays were performed in a ProFlex™ Dual 384-well PCR system (Applied Biosystems, Foster City, CA) using a 4-uL reaction volume including 1.94 uL 2 x PACE ™ Genotyping Master Mix (3CR Bioscience, Harlow, Essex, UK),), 0.06 uL KASP primer mix and 2 uL genomic DNA at 25 ng/uL.

The PCR started with an initial denaturation step of 94 ℃ for 15 min, followed by 10 touch-down PCR cycles at 94 ℃ for 20 sec, and 60 ℃ for 1 min with -0.5 ℃/cycle, and then went through 35 cycles of 94 ℃ for 20 sec and 57 ℃ for 1 min. The PCR products were scanned in a FLUOstar® Omega microplate reader (BMG Labtech Inc., Cary, NC) and the signal data were analyzed using the KlusterCaller software v3.4.1.39 9 (LGC group, Teddington, UK). The newly designed KASP markers were evaluated for polymorphisms between the two parents, and the polymorphic markers were then used to genotype the mapping population to update the linkage map. The new map was used to re-map the QTL and the QTL map was drawn using MapChart v 2.32 (Voorrips 2002).

2.2.7 Statistical analysis

Experimental locations and years were combined as an environmental variable. Histogram and Pearson's correlation for each trait were calculated using ggplot2 and corrplot package in R, respectively (Wei et al. 2017; Villanueva and Chen 2019). Best linear unbiased prediction (BLUP) values for agronomic traits across greenhouse and field experiments were calculated separately using a mixed linear model, Y = R + E + G + G x E, implemented in the R packages lme4 (Bates et al. 2014). In the model, R was replication, E was environment, G was genotype, and G x E was interaction between genotypes and environments. All the variables were considered as random effects. Mean values of two replications in each experiment and the BLUP values were used for QTL mapping. Analysis of variance (ANOVA) was conducted separately for greenhouse and field experiments in all replicated trials using aov function (R Core Team, 2021). The broad-sense heritability was calculated using the formula $H^2=V_G/[V_G+V_{GxE}/E+Ve/(R*E)]$, where V_G was genotypic variance, V_{GxE} was variance of G x E, Ve is the residual variance, E is the number of environments, R is the number of replications. For FHB trait in greenhouse, the same model and analysis were conducted as above. For FHB traits in field, only one environment (FHB_RF2020S) was used to calculate the BLUP value using the following model, Y=R+G. ANOVA was conducted using the same model. The broad-sense heritability was calculated using the formula $H^2=V_G/(V_G+Ve/R)$. The BLUP value was used to do the QTL mapping instead of original phenotype data in field FHB trial.

To remove confounding effects of HD and HT on FHB traits, FHB traits were corrected by regression analysis using HD and HT as covariate factors. In greenhouse experiments, the corrected BLUP values of PSS were calculated using this mixed linear model, Y = R + E + G + G x E + HD + HT. In field experiments, the corrected BLUP values of PSS, FDK and DON content

were calculated using the following mixed linear model, Y = R + G + HD + HT, respectively. Both models were implemented in the R packages lme4 (Bates et al. 2014). All the variables were considered as random effects. The corrected BLUP values were used to re-map QTL. Analysis of variance was re-conducted using the same model as greenhouse and field, respectively. The broad-sense heritability was corrected using the formula $H^2=V_G/[V_G+V_{GxE}/E+Ve/(R*E)+V_{HD}+V_{HT}]$ in greenhouse but the formula $H^2=V_G/(V_G+Ve/R+V_{HD}+V_{HT})$ in field, where V_{HD} and V_{HT} were the variance of HD and HT, respectively.

2.3 Results

2.3.1 Phenotypic variation for wheat FHB resistance and other traits

In the greenhouse experiments, genotypic (G), environmental (E) effects and the G x E interactions were significant ($p < 0.01$) for all the traits measured in the RIL population except KNS, FDK and DON (Tables 2.3 & 2.4). In the field experiments, genotypic (G) effects were significant ($p < 0.01$) for all traits (Tables 2.3 & 2.4). The environmental (E) effects were significant ($p < 0.01$) for all agronomic traits (Tables 2.3 & 2.4). The G x E interactions were significant ($p < 0.05$) for most of these traits except GL and KNS (Tables 2.3 & 2.4).

The continuous distributions of the BLUP values were observed for three FHB traits (PSS, FDK, DON), and nine agronomic traits (HT, HD, SL, SC, KNS, SNS, GA, GW, GL) in the RIL population evaluated in both greenhouse and field experiments (Figures 2.1 & 2.2). BLUP values for most of traits fit normal distribution with exceptions of HD and SNS that showed bimodal distributions in both greenhouse and field experiments. The heritability was high for FHB traits (65%-88%) and agronomic traits (70%-96%) based on BLUP values (Tables 2.3 & 2.4), indicating that major portion of the variance for these traits was heritable.

HT significantly affected PSS in both greenhouse and field environments as well as DON in the field, whereas HD showed significant effects on PSS in the greenhouse and FDK in the field (Table 2.5). Therefore, HD and HT were used as covariant for ANOVA of FHB traits. The results indicated that genotypic effects of PSS, FDK and DON remained significant ($p < 0.01$) in both greenhouse and field experiments, but the heritability of PSS, FDK and DON were significantly reduced (Tables 2.3 & 2.5), indicating that HD and HT might have confounding effects on FHB traits in greenhouse and field conditions.

2.3.2 Correlations among FHB resistance and agronomic traits.

In the greenhouse experiments, PSS positively correlated with SC and kernel traits (TGW, GA, GW and GL) ($0.31 < r < 0.42$, $p < 0.01$), but negatively correlated with HD, HT, SNS and SL ($-0.82 < r < -0.55$, $p < 0.01$), suggesting that FHB resistant lines in general had later HD, taller HT, more SNS and longer SL but lower TGW and grain size than FHB susceptible lines in the population under greenhouse conditions (Table 2.6).

In the field experiment, highly positive correlations ($0.65 < r < 0.85$, $p < 0.01$) were observed among PSS, FDK and DON (Table 2.6). The three FHB traits also showed significantly negative correlations ($-0.64 < r < -0.41$, $p < 0.01$) with HD, HT, SNS and SL, but positive correlations with GW ($r = 0.26 - 0.31$, $p < 0.01$). The correlations were not significant between PSS and other kernel traits. Additionally, DON showed significant positive correlations with TGW, GA and GW ($0.23 < r < 0.31$, $p < 0.01$), and both FDK and DON were negatively correlated with KNS ($r = -0.29$ and -0.32, respectively, $p < 0.01$) (Table 2.6).

2.3.3 QTL for FHB resistance

Only one QTL (*QFhb.hwwg-2DS*) with a major effect on PSS (FHB severity) between markers *KASP2D58574820* and *KASP-Ppd-D1* on chromosome arm 2DS was significant in all

four greenhouse experiments and two BLUP datasets (Table 2.7), which explained 22.9 to 71.8% of the phenotypic variation for PSS in different experiments. *KASP-Ppd-D1* is a diagnostic KASP marker for gene *Ppd-D1*, a major photoperiod response gene regulating wheat heading date (Beales et al. 2007). The QTL for FDK and DON overlapped with *QFhb.hwwg-2DS* and explained 38.8% and 45.1% of the phenotypic variation for the two traits, respectively, in the field experiment. G97252W contributes the resistance alleles at this QTL for all the three traits, suggesting *QFhb.hwwg-2DS* is a major FHB resistance QTL with a pleiotropic effect on all three FHB traits (Table 2.7).

Since HD and HT were highly correlated with FHB traits, they may have confounding effects on FHB resistance, therefore, the FHB phenotypic data were adjusted with HD and HT data. The QTL on 2DS from G97252W remained highly significant and explained 28.9% of the phenotypic variation for PSS (*QFhb.hwwg-2DS*) in the greenhouse environments and 17.1% of the phenotypic variation for DON (*QDon.hwwg-2DS*) after removing these confounding effects due to HT and HD (Table 2.8). In the field conditions, the FHB resistance QTL was overlapped with *QDon.hwwg-2DS* and explained 11.7% of the phenotypic variation, but the QTL for FHB severity was mapped in a slightly different position from *QFhb.hwwg-2DS* identified from greenhouse data (Table 2.8).

2.3.4 QTL for yield-related traits

Three QTL were detected for KNS on chromosomes 2AL, 2DS and 4BS (Table 2.7). *QKns.hwwg-2DS* showed the largest effect in two field experiments and the field BLUP dataset, explained 19.1 to 25.1% of the phenotypic variation. *Ppd-D1* is within the QTL region. *QKns.hwwg-2AL* explained 8.4% to 11.3% of the phenotypic variation and *QKns.hwwg-4BS* explained 9.6% to 10.3% of the phenotypic variation in one field experiment and the field BLUP

dataset. G97252W contributes alleles for increased KNS at *QKns.hwwg-2DS* and *QKns.hwwg-2AL* but allele for decreased KNS at *QKns-4BS*. The same QTL interval on 2DS also showed a major effect on SNS, explaining 27.9 to 75.1% of the phenotypic variation in all greenhouse and field experiments with the increased SNS allele from G97252W.

Three QTL were significant for TGW and GA (Table 2.7). *QTgw.hwwg-2DS* showed the largest effect on TGW and GA, explaining up to 35.8% and 34.7% of the phenotypic variation, respectively, in greenhouse and field experiments. *QTgw.hwwg-2AL* explained up to 21.3% and 28.7% of the phenotypic variation for TGW and GA, respectively. *QTgw.hwwg-3AL* explained up to 12.9% and 16.1% of the phenotypic variation for TGW and GA, respectively. G79252W contributes high TGW and large GA alleles at *QTgw.hwwg-3AL*, whereas G97380A contributes the positive alleles at the other two QTL.

Two QTL were detected for GW (Table 2.7). G97380A contributes the wide grain alleles at both loci. Five QTL were detected for GL (Table 2.7). G97252W contributes the long grain alleles at *QGl.hwwg-3AL* and Q*Gl.hwwg-5AL* and G97380A contributes the long grain alleles at other three QTL.

2.3.5 QTL for other traits

Six QTL were detected for plant HT: two each on chromosome arms 2DS and 3AL, and one each on 2DL and 6BL (Table 2.7). *QHt.hwwg-2DS.2*, close to *Ppd-D1* and overlapping with *QFhb.hwwg-2DS*, showed the largest effect, explaining 22.0 to 48.9% of the phenotypic variation, and was significant in two greenhouse experiments, three field experiments and two BLUP datasets. *QHt.hwwg-2DS.1* including *Rht8* in this region was significant in three greenhouse experiments, three field experiments and two BLUP datasets, explaining 6.4 to 25.3% of the phenotypic variation. *QHt.hwwg-2DL* explained 7.4 to 14.0% of the phenotypic variation in one

greenhouse experiment, two field experiments and two BLUP datasets. Other QTL (*QHt.hwwg-3AL.1*, *QHt.hwwg-3AL.2* and *QHt.hwwg-6BL*) explained 3.8 to 11% of phenotypic variation in some of the greenhouse and field experiments. G97252A contributes the short alleles at all loci except for *QHt.hwwg-2DL* and *QHt.hwwg-6BL*.

Two QTL on chromosome arms 2DS and 7DS were significant for HD in at least two experiments (Table 2.7). Among them, *QHd.hwwg-2DS* showed the largest effect in all experiments, explaining 41.3 to 84.4 % of the phenotypic variation and *Ppd-D1* within the *QHd.hwwg-2DS* interval might be the causal gene. *QHd.hwwg-7DS* showed only a minor effect and explained only 4.1 to 4.9% of the phenotypic variation. G97252A carries early heading alleles at both loci.

Five SL QTL were mapped on chromosome arms 2DS, 3AL and 6BS, respectively (Table 2.7). G97252W contributes the long spike alleles at all loci except *QSl.hwwg-6BS*. Four QTL for SC were significant on chromosome arms 2DS, 3AL, 7BL and 7DS (Table 2.7). G97380A contributes the compactness alleles at *QSc.hwwg-2DS*, *QSc.hwwg-3AL* and *QSc.hwwg-7BL*.

2.3.6 QTL clusters for multiple traits

A total of four QTL clusters were discovered on chromosome arms 2AL, 2DS and 3AL in the mapping population (Table 2.9). Two QTL clusters for different traits were mapped on chromosome 2DS (Figure 2.3). The cluster 2DS-1 including *Rht8* that is flanked by *Xgwm261* (20.4Mb) and *KASP2D26715133* (26.7 Mb) based on IWGSC RefSeq v2.1 (Zhu et al. 2021) contains overlapping QTL for HT, SL and SC. The cluster 2DS-2 including *Ppd-D1* that is flanked by *KASP35014114* (35.0 Mb) and *KASP2D64237023* (64.2 Mb) contains QTL for PSS, FDK and DON and all nine agronomic traits (KNS, SNS, TGW, GA, GW, GL, HT, HD and SL). The cluster 2AL contains QTL for five kernel traits (KNS, TGW, GA, GW and GL) and was flanked by

GBS2A_460238480 (460.2 Mb) and *GBS2A_694523139* (694.5 Mb). The cluster 3AL flanked by *GBS3A_522003888* (522.0 Mb) and *GBS3A_667967807* (668.0 Mb) contains QTL for six agronomic traits (TGW, GA, GL, HT, SL and SC)

G97252W contributes positive alleles for FHB resistance and KNS and SNS, but negative alleles for TGW and grain size (GL and GW) at the cluster 2DS-2 (Table 2.9). Similarly, G97252W contributes alleles for more KNS, but lower TGW and smaller grain size at the cluster 2AL. However, G97252W contributes alleles for higher TGW and larger grain size at the cluster 3AL without adverse effects on FHB resistance, KNS and SNS. The QTL for different traits at the same locations may have pleiotropic effects on these traits or may be tightly linked.

2.4 Discussion

2.4.1 *QFhb.hwwg-2DS* is a stable major QTL for FHB type II resistance.

Sumai3 and its derivatives are derived from Chinese landraces and have been extensively used as sources of FHB resistance in wheat breeding programs worldwide (Zhu et al. 2019). However, exotic germplasm shows poor adaptability to geographical environments in US, which limits the deployment of exotic resource. Native resistance genes may provide a better alternative for developing locally adapted FHB resistant varieties (Ma et al. 2020; Ghimire et al. 2020). However, unfavorable association between FHB resistance and key agronomic traits complicates their adoption in U.S. hard winter wheat improvement (Suzuki et al. 2012; Gaire et al. 2021), therefore significant pre-breeding work needs to be done before those resistance genes can be used in breeding.

In this study, marker analysis indicated that G97380A carries the semi-dwarfing *Rht8* allele and the photoperiod-insensitive *Ppd-D1a* allele; while G97252W carries the alternative alleles for the two genes. *QFhb.hwwg-2DS* for three FHB resistance traits was mapped in the vicinity of the

two genes regulating plant HT and HD, respectively, on chromosome arm 2DS of G97252W in the G97252W x G97380A population. *QFhb.hwwg-2DS* showed major effect on PSS, FDK and DON, and explained up to 71.8% of the phenotypic variation for PSS in the greenhouse using the single floret inoculation and 22.9% in the field where plants were inoculated by *Fusarium* infected corn spawn (Table 2.7). In the field condition, FHB resistance of *QFhb.hwwg-2DS* may be contributed by both type I (resistance to initial infection) and type II (resistance to FHB spread within a spike) because ascospores produced from infected corn spawn randomly landed on wheat spikes and each spike could have multiple initial infection sites. However, in greenhouse conditions, a larger effect on FHB resistance was detected for the QTL than that in the field. The single floret inoculation evaluates type II resistance only, therefore *QFhb.hwwg-2DS* is most likely a QTL mainly for type II resistance.

QFhb.hwwg-2DS was mapped between markers *KASP2D30932191* (30.9 Mb) and *KASP2D58574820* (58.6 Mb) (Table 2.7). Previously, McCartney et al. (2016) reported three FHB resistance QTL on 2DS (*QFhb.crc-2D.1*, *QFhb.crc-2D.2* and *QFhb.crc-2D.3*) between *Xgwm261* (20.4 Mb) and *Xgwm484* (50.6 Mb) based on IWGSC RefSeq v2.1. The Canadian spring wheat 'Kenyon' contributed the FHB resistance alleles at all three QTL. The QTL for multiple FHB traits were also mapped in the same 2DS region of several Canadian winter wheat cultivars and a U.S. soft winter wheat 'Truman' in other studies (Islam et al. 2016; Tamburic-Ilincic and Rosa 2019; Dhariwal et al. 2020). Based on the physical positions of the flanking markers (Table 2.7), *QFhb.hwwg-2DS* identified in this study is most likely the same as the previously reported QTL on 2DS. Consistent detection of *QFhb.hwwg-2DS* in diverse germplasm indicates that *QFhb.hwwg-2DS* is a stable QTL in North American wheat and *QFhb.hwwg-2DS* reduces not only FHB disease severity but also DON content in harvested grain in different genetic backgrounds

and testing environments. The KASP markers, *KASP2D35014114* and *KASP-Ppd-D1*, flanking *QFhb.hwwg-2DS* can be used to select *QFhb.hwwg-2DS* in wheat breeding.

2.4.2 Relationship between *QFhb.hwwg-2DS* and other agronomic traits

In the *QFhb.hwwg-2DS* region, QTL were also detected for plant HT (*QHt.hwwg-2DS.2*) and HD (*QHd.hwwg-2DS*). *QHd.hwwg-2DS* was responsible for HD and mapped at the *Ppd-D1* locus (Figures 2.3 & 2.4; Table 2.7), suggesting that *Ppd-D1* is most likely the causal gene; whereas *QHt.hwwg-2DS.1* for plant HT was detected in the *Rht8* position, indicating *Rht8* is the major contributor to the plant height variation in the QTL region. In field conditions, tall and late headed plants might have reduced FHB infection due to disease escaping mechanism under natural infection conditions (Bai et al. 2018). However, *QFhb.hwwg-2DS* was still highly significant with a major effect on PSS and DON content in the field condition after removing the effects of HD and HT (Table 2.8), demonstrating the QTL for FHB resistance is real. In the greenhouse trials, the plants were manually inoculated using point inoculation, therefore, disease infection and development should not be affected by HD and HT under relatively controlled environments. To validate this, *QFhb.hwwg-2DS* was re-mapped with the greenhouse FHB data after adjusted by HD and HT data. The results showed that the QTL was still highly significant, but the effect was significantly reduced (Table 2.8). Those results indicate that *QFhb.hwwg-2DS* is a real QTL for FHB resistance that may be either tightly linked to or pleiotropy of *Ppd-D1b* and *Rht8*. Previously, Xu et al. (2020) reported the association between the semi-dwarfing *Rht8* allele and increased FHB susceptibility. *Rht8* was physically located in the 25.6 Mb position on 2DS of Chinese cultivar ‘Y8679’ (Chai et al. 2022), which is about 10 Mb from *QFhb.hwwg-2DS*. Factors such as difference in mapping populations and FHB evaluation conditions may cause shift of QTL positions in different experiments. The results from this study cannot determine the causal gene

for *QFhg.hwwg-2DS* and further fine mapping in the QTL region may provide insight on the relationship between *QFhb.hwwg-2DS* and *Ppd-D1b* or *Rht8*.

Besides HD and HT genes, three QTL (*QSns.hwwg-2DS*, *QKns.hwwg-2DS* and *QTgw.hwwg-2DS*) for SNS, KNS, TGW on 2DS also overlapped with *QFhb.hwwg-2DS* in this study (Table 2.7; Figure 2.3). Previously, QTL for SNS (Zhou et al. 2017; Ma et al. 2019a; Li et al. 2020), KNS (Lin et al. 2021) and TGW (Maphosa et al. 2014; Ma et al. 2019b) were detected in the *QFhb.hwwg-2DS* region in several wheat cultivars or landraces. Based on their physical positions, *QSns.hwwg-2DS*, *QKns.hwwg-2DS* and *QTgw.hwwg-2DS* are likely the same QTL as previously reported. *QFhb.hwwg-2DS* is likely the QTL with pleiotropic effects on SNS, KNS and TGW or linked to QTL for these traits.

In the 2DS-2 QTL cluster where *Ppd-D1* is located, 12 QTL were identified in the current study (Figure 2.3; Table 2.9). This QTL cluster in G97252W showed increased FHB resistance, improved KNS and SNS, but reduced TGW and grain size, suggesting that high spikelet fertility decreases host vulnerability to *Fusarium* infection in spikes and high KSN and SNS usually lower TGW. *QFhb.hwwg-2DS* in G97252W contributed to taller plants and later HD that are not desired in modern cultivars (Table 2.9). Fortunately, 3AL QTL cluster in G97252W showed increased TGW and grain size without negative effects on FHB resistance, KNS and SNS (Table 2.9), thus this QTL can be pyramided with *QFhb.hwwg-2DS* to improve FHB resistance and reduce yield penalty (Table 2.10). To balance the adverse effects of later HD due to *Ppd-D1*, *QFhb.hwwg-2DS* can be deployed in climatic regions of high latitude where have strong winter and long daylength during wheat heading and late onset of hot-dry weather at grain filling stage to maximize yield potential. In addition, *QFhb.hwwg-2DS* can be pyramided with positive alleles for yield traits at different QTL from different sources to simultaneously improve FHB resistance and agronomic

traits. Also, breaking the unfavorable linkage between *QFhb.hwwg-2DS* and *Ppd-D1b* using cytogenetic and genomic tools is possible if they are linked genes.

2.4.3 Other QTL for grain yield component traits

In the current study, two overlapping QTL for KNS and TGW were mapped on 2AL (*QKns.hwwg-2AL*, *QTgw.hwwg-2AL*) between *GBS2A_460238480* (60.2 Mb) and *GBS2A_626218088* (626.2 Mb) (Table 2.7). The two QTL are either tightly linked or pleiotropic. G97252W contributed alleles for more KSN but lower TGW. Shi et al. (2017) identified a SNP *IWB7310* (616.6 Mb) that was associated with a QTL for KNS and Liu et al. (2017) found a SNP *B4170* (522.6 Mb) for a TGW QTL in the region. These QTL were located in the same region and are most likely the same QTL as the ones identified in this study.

QKns.hwwg-4BS was physically mapped between 18.5 Mb and 20.5 Mb (Table 2.7) in this study, which may be the same QTL reported by Li et al. (2018). Because *IWB45065* (18.5 Mb) is a tightly linked marker to both QTL. *QTgw.hwwg-3AL* was flanked by *GBS3A_648390973* (648.4 Mb) and *GBS3A_659255761* (659.3 Mb) on 3AL (Table 2.7), which is likely the same QTL reported by Yang et al. (2021).

In this study, SNP and SSR markers were developed for the QTL of both FHB resistance and agronomic traits (Tables 2.2 & 2.11). Some of the markers can be used in marker-assisted selection (MAS) in breeding or in fine mapping of those QTL to further dissect the genetic relationship between FHB resistance and agronomic traits.

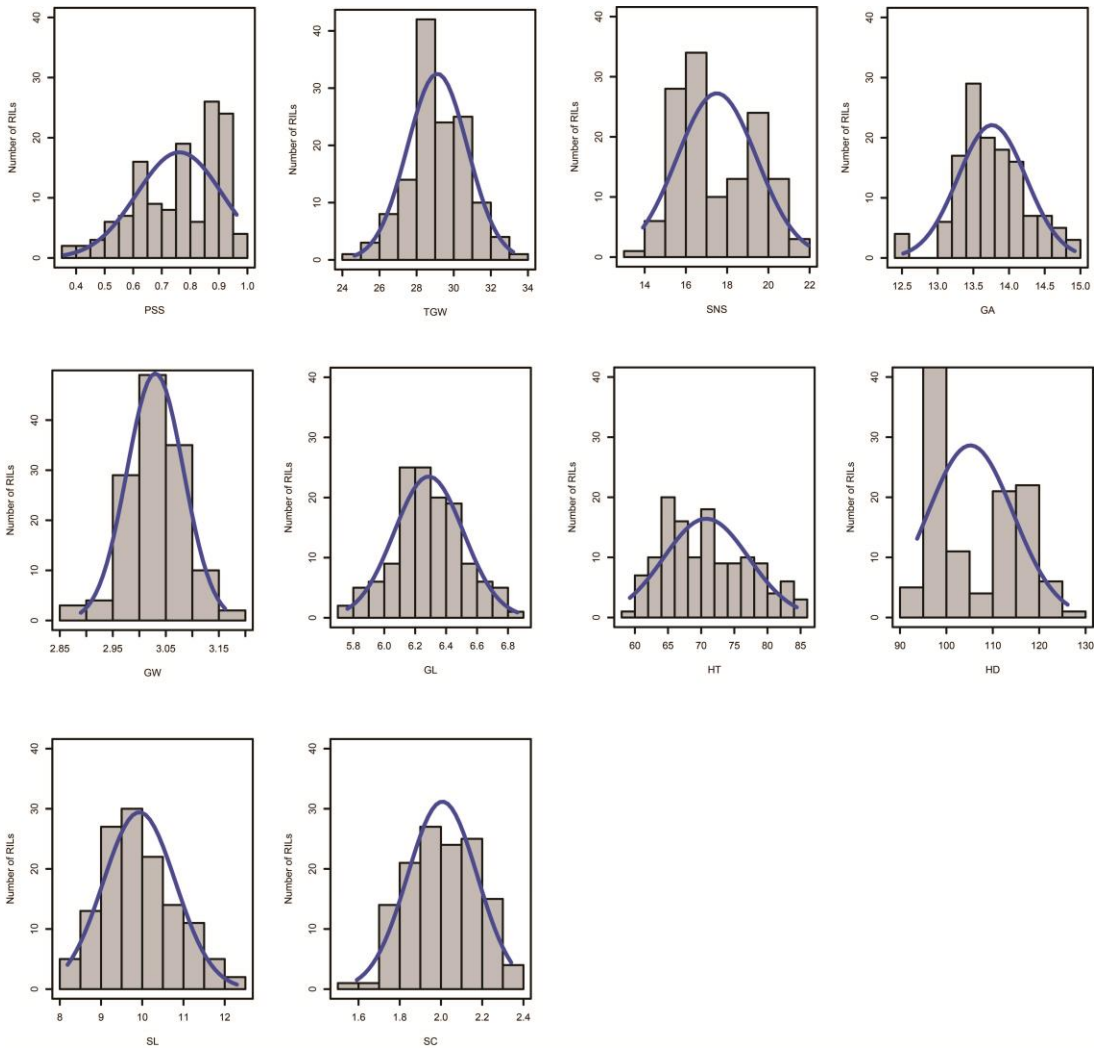

Figure 2.1 Distribution of best linear unbiased prediction (BLUP) values of Fusarium head blight (FHB) and agronomic traits in greenhouse experiments.

PSS, percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length.

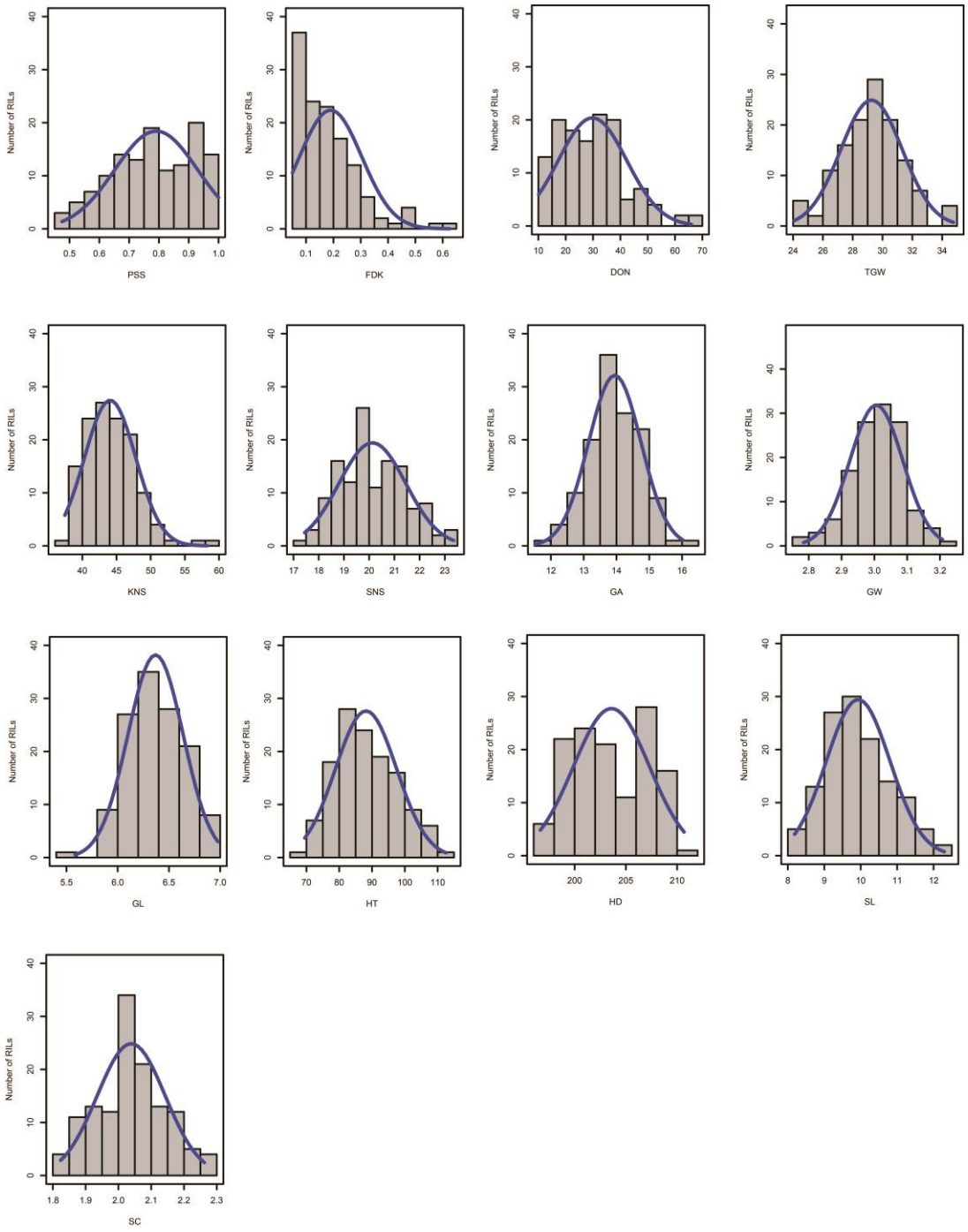

Figure 2.2 Distribution of best linear unbiased prediction (BLUP) values of Fusarium head blight (FHB) and agronomic traits in field experiments.

PSS, percentage of symptomatic spikelets; FDK, Fusarium damaged kernel; DON, deoxynivalenol; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; KNS, kernel number per spike.

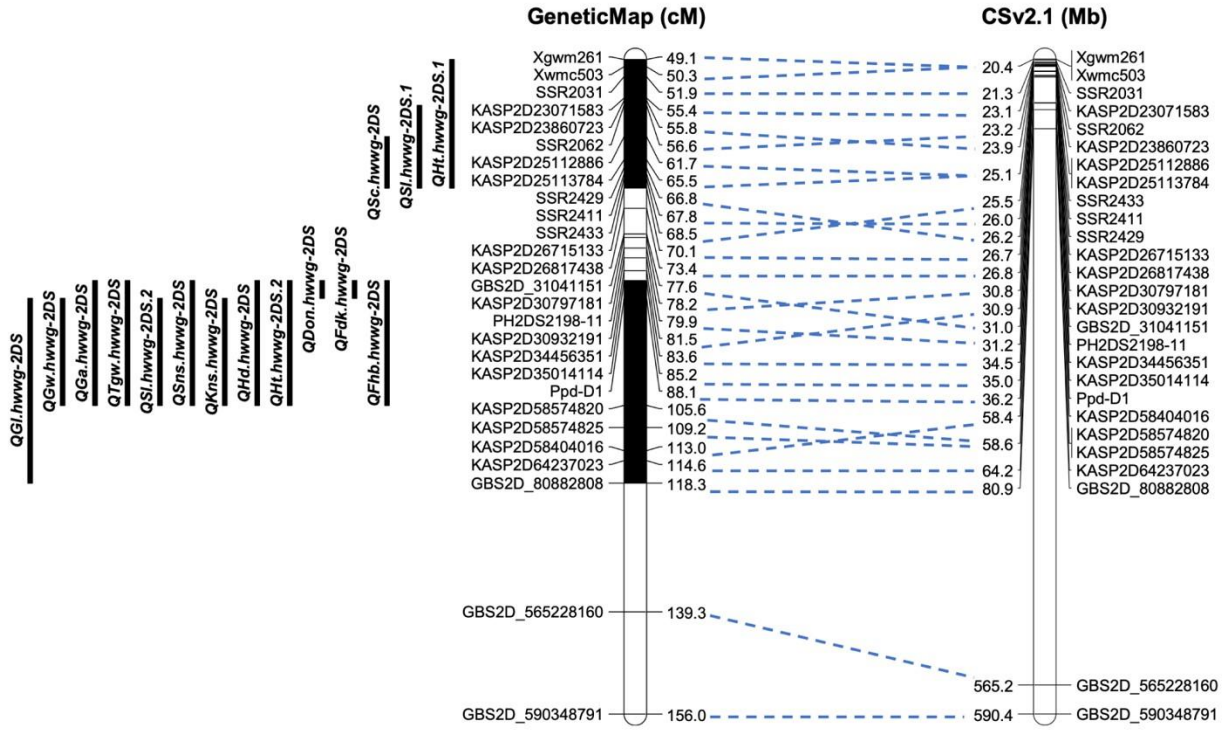

Figure 2.3 Partial genetic map (left) and physical map (right) based on Chinese Spring v2.1 (CSv2.1) for chromosome 2D to show the quantitative trait locus (QTL) regions (black bars in the linkage map) for multiple traits (QTL names and intervals on the left).

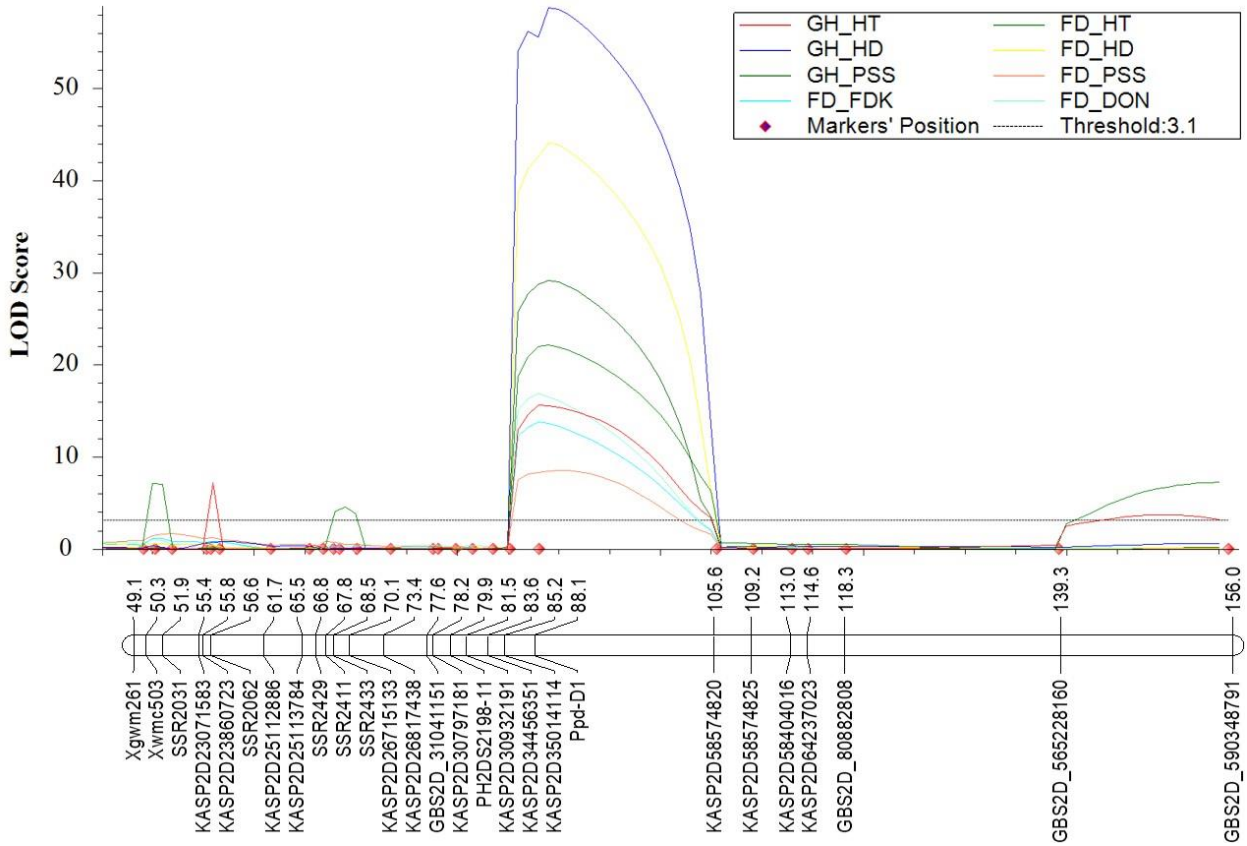

Figure 2.4 The overlapping quantitative trait locus (QTL) on 2DS based on best linear unbiased prediction (BLUP) values of percentage of symptomatic spikelets (PSS), Fusarium damaged kernel (FDK, deoxynivalenol (DON) content, heading date (HD) and plant height (HT) under greenhouse (GH) and field (FD) experiments.

Table 2.1 Phenotypic variation of traits based on best linear unbiased prediction (BLUP) values in greenhouse and field environments.

Trait	Field				Greenhouse			
	RIL		G97252W	G97380A	RIL		G97252W	G97380A
	Range	Mean ± SD	Mean ± SD	Mean ± SD	Range	Mean ± SD	Mean ± SD	Mean ± SD
PSS (%)	20.0 - 100.0	80.0 ± 20.0	46.3 ± 12.5	80.0 ± 8.2	10.0 - 100.0	80.0 ± 30.0	37.0 ± 23.7	92.0 ± 12.7
FDK	0.1 - 0.7	0.2 ± 0.1	18.3 ± 5.8	26.7 ± 7.6	NA	NA	NA	NA
DON (ppm)	5.9 - 78.9	29.7 ± 13.8	NA	NA	NA	NA	NA	NA
HT (cm)	61 - 125.7	88.2 ± 12.0	97.7 ± 5.5	77.9 ± 4.4	38.0 - 110.5	70.8 ± 9.8	70.8 ± 3.3	61.9 ± 6.0
HD	191.0 - 215.0	203.5 ± 5.3	211.6 ± 1.7	194.3 ± 1.4	76.0 -150	104.9 ± 14.9	102.3 ± 3.5	90.1 ± 3.8
SNS	16.2 - 25.4	20.1 ± 1.7	23.6 ± 0.6	19 ± 0.5	10.4 - 28.3	17.5 ± 2.8	18.7 ± 0.9	16.2 ± 0.7
SL (cm)	6.9 - 13.6	9.9 ± 1.2	11.4 ± 0.5	10.3 ± 0.7	5.6 - 16.2	8.9 ± 2.0	9.1 ± 0.5	7.6 ± 0.4
SC	1.6 - 2.5	2.0 ± 0.2	2.1 ± 0.1	1.8 ± 0.1	1.0 - 3.2	2.0 ± 0.3	2.1 ± 0.1	2.1 ± 0.1
TGW (g)	16.1 - 40.4	29.4 ± 4.1	22.6 ± 2.4	30.8 ± 2.3	8.6 - 46.5	29.2 ± 6.2	25.2 ± 3.5	33.2 ± 4.8
GA (mm^2)	10 - 17.3	14 ± 1.2	11.9 ± 0.6	14.5 ± 0.4	7.7 - 18.2	13.8 ± 1.6	12.4 ± 0.8	14.4 ± 1.5
GW (mm)	2.4 - 3.4	3.0 ± 0.2	2.7 ± 0.1	3.1 ± 0.1	2.1 - 3.5	3.0 ± 0.2	2.8 ± 0.1	3.1 ± 0.2
GL (mm)	5.4 - 7.2	6.4 ± 0.3	6.0 ± 0.2	6.4 ± 0.1	4.6 - 7.5	6.3 ± 0.4	6.0 ± 0.2	6.2 ± 0.3
KNS	17.4 - 75.9	43.9 ± 8.6	61.2 ± 7.3	40 ± 3.1	NA	NA	47.8 ± 8.2	36.2 ± 4.0

SD, standard deviation; PSS, Percentage of symptomatic spikelet; FDK, Fusarium damaged kernel; DON, deoxynivalenol; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; KNS, kernel number per spike; GA, grain area; GW, grain width; GL, grain length; ppm, parts per million; mm2, square of millimeter; RILs, recombinant inbred lines; NA, not available data.

Table 2.2 Kompetitive allele specific polymerase chain reaction (KASP) and simple sequence repeat (SSR) primers for quantitative trait locus (QTL) on chromosome 2D.

Marker	Physical position (bp)	FAM primer	HEX primer	Common primer
KASP2D15149539	chr2D:15149539	TCCCGCCACTGATCGACC	TCCCGCCACTGATCGACT	CAAGAGAGAGTCGGAGCGTC
KASP2D22467690	chr2D:22467690	GCATGCCGATCTCATTCGCT	GCATGCCGATCTCATTCGCA	AGAGCAGACGAGATCGGAGATA
KASP2D23071583	chr2D:23071583	TGAGAAAGTCTATCGCGGCG	TGAGAAAGTCTATCGCGGCC	TGATGCTCGGATCTGACATGG
KASP2D23860723	chr2D:23860723	TGGACATAGACTTCTTCCTGGAC	TGGACATAGACTTCTTCCTGGAG	GATGTCCGCTCCACGTTCT
KASP2D32741426	chr2D:32741426	TACTGCAGCACGTAGGAGAA	TACTGCAGCACGTAGGAGAG	ACTTTCTGCTTCGCCTTCGA
KASP2D32741615	chr2D:32741615	AGGAGGGCGAGAGGTCCA	AGGAGGGCGAGAGGTCCG	TGGCTGCAGCTCTTGTACTC
KASP2D33555125	chr2D:33555125	AGCAGGTGGGAATGATTGGA	AGCAGGTGGGAATGATTGGG	CGATGGTAATTCTCTCCGGTCTA
KASP2D33652726	chr2D:33652726	CTTTCAACCTGCAGCTCCCTA	CTTTCAACCTGCAGCTCCCTG	CAAACTCGACCGAAAGACGC
KASP2D34056946	chr2D:34056946	GTGCAGAACGTCGAGGCAA	GTGCAGAACGTCGAGGCAG	CCGATCTGAAGTGTAGTGCCTT
KASP2D34074329	chr2D:34074329	GGTGGAGCTGAAAGACGAACA	GGTGGAGCTGAAAGACGAACG	CCTCCTCCGCCATTTGACAT
KASP2D34211948	chr2D:34211948	GTGCCCGAGACGATGCGG	GTGCCCGAGACGATGCGC	GACCTGCAGCACGCGGCC
KASP2D34456351	chr2D:34456351	CCAATGCTCTTCACACACTACA	CCAATGCTCTTCACACACTACG	TGCAGGGTCTTTGGAAGGAC
KASP2D35014114	chr2D:35014114	GCTCGCATGCACGTACTGT	GCTCGCATGCACGTACTGC	CTCGCTGGCCAGTAGTAACT
KASP2D53864222	chr2D:53864222	GAAACCAACTCCGGCGGCTA	GAAACCAACTCCGGCGGCTG	CCACTGCAGCCATCGCTC
KASP2D58404016	chr2D:58404016	CCTCGCCATGACAACAGC	CCTCGCCATGACAACAGG	CTGCACGCCATCTTTTGCTT
KASP2D58574820	chr2D:58574820	CATAGGATCGGCCACGTGT	CATAGGATCGGCCACGTGC	TCAGAATTTCTCCATGTGTGTGC
KASP2D58574825	chr2D:58574825	GGTATAGCATAGGATCGGCCA	GGTATAGCATAGGATCGGCCG	ATTTCTCCATGTGTGTGCGG
KASP2D64237023	chr2D:64237023	ATCCTCCTCCCGGCAGAAT	ATCCTCCTCCCGGCAGAAC	GTCGTCTTCTTCTTCCTCGTCA
KASP2D25112886	chr2D:25112886	CTTTGAGGCAGTCCAGTCCC	CTTTGAGGCAGTCCAGTCCA	CCTGAGCAACCTAATTCAATAGC
KASP2D25113784	chr2D:25113784	CGAGGGGAAGTGGATGCTG	CGAGGGGAAGTGGATGCTA	CCATGTCAACCTCCTCCTCA
KASP2D26715133	chr2D:26715133	CCAAGAATCGACAACTCCACA	CCAAGAATCGACAACTCCACC	GGAGGATATTCGTCACCAAAGG
KASP2D26817438	chr2D:26817438	ACGAAGCTTCTTTTGTCGCG	ACGAAGCTTCTTTTGTCGCA	GCCTTGCTGAGAAAATCGCA
KASP2D28294547	chr2D:28294547	GACTTTACCTTGAGATTGCCCA	GACTTTACCTTGAGATTGCCCG	CGTCGAAGATTGCGGGGTG
KASP2D30797181	chr2D:30797181	CATGCGTCGTCACCAAGC	CATGCGTCGTCACCAAGT	GGTGCCGAAGTACTCTATCACT
KASP2D30932191	chr2D:30932191	GACTTGACGACGAGGCCT	GACTTGACGACGAGGCCC	GACCTCGACGCGCTCCTT

KASP-Ppd-D1	chr2D:36208612	CAAGGAAGTATGAGCAGCGGTT	AAGAGGAAACATGTTGGGGTCC	GCCTCCCACTACACTGGGC
SSR2212	chr2D:20348096	ACAACGTGCAAGTCACCATA	-	ACCCTGCACGATAGCAATAC
Xgwm261	chr2D:20423444	CTCCCTGTACGCCTAAGGC	-	CTCGCGCTACTAGCCATTG
Xwmc503	chr2D:20431920	GCAATAGTTCCCGCAAGAAAAG	-	ATCAACTACCTCCAGATCCCGT
SSR2031	chr2D:21284434	CAGGTTCCTAGATCATCAAGTT	-	GATGGTTGTAGGGTACAATGTT
SSR2062	chr2D:23150762/chr2D:23176570/chr2D:23193161	GCTAGGTGTGTTTAAAAGTTGG	-	AGCTGATCGATGCTTATCTAGT
SSR2089	chr2D:23911137/chr2D:23932350	CAACGTTTGACCTCTCTCTC	-	AAGGGATAGATACTGCCACA
SSR2087	chr2D:24023554	GTAAATTGGGCTCAACAAGT	-	TGCACGAAGGACCTAATAGT
Xcfd53	chr2D:24726238	CCCTATTTCCCCCATGTCTT	-	AAGGAGGGCACATATCGTTG
SSR2433	chrUn:18734097	GCAATTCCTAGAGATCAAATTC	-	GTGTAGCATTCCATCTCATTC
SSR2411	chr2D:25967481	GGACCACATTTTCTCTTTCTT	-	CTTGATCACATTCACATTCCT
SSR2429	chr2D:26243135	GTCGGCTATAATTACCCTAGC	-	CTCTCACACACACACACTCTG
PH2DS2198-11	chr2D:31180131	AGCCATAGGAGCCATAGTCAT	-	TCCGACCCCTGTAATAGCC

Table 2.3 Analysis of variance (ANOVA) of Fusarium head blight (FHB) and developmental traits across greenhouse and field environments in G97252W x G97380A population.

Location	Source of variation	PSS			FDK			DON			HT			HD		
		df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)
Greenhouse	Replication	1	0.19	88		NA			NA		1	3.3	91	1	0.17	93
	Environment (E)	3	97.74**								3	25.66**		3	1890.88**	
	Genotype (G)	131	9.11**								131	13.24**		131	51.21**	
	G x E	383	1.49**								383	1.83**		383	5.05**	
	Residuals	512	NA								514	NA		509	NA	
Field	Replication	1	0.34	65	1	1.37	79	1	3.66	78	1	5.97*	93	1	0.11	89
	Environment (E)	NA			NA			NA			2	214.29**		2	358.05**	
	Genotype (G)	129	2.86**		129	4.88**		129	4.73**		131	19.04**		130	19.40**	
	G x E	NA			NA			NA			244	1.34**		244	2.12**	
	Residuals	129	NA		126	NA		129	NA		370	NA		369	NA	

PSS, Percentage of symptomatic spikelet; FDK, Fusarium damaged kernel; DON, deoxynivalenol; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; KNS, kernel number per spike; NA, not available data; df, freedom degree; H^2, broad-sense heritability; * and ** are significant at p=0.05 and p=0.01, respectively.

Table 2.4 Analysis of variance (ANOVA) of spike and yield component traits across greenhouse and field environments in G97252W x G97380A population.

Location	Source of variation	SNS			SL			SC			TGW		
		df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)
Greenhouse	Replication	1	0.21	95	1	4.60*	95	1	0.2	90	1	55.97**	70
	Environment (E)	3	466.08**		3	321.80**		3	116.05**		1	188.93**	
	Genotype (G)	131	21.68**		131	37.69**		131	10.43**		131	3.05**	
	G x E	383	1.47**		383	2.92**		382	1.47**		130	1.66**	
	Residuals	512	NA		513	NA		510	NA		252	NA	
Field	Replication	1	0.72	93	1	0.25	92	1	1.25	85	1	6.27*	77
	Environment (E)	2	258.35**		2	247.85**		2	53.29**		1	92.44**	
	Genotype (G)	131	20.90**		131	21.28**		131	11.28**		129	4.58**	
	G x E	244	1.61**		244	1.81**		244	1.74**		118	1.49**	
	Residuals	358	NA		359	NA		358	NA		228	NA	
Location	Source of variation	GA			GW			GL			KNS		
		df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)
Greenhouse	Replication	1	66.51**	72	1	45.84**	67	1	77.36**	92		NA	
	Environment (E)	1	126.82**		1	63.74**		1	105.13**				
	Genotype (G)	131	3.89**		131	2.64**		131	7.63**				
	G x E	130	2.03**		130	1.54**		130	1.62**				
	Residuals	252	NA		252	NA		252	NA				
Field	Replication	1	1.25	87	1	0.64	79	1	0.5	96	1	1.48	74
	Environment (E)	1	60.35**		1	43.94**		1	83.94**		1	67.05**	
	Genotype (G)	129	7.38**		129	4.54**		129	19.59**		129	3.01**	
	G x E	118	1.47**		118	1.40*		118	1.24		118	1.11	
	Residuals	228	NA		228	NA		228	NA		228	NA	

PSS, Percentage of symptomatic spikelet; FDK, Fusarium damaged kernel; DON, deoxynivalenol; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; KNS, kernel number per spike; NA, not available data; df, freedom degree; H2, broad-sense heritability; * and ** are significant at p=0.05 and p=0.01, respectively.

Table 2.5 Corrected analysis of variance (ANOVA) for Fusarium head blight (FHB) traits after masking confounding effects across greenhouse and field environments.

Location	Source of variation	PSS			FDK			DON		
		df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)
Greenhouse	Replication	1	0.46	20	NA			NA		
	Environment (E)	3	98.3**							
	Genotype (G)	131	9.10**							
	G x E	382	1.44**							
	HT	1	10.24**							
	HD	1	9.89**							
	Residuals	505	NA							
Field	Replication	1	0.39	46	1	1.41	45	1	3.88	55
	Genotype (G)	129	3.31**		129	5.04**		129	5.01**	
	HT	1	19.94**		1	0.57		1	6.29*	
	HD	1	2.41		1	5.65*		1	3.43	
	Residuals	127	NA		124	NA		127	NA	

PSS, Percentage of symptomatic spikelets; FDK, Fusarium damaged kernel; DON, deoxynivalenol; HD, heading date; HT, plant height; NA, not available data; df, freedom degree; H^2, broad-sense heritability; * and ** are significant at p=0.05 and p=0.01, respectively.

Table 2.6 The correlation coefficients between Fusarium head blight (FHB) and agronomic traits based on best linear unbiased prediction (BLUP) values.

Environment	Traits	FDK	DON	HT	HD	SNS	SL	SC	TGW	GA	GW	GL	KNS
Field	PSS	0.65**	0.65**	-0.41**	-0.48**	-0.44**	-0.42**	0.14	0.19	0.22	0.26**	0.1	-0.19
	FDK		0.85**	-0.51**	-0.64**	-0.54**	-0.48**	0.12	0.19	0.21	0.26**	0.1	-0.29**
	DON			-0.53**	-0.62**	-0.51**	-0.5**	0.19	0.23**	0.24**	0.31**	0.1	-0.32**
Greenhouse	PSS	NA	NA	-0.55**	-0.82**	-0.8**	-0.72**	0.31**	0.42**	0.38**	0.32**	0.37**	NA

FDK, Fusarium damaged kernel; PSS, percentage of symptomatic spikelet in a spike; DON, deoxynivalenol; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; KNS, kernel number per spike; NA, not available data; ** indicates significant at $p = 0.01$.

Table 2.7 Quantitative trait locus (QTL) mapping results in G97252W x G97380A population.

Trait	QTL	Experiment	Interval	LOD	PVE (%)	Add
FHB severity	*QFhb.hwwg-2DS*	FHB_GH2019S	KASP2D35014114:KASP-Ppd-D1	13.0	25.8	0.1
		FHB_GH2019F	KASP-Ppd-D1:KASP2D58574820	38.2	71.8	0.2
		FHB_GH2019W	KASP-Ppd-D1:KASP2D58574820	18.9	47.1	0.1
		FHB_GH2020S	KASP2D35014114:KASP-Ppd-D1	10.5	31.8	0.1
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	29.1	63.7	0.1
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	8.6	22.9	0.1
FDK	*QFdk.hwwg-2DS*	FD_BLUP	KASP2D35014114:KASP-Ppd-D1	13.8	38.8	0.1
DON	*QDon.hwwg-2DS*	FD_BLUP	KASP2D35014114:KASP-Ppd-D1	16.9	45.1	6.6
KNS	*QKns.hwwg-2AL*	Yld_AB2020S	GBS2A_460238480:GBS2A_570889235	3.6	8.4	2.1
		FD_BLUP	GBS2A_570889235:GBS2A_626218088	4.5	11.3	1.2
	QKns.hwwg-2DS	Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	7.3	22.6	3.6
		Yld_AB2021S	KASP-Ppd-D1:KASP2D58574820	5.6	19.1	2.7
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	9.6	25.1	1.8
	QKns.hwwg-4BS	Yld_AB2021S	GBS4B_18531114:GBS4B_15729471	3.4	9.6	-1.9
		FD_BLUP	GBS4B_20496100:GBS4B_18531114	4.5	10.3	-1.2
SNS	*QSns.hwwg-2DS*	FHB_GH2019S	KASP-Ppd-D1:KASP2D58574820	30.4	61.8	1.2
		FHB_GH2019F	KASP2D35014114:KASP-Ppd-D1	34.0	63.5	2.1
		FHB_GH2019W	KASP-Ppd-D1:KASP2D58574820	33.1	68.4	2.4
		FHB_GH2020S	KASP2D35014114:KASP-Ppd-D1	29.8	62.2	1.6
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	41.4	75.1	1.7
		FHB_RF2020S	KASP-Ppd-D1:KASP2D58574820	13.5	27.9	0.7
		Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	21.5	54.2	1.3

		Yld_AB2021S	KASP-Ppd-D1:KASP2D58574820	21.9	53.4	1.4
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	23.3	54.6	1.0
TGW	*QTgw.hwwg-2AL*	Yld_AB2021S	GBS2A_460238480:GBS2A_570889235	3.2	11.9	-1.2
		Yld_AB2020S	GBS2A_570889235:GBS2A_626218088	11.2	20.8	-1.9
		FD_BLUP	GBS2A_460238480:GBS2A_570889235	9.4	21.3	-1.0
	QTgw.hwwg-2DS	Yld_GH2015F	KASP2D35014114:KASP-Ppd-D1	14.7	35.8	-3.3
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	8.0	20.5	-0.8
		Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	7.2	14.1	-1.6
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	3.3	9.7	-0.7
	QTgw.hwwg-3AL	Yld_GH2015S	GBS3A_659202246:GBS3A_621315736	3.1	10.3	1.2
		GH_BLUP	GBS3A_648390973:GBS3A_659255761	6.4	12.9	0.6
		Yld_AB2020S	GBS3A_648390973:GBS3A_659255761	4.0	6.4	1.0
		FD_BLUP	GBS3A_648390973:GBS3A_659255761	3.4	6.9	0.6
GA	*QGa.hwwg-2AL*	Yld_GH2015F	GBS2A_626218088:GBS2A_690424019	5.3	9.0	-0.5
		Yld_AB2020S	GBS2A_570889235:GBS2A_626218088	14.8	27.6	-0.7
		Yld_AB2021S	GBS2A_460238480:GBS2A_570889235	6.0	18.6	-0.4
		FD_BLUP	GBS2A_460238480:GBS2A_570889235	14.8	28.7	-0.5
	QGa.hwwg-2DS	Yld_GH2015F	KASP2D35014114:KASP-Ppd-D1	17.4	34.7	-0.9
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	6.5	12.8	-0.2
		Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	7.6	13.6	-0.5
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	5.9	12.7	-0.3
	QGa.hwwg-3AL	Yld_GH2015S	GBS3A_659202246:GBS3A_621315736	4.1	12.2	0.3
		Yld_GH2015F	GBS3A_648390973:GBS3A_659255761	4.6	7.1	0.4
		GH_BLUP	GBS3A_623594682:GBS3A_553116586	8.3	16.1	0.2
		Yld_AB2020S	GBS3A_648390973:GBS3A_659255761	5.9	8.9	0.4

		FD_BLUP	GBS3A_667967807:GBS3A_655968576	5.4	8.9	0.3
GW	*QGw.hwwg-2AL*	Yld_AB2020S	GBS2A_570889235:GBS2A_626218088	7.0	16.8	-0.1
		FD_BLUP	GBS2A_460238480:GBS2A_570889235	5.4	13.9	0.0
	QGw.hwwg-2DS	Yld_GH2015F	KASP-Ppd-D1:KASP2D58574820	10.1	28.9	-0.1
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	3.3	11.6	0.0
		Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	4.1	10.2	0.0
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	3.1	9.5	0.0
GL	*QGl.hwwg-2AL*	Yld_GH2015S	GBS2A_690424019:GBS2A_694523139	7.6	15.8	-0.1
		Yld_GH2015F	GBS2A_626218088:GBS2A_690424019	11.7	16.5	-0.2
		GH_BLUP	GBS2A_570889235:GBS2A_626218088	9.3	17.5	-0.1
		Yld_AB2020S	GBS2A_570889235:GBS2A_626218088	12.1	21.4	-0.1
		Yld_AB2021S	GBS2A_690424019:GBS2A_694523139	14.2	30.2	-0.2
		FD_BLUP	GBS2A_570889235:GBS2A_626218088	9.3	16.5	-0.1
	QGl.hwwg-2DS	Yld_GH2015F	KASP-Ppd-D1:KASP2D58574820	16.1	25.3	-0.2
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	8.0	14.8	-0.1
		Yld_AB2020S	KASP2D58404016:KASP2D64237023	8.1	12.7	-0.1
		FD_BLUP	KASP2D58404016:KASP2D64237023	6.8	11.3	-0.1
	QGl.hwwg-3AL	Yld_GH2015S	GBS3A_621315736:GBS3A_623594682	5.7	11.4	0.1
		Yld_GH2015F	GBS3A_623594682:GBS3A_553116586	7.8	10.1	0.1
		GH_BLUP	GBS3A_621315736:GBS3A_623594682	8.0	14.1	0.1
		Yld_AB2020S	GBS3A_623594682:GBS3A_553116586	10.4	17.4	0.1
		Yld_AB2021S	GBS3A_623594682:GBS3A_553116586	6.4	12.1	0.1
		FD_BLUP	GBS3A_621315736:GBS3A_623594682	11.3	20.8	0.1
	QGl.hwwg-5AL	Yld_AB2020S	GBS5A_496548447:GBS5A_480971991	4.4	6.5	0.1
		FD_BLUP	GBS5A_503422205:GBS5A_496548447	3.9	6.2	0.1

	QGl.hwwg-6BL	Yld_GH2015F	GBS6B_692199975:GBS6B_696390689	3.5	4.8	-0.1
		Yld_AB2021S	GBS6B_692199975:GBS6B_696390689	4.2	8.2	-0.1
HT	*QHt.hwwg-2DS.1*	FHB_GH2019S	SSR2433:KASP2D26715133	5.2	16.7	2.9
		FHB_GH2019F	KASP2D23860723:SSR2062	4.9	9.8	2.9
		FHB_GH2019W	SSR2062:KASP2D25112886	6.3	14.3	3.1
		GH_BLUP	KASP2D23860723:SSR2062	7.2	12.2	2.0
		FHB_RF2020S	SSR2031:KASP2D23071583	16.0	25.3	4.9
		Yld_AB2020S	SSR2433:KASP2D26715133	9.3	15.6	3.5
		Yld_AB2021S	Xgwm261:Xwmc503	3.3	6.4	2.5
		FD_BLUP	Xgwm261:Xwmc503	7.1	9.1	2.3
	QHt.hwwg-2DS.2	FHB_GH2019F	KASP2D35014114:KASP-Ppd-D1	18.8	48.9	6.4
		FHB_GH2019W	KASP-Ppd-D1:KASP2D58574820	14.1	38.6	5.0
		GH_BLUP	KASP2D35014114:KASP-Ppd-D1	15.6	30.9	3.2
		FHB_RF2020S	KASP2D35014114:KASP-Ppd-D1	14.5	22.0	4.6
		Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	17.0	36.5	5.4
		Yld_AB2021S	PH2DS2198-11:KASP2D30932191	17.0	43.3	6.6
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	22.1	38.6	4.8
	QHt.hwwg-2DL	FHB_GH2020S	GBS2D_565228160:GBS2D_590348791	5.9	14.0	-2.8
		GH_BLUP	GBS2D_565228160:GBS2D_590348791	3.7	8.2	-1.7
		FHB_RF2020S	GBS2D_565228160:GBS2D_590348791	4.4	7.4	-2.7
		Yld_AB2020S	GBS2D_565228160:GBS2D_590348791	5.1	9.2	-2.7
		FD_BLUP	GBS2D_565228160:GBS2D_590348791	7.2	9.6	-2.4
	QHt.hwwg-3AL.1	GH_BLUP	GBS3A_497445629:GBS3A_441099949	3.9	6.3	1.5
		FHB_RF2020S	GBS3A_497445629:GBS3A_441099949	7.7	10.6	3.2
		Yld_AB2020S	GBS3A_497445629:GBS3A_441099949	5.2	7.9	2.5

	QHt.hwwg-3AL.2	FHB_GH2020S	GBS3A_623594682:GBS3A_553116586	5.5	11.0	2.5
		FD_BLUP	GBS3A_553116586:GBS3A_559105145	6.7	8.3	2.2
	QHt.hwwg-6BL	FHB_GH2019W	GBS6B_479253951:GBS6B_484276412	3.5	7.4	-2.2
		GH_BLUP	GBS6B_479253951:GBS6B_484276412	5.3	8.7	-1.8
		FD_BLUP	GBS6B_664175992:GBS6B_479253951	3.2	3.8	-1.5
HD	*QHd.hwwg-2DS*	FHB_GH2019S	KASP-Ppd-D1:KASP2D58574820	39.1	66.0	5.5
		FHB_GH2019F	KASP-Ppd-D1:KASP2D58574820	53.2	80.6	13.7
		FHB_GH2019W	KASP-Ppd-D1:KASP2D58574820	66.8	41.3	15.1
		FHB_GH2020S	KASP2D35014114:KASP-Ppd-D1	35.9	67.3	5.1
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	58.8	84.4	8.6
		FHB_RF2020S	KASP-Ppd-D1:KASP2D58574820	38.3	65.1	4.4
		Yld_AB2020S	KASP-Ppd-D1:KASP2D58574820	27.1	60.0	2.6
		Yld_AB2021S	KASP-Ppd-D1:KASP2D58574820	31.8	65.7	4.7
		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	44.1	72.9	3.4
	QHd.hwwg-7DS	FHB_RF2020S	GBS7D_73324992:GBS7D_60510704	4.2	4.9	1.2
		FD_BLUP	GBS7D_73324992:GBS7D_60510704	4.2	4.1	0.8
SL	*QSl.hwwg-2DS.1*	FHB_GH2019S	SSR2433:KASP2D26715133	7.6	11.6	0.4
		GH_BLUP	SSR2433:KASP2D26715133	6.1	6.2	0.3
		Yld_AB2020S	SSR2062:KASP2D25112886	4.6	10.8	0.3
	QSl.hwwg-2DS.2	FHB_GH2019S	KASP-Ppd-D1:KASP2D58574820	21.2	45.5	0.8
		FHB_GH2019F	KASP-Ppd-D1:KASP2D58574820	31.0	57.9	1.6
		FHB_GH2019W	KASP-Ppd-D1:KASP2D58574820	29.8	55.1	1.7
		FHB_GH2020S	KASP-Ppd-D1:KASP2D58574820	28.7	59.7	0.9
		GH_BLUP	KASP-Ppd-D1:KASP2D58574820	36.5	61.4	1.1
		FHB_RF2020S	KASP-Ppd-D1:KASP2D58574820	11.9	27.2	0.5

		FD_BLUP	KASP-Ppd-D1:KASP2D58574820	24.5	30.8	0.5
	QSl.hwwg-3AL.1	FHB_GH2019S	GBS3A_568343551:GBS3A_522003888	4.8	7.1	0.3
		Yld_AB2020S	GBS3A_568343551:GBS3A_522003888	3.3	6.9	0.2
		FD_BLUP	GBS3A_568343551:GBS3A_522003888	4.4	3.7	0.2
	QSl.hwwg-3AL.2	GH_BLUP	GBS3A_648390973:GBS3A_659255761	4.4	4.1	0.3
		FHB_RF2020S	GBS3A_648390973:GBS3A_659255761	5.2	8.7	0.3
	QSl.hwwg-6BS	FHB_RF2020S	GBS6B_41423615:GBS6B_51230810	3.5	7.0	-0.2
		FD_BLUP	GBS6B_41423615:GBS6B_51230810	4.0	3.4	-0.2
SC	*QSc.hwwg-2DS*	FHB_GH2019S	SSR2433:KASP2D26715133	15.7	31.7	-0.1
		FHB_GH2019F	SSR2433:KASP2D26715133	7.8	18.8	-0.1
		FHB_GH2019W	KASP2D25112886:KASP2D25113784	15.2	33.5	-0.2
		FHB_GH2020S	SSR2411:SSR2433	5.7	11.0	0.0
		GH_BLUP	SSR2433:KASP2D26715133	8.5	20.0	-0.1
		FHB_RF2020S	SSR2411:SSR2433	11.5	22.2	-0.1
		Yld_AB2020S	KASP2D25112886:KASP2D25113784	17.1	34.2	-0.1
		FD_BLUP	SSR2411:SSR2433	10.5	22.9	-0.1
	QSc.hwwg-3AL	FHB_GH2019S	GBS3A_621315736:GBS3A_623594682	5.0	8.2	-0.1
		FHB_GH2020S	GBS3A_623594682:GBS3A_553116586	4.4	9.4	0.0
		GH_BLUP	GBS3A_621315736:GBS3A_623594682	5.2	11.2	0.0
		FD_BLUP	GBS3A_623594682:GBS3A_553116586	3.4	7.6	0.0
	QSc.hwwg-7BL	FHB_GH2019W	GBS7B_608781847:GBS7B_523416506	5.3	9.7	-0.1
		FHB_GH2020S	GBS7B_608781847:GBS7B_523416506	7.7	15.4	-0.1
		GH_BLUP	GBS7B_608781847:GBS7B_523416506	7.5	16.7	-0.1
		FHB_RF2020S	GBS7B_608781847:GBS7B_523416506	7.9	13.9	-0.1
		Yld_AB2020S	GBS7B_608781847:GBS7B_523416506	7.6	12.6	-0.1

	FD_BLUP	GBS7B_608781847:GBS7B_523416506	7.6	15.6	0.0
QSc.hwwg-7DS	GH_BLUP	GBS7D_179816940:GBS7D_106112957	4.0	8.4	0.0
	FHB_RF2020S	GBS7D_179816940:GBS7D_106112957	3.8	6.3	0.0

FHB, Fusarium head blight; FDK, *Fusarium* damaged kernel; DON, deoxynivalenol; SNS, spikelet number per spike; TGW, thousand grain weight; KNS, kernel number per spike; HD, heading date; HT, plant height; SL, spike length; SC, spike compactness; GA, grain area; GW, grain width; GL, grain length; GH, greenhouse; FD, field; BLUP, best linear unbiased predictions; RF, rocky ford; AB, ashland bottoms; Yld, yield; S, spring; F, fall; W, winter; LOD, logarithm of odds value; PVE, the phenotypic variation explained by a QTL; Add, additive effect in which a positive value indicates beneficial allele contributed by G97252W.

Table 2.8 Quantitative trait locus (QTL) of Fusarium head blight (FHB) traits using corrected best linear unbiased prediction (BLUP) values by masking confounding effects of heading date (HD) and plant height (HT).

Trait	QTL	Experiment	Interval	LOD	PVE (%)	Add
FHB severity	*QFhb.hwwg-2DS*	GH_corrected_BLUP	KASP-Ppd-D1:KASP2D58574820	9.2	28.9	-0.045
	QFhb.hwwg-2DS	FD_corrected_BLUP	PH2DS2198-11:KASP2D30932191	4.1	11.7	-0.02
DON	*QDon.hwwg-2DS*	FD_corrected_BLUP	KASP2D30932191:KASP2D34456351	5.2	17.1	-3.027

FHB, Fusarium head blight; DON, deoxynivalenol; GH, greenhouse; FD, field; LOD, logarithm of odds value; PVE, the phenotypic variation explained by a QTL; Add, additive effect in which a positive value indicates beneficial allele contributed by G97252W;

Table 2.9 Quantitative trait locus (QTL) clusters for different traits on chromosome 2A, 2D, 3A and 6B.

Cluster	Trait	QTL	Dataset	Interval	Donor
2AL	KNS	*QKns.hwwg-2AL*	2	GBS2A_460238480:GBS2A_626218088	G97252W
	TGW	*QTgw.hwwg-2AL*	3	GBS2A_460238480:GBS2A_626218088	G97380A
	GA	*QGa.hwwg-2AL*	4	GBS2A_460238480:GBS2A_690424019	G97380A
	GW	*QGw.hwwg-2AL*	2	GBS2A_460238480:GBS2A_626218088	G97380A
	GL	*QGl.hwwg-2AL*	6	GBS2A_570889235:GBS2A_694523139	G97380A
2DS-1	HT	*QHt.hwwg-2DS.1*	8	Xgwm261:KASP2D26715133	G97252W
	SL	*QSl.hwwg-2DS.1*	3	SSR2062:KASP2D26715133	G97252W
	SC	*QSc.hwwg-2DS*	8	KASP2D25112886:KASP2D26715133	G97380A
	FHB severity	*QFhb.hwwg-2DS*	6	KASP2D35014114:KASP2D58574820	G97252W
	FDK	*QFdk.hwwg-2DS*	1	KASP2D35014114:KASP-Ppd-D1	G97252W
	DON	*QDon.hwwg-2DS*	1	KASP2D35014114:KASP-Ppd-D1	G97252W
	SNS	*QSns.hwwg-2DS*	9	KASP2D35014114:KASP2D58574820	G97252W
2DS-2	KNS	*QKns.hwwg-2DS*	3	KASP-Ppd-D1:KASP2D58574820	G97252W
	TGW	*QTgw.hwwg-2DS*	4	KASP2D35014114:KASP2D58574820	G97380A
	GA	*QGa.hwwg-2DS*	4	KASP2D35014114:KASP2D58574820	G97380A
	GW	*QGw.hwwg-2DS*	4	KASP-Ppd-D1:KASP2D58574820	G97380A
	GL	*QGl.hwwg-2DS*	4	KASP-Ppd-D1:KASP2D64237023	G97380A
	HT	*QHt.hwwg-2DS.2*	7	KASP2D35014114:KASP2D58574820	G97252W
	HD	*QHd.hwwg-2DS*	9	KASP2D35014114:KASP2D58574820	G97252W
	SL	*QSl.hwwg-2DS.2*	7	KASP-Ppd-D1:KASP2D58574820	G97252W
3AL	TGW	*QTgw.hwwg-3AL*	4	GBS3A_621315736:GBS3A_659255761	G97252W
	GA	*QGa.hwwg-3AL*	5	GBS3A_553116586:GBS3A_667967807	G97252W

GL	*QGl.hwwg-3AL*	6	GBS3A_553116586:GBS3A_623594682	G97252W
HT	*QHt.hwwg-3AL.2*	2	GBS3A_553116586:GBS3A_623594682	G97252W
SL	*QSl.hwwg-3AL.1*	3	GBS3A_522003888:GBS3A_568343551	G97252W
SL	*QSl.hwwg-3AL.2*	2	GBS3A_648390973:GBS3A_659255761	G97252W
SC	*QSc.hwwg-3AL*	4	GBS3A_553116586:GBS3A_623594682	G97380A

FHB, Fusarium head blight; FDK, *Fusarium* damaged kernel; DON, deoxynivalenol; SNS, spikelet number per spike; TGW, thousand grain weight; KNS, kernel number per spike; HD, heading date; HT, plant height; SL, spike length; SC, spike compactness; GA, grain area; GW, grain width; GL, grain length; Dataset indicates number of repeated QTL; Donor, parents providing beneficial allele.

Table 2.10 Comparison of best linear unbiased prediction (BLUP) values of phenotype between different recombinant inbred line (RIL) groups with contrasting alleles at *QFhb.hwwg-2DS* and *QTgw.hwwg-3AL*.

Environment	Genotype	PSS (%)	TGW (g)	SNS	KNS	HT (cm)	HD
Greenhouse	2DS(+)3A(+)	63.8	29.3	19.6	NA	78.8	113.1
	2DS(+)3A(-)	63.9	27.98**	19.3	NA	73.9**	115.4*
	2DS(-)3A(+)	85.7**	30.3*	16**	NA	67.8**	98.1**
	2DS(-)3A(-)	86.6**	29.1	15.9**	NA	66.1**	97.5**
Field	2DS(+)3A(+)	75.7	29.5	21.6	47.2	100.2	207.8
	2DS(+)3A(-)	69.6	28.9	20.9*	45.1	93.8**	206.7*
	2DS(-)3A(+)	84.3*	30.1	19.2**	42.4**	84.5**	201.4**
	2DS(-)3A(-)	84.3*	29.1	19.1**	42.3**	80.3**	200.5**

PSS, Percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; TGW, thousand grain weight; KNS, kernel number per spike; NA, not available data; g, gram; cm, centimeter; 2DS(+) and 2DS(-) refer to the G97252W allele and G97380A allele, respectively, at QFhb.hwwg-2DS; 3AL(+) and 3AL(-) refer to, G97252W allele and G97380A allele, respectively, at QTgw.hwwg-3AL; All the statistical analyses were compared to the group 1 with genotype 2DS(+)3AL(+); * means significant at $p = 0.05$; ** means significant at $p = 0.01$.

Table 2.11 Physical positions of quantitative trait locus (QTL) flanking single nucleotide polymorphisms (SNPs) based on Chinese Spring v2.1 reference genome.

SNP name	Flanking sequence	Physical location (bp)
GBS2A_460238480	GCTATGGAGCTGGAGAGGCA[T/A]CGGAGTTGCTGATGA	chr2A:460238480
GBS2A_570889235	ACACGTACGCATCCACACAC[A/T]CTCTCTCTCTTCATC	chr2A:570889235
GBS2A_626218088	CCAATTCACGTACAGGGTAC[A/C]AGTAGCACTCCAAAC	chr2A:626218088
GBS2A_690424019	GGGACCCTGAGGGTGCCCTA[C/T]GAGTCCCTCGGCACG	chr2A:690424019
GBS2A_694523139	TCTGCAGTGGCATGCGCATG[A/G]CGAGACGCCGAGGTG	chr2A:694523139
GBS2D_80882808	CAGAGTCCTGCAGGCTCACC[G/A]ACGTCTTGAAGGCGA	chr2D:80882808
GBS2D_565228160	TCCTGTGCATGATATGTTTC[C/G]CTGTTGCTCGCCGCA	chr2D:565228160
GBS2D_590348791	CCATGGGAGAAGGTGTTTGT[A/C]CGTTGGTCGATCGAA	chr2D:590348791
GBS3A_441099949	GGCTCCACTATGTCTTCTCT[C/T]CTCTTGTGCAGGCAG	chr3A:441099949
GBS3A_497445629	GACGCCGTGGCCTACCTCGG[C/T]GCCCCCGTCACGGAC	chr3A:497445629
GBS3A_522003888	TGTTGCAGCTGGCACGTCCG[C/T]GTGCGTGTGCATGCC	chr3A:522003888
GBS3A_553116586	TCTGCAGAATCGAGCCGTAG[G/A]CTCCTCTGCCCCTTC	chr3A:553116586
GBS3A_559105145	CGGCTGCAGGCGGGACCACG[C/T]GCGTGGGAGTGGACG	chr3A:559105145
GBS3A_568343551	GAGCACACAATATGACCATT[G/T]CGCTGCTTCACCTTC	chr3A:568343551
GBS3A_621315736	CAGCGTGAAGAGCTCCGCCA[T/C]GAGCAGCAGCCACAC	chr3A:621315736
GBS3A_623594682	CAACTACGACCAAGCATCGA[T/C]CGAAACTGACAAAAG	chr3A:623594682
GBS3A_648390973	GATCCATTGTTCTTGTCGTC[G/A]TTGGGCTGGATTGGT	chr3A:648390973
GBS3A_655968576	CGGGCGGTTCGGTGGCAACG[T/C]GGAGAGGTTCCGAGG	chr3A:655968576
GBS3A_659202246	GTAGCCGTGCTTCTGCACCG[T/A]GCTGTCGTGGTGGAC	chr3A:659202246
GBS3A_659255761	GTTCCTGGAGATGAGCTGGC[C/T]GCGCTGCAGCTTTGT	chr3A:659255761
GBS3A_667967807	CGTCGTGAGACCACCGATGG[G/T]TGATGTGCTGCAGGT	chr3A:667967807
GBS4B_15729471	GTGTGTGTGTGCGCGCGGTA[A/G]CTTTTCACATTTCCA	chr4B:15729471

GBS4B_18531114	CCGCATCTGCCAAGACGGGA[G/C]TCACGCAGTCACACT	chr4B:18531114
GBS4B_20496100	CCATCATTCATTCATTCCTT[C/G]GCTGAACCTCTGCTT	chr4B:20496100
GBS5A_480971991	CCACCCCACGTACCACTTGG[T/C]AGTCGGACAATTGGC	chr5A:480971991
GBS5A_496548447	TGTCACTTACGTCGGTGCGA[G/A]GAACACCAGGAAGGA	chr5A:496548447
GBS5A_503422205	GCAGGGACCAAAACACCACA[T/C]ATATAGATGTGCGCC	chr5A:503422205
GBS6B_41423615	CGACGCGCTGCAGAGCTAGG[C/T]GGACGCCATCGCCGC	chr6B:41423615
GBS6B_51230810	GTTGCTGCGTGGTGATGACC[A/G]CTGGTGCACTGCAGC	chr6B:51230810
GBS6B_479253951	CGCGACAAGAGGAGAGGCAT[A/T]GTGGGGCTTGGCCAG	chr6B:479253951
GBS6B_484276412	CGCTACCGCGTCCGCCTTAA[G/A]AACTCCGCAGACGCA	chr6B:484276412
GBS6B_643333379	AGTGCGCCTGGCTGTACCCT[C/G]CTGACACCAATATCC	chr6B:643333379
GBS6B_664175992	CAGCGTCGAGGGAAGCTACA[A/T]CGCGCAACCAGCGAC	chr6B:664175992
GBS6B_692199975	AAAGATCATGTGTTTACCAA[T/C]GAGAGACGGCTCAAA	chr6B:692199975
GBS6B_692200005	CTCAAACACTCTTTTGAAAA[G/C]TCTGATCAGTGCCCT	chr6B:692200005
GBS6B_696390689	ATTTCAGCCAGCCACCGCAT[C/G]ACCCTAGCGGTGGAT	chr6B:696390689
GBS7A_726405117	TGCTGTCGTGCTCGCTACCT[C/T]GTCGAGGGCCATGAT	chr7A:726405117
GBS7A_730488230	ACGAGCCTATAGAACAGATC[T/C]TGTTCAAGTAAGGTC	chr7A:730488230
GBS7A_731570297	CCTGTTAAGAAAAACCATGT[G/C]TGCGGTACCGAACTC	chr7A:731570297
GBS7A_733827431	GATCCGAGGAGCCGTGGCTT[C/T]GGTGCAGCTTGGTGC	chr7A:733827431
GBS7B_523416506	GCCTCCTTGTGTGATCTAAT[T/C]GATGCTCTAGTGCTC	chr7B:523416506
GBS7B_608781847	GCCCCCTCTGCAGTGTCTCC[A/G]CGCGACCCACCCCGA	chr7B:608781847
GBS7D_60510704	GCAGCCAACATCTTTGAACC[A/G]AACCGAGCAGCGGAG	chr7D:60510704
GBS7D_73324992	TAAGCTTCTCTAGCTTTGGT[C/T]TTGCTCCTTCTCCAA	chr7D:73324992
GBS7D_106112957	CTGCAGTGGATCATGGCGAA[T/C]TTGGTGAAGCATCTG	chr7D:106112957
GBS7D_179816940	TATTTGGGATCATCGTGCAT[T/A]CTAGGTCCAGCCTGC	chr7D:179816940

Chapter 3 - Characterization of QTL for FHB resistance and agronomic traits in a hard winter wheat population derived from Jagger

3.1 Introduction

Wheat (*Triticum aestivum*) is one of the most important staple crops worldwide yielding over 760 million tons annually (Singh et al. 2023). Pathogens and pests account for about 21.5% of wheat losses annually and threaten global wheat production and food security. In North America, diseases reduce approximate 17.91% of wheat yield. FHB, also called scab, is one of the most devastating diseases of wheat, which resulted in 3.20% losses in this region (Savary et al. 2019). The U.S. suffered a total loss of $7.6 billion attributable to FHB damage between 1993 and 2001 (McMullen et al. 2012). FHB is primarily caused by *Fusarium graminearum* in North America. The infected wheat heads displayed premature senescence before harvesting. The infected kernels usually become shriveled, discolored and contaminated with mycotoxins, which resulted in reduced grain yield and quality. These mycotoxins pose food safety risk and health hazard to human and animals by causing immunological, teratogenic problems and feed refusal (McMullen et al. 2012).

Using resistant wheat varieties is the most effective and environment friendly approach to combat FHB damage. Generally, wheat breeders usually improve wheat FHB resistance by introducing exotic and alien resistance or utilizing native resistance from locally adapted wheat germplasm (McMullen et al. 2012). Exotic and alien resistance sources are often not adapted to local environment and usually associated with unfavorable agronomic traits, such as poor grain quality, shattering, tall plant height, reduced yield potential

and susceptibility to other disease (Kang et al. 2011; Brar et al. 2019). It is always necessary and required to continuously discover new sources of resistance, especially native resistance which are easier to be used in breeding than exotic sources due to their better adaptability and agronomic performances under local environments. Many previously reported FHB resistance QTL are often adversely associated with plant HT, HD, SC and yield component traits including TGW, SNS and KNS (Buerstmayr et al. 2020; Hu et al. 2023). Dissecting the genetic relationship between FHB resistance and agronomic traits will facilitate development of new high-yielding FHB resistant wheat cultivars. Jagger has been a locally adapted hard winter wheat with excellent agronomic performances in the central and southern Great Plains and shows moderately susceptibility to FHB. We screened an ethyl methanesulfonate (EMS) induced mutant population developed from 'Jagger' (Rawat et al. 2019) and identified several FHB resistant lines. 'JagR1097' is one of the mutants identified with a high level of resistance to FHB. We developed a RIL population from the cross of JagR1097 x Jagger to map the QTL for FHB resistance in JagR1097. The objectives of current study are to (1) investigate the genetic relationship between FHB resistance and agronomic traits, (2) characterize the genetic architecture of FHB resistance and agronomic traits in Jagger (3) develop tightly linked molecular markers for native FHB resistance QTL.

3.2 Materials and methods

3.2.1 Plant materials

Jagger (PI 593688) is a hard winter wheat variety from Kansas and shows moderately susceptibility. JagR1097 is a mutant that was identified from the Jagger EMS-mutant population with moderate FHB resistance. It also showed significant phenotypic differences

in agronomic traits from Jagger including plant HT, HD, SL, SC, SNS, TGW, GA, GW and GL (Table 3.1). A population of 149 $F_{5:8}$ RILs was developed from the cross between JagR1097 and Jagger by single seed descent (SSD) and used for QTL analysis in this study.

3.2.2 Evaluation of FHB and agronomic traits in greenhouse experiments

Two parents and the RILs were evaluated for type II FHB resistance in four greenhouse experiments in 2018 spring (FHB_GH2018S) and fall (FHB_GH2018F), 2019 spring (FHB_GH2019S) and winter (FHB_GH2019W) at Kansas State University using a randomized complete block design with one, two, three and two replications, respectively. This RIL population was also evaluated for the kernel traits including TGW, GW, GL and GA in other two separate greenhouse experiments in fall 2018 (Yld_GH2018F) and spring 2019 (Yld_GH2019S). The wheat plant management and trait measurement were the same as described in chapter 2.2.2.

3.2.3 Linkage map construction and QTL analysis

DNA extraction, SNP genotyping and QTL mapping were the same as described in chapter 2.2.4 & 2.2.5. Totally, 773 GBS-SNPs were used for linkage map construction using IciMapping 4.1 software and a minimum LOD value of 5.0. The linkage map was used for QTL analysis. All QTL names started with ‘Q’, followed by a trait designator, a hyphen (-) and a symbol for the chromosome or chromosome arm where the QTL was located. if more than one QTL were identified for a certain trait in the same chromosome, a serial number (1, 2, 3, etc.) was added after the chromosome name to show their order from the short arm to the long arm of the chromosome.

3.2.4 Conversion of SNPs to KASP markers

The GBS-SNPs within the major QTL interval for FHB resistance were converted to KASP assays (https://biosearch-cdn.azureedge.net/assetsv6/kasp-explanation-fact-sheet.pdf) using the same method described in chapter 2.2.6.

3.2.5 Statistical analysis

The same software, models and methods as chapter 2.2.7 were used for statistical analysis in this study.

3.3 Results

3.3.1 Phenotypic variation and correlations for FHB resistance and agronomic traits

PSS showed negative correlations with HD, SNS and SL ($-0.45 < r < -0.43$, $p < 0.01$), but had no significant correlation with HT, SC and kernel traits (TGW, GA, GW and GL), implying that FHB resistant lines in the population usually showed later HD, greater SNS and longer SL than FHB susceptible lines in the population under the greenhouse environments (Table 3.2).

ANOVA showed that effects of genotypes (G), environments (E) and the G x E interactions were significant ($p < 0.01$) for all traits investigated in the JagR1097 x Jagger RIL population (Tables 3.3 & 3.4). The continuous distributions of the BLUP values were observed for PSS, HT, HD, SL, SC, TGW, SNS, GA, GW, and GL in the RIL population (Figure 3.1). The heritability was high for both PSS (0.81) and agronomic traits (0.85 – 0.97) based on BLUP values (Tables 3.3 & 3.4), indicating a large portion of the variance for these traits was inheritable.

All the genetic and non-genetic effects were significant ($p < 0.01$) for PSS in greenhouse after masking confounding effects of HD and HT (Table 3.5). The heritability of PSS was significantly reduced after covariate analysis (Tables 3.3 & 3.5), which indicates that HD and HT had confounding effects on PSS in the greenhouses.

3.3.2 Genetic linkage map construction

A total of 2,717 SNPs were generated from GBS on the 149 RILs and their parents after removal of SNPs with >20% missing datapoints, minor allele frequency (MAF) <0.20 and heterozygosity >10%. These GBS-SNPs were further clustered into 774 bins, and SNPs with the least missing data points in each bin were chosen for constructing genetic linkage map. The final linkage map consists of 750 bin SNPs in 40 linkage groups that covered a total genetic distance of 2,370.90 cM at an average marker density of 3.16 cM per bin marker (Table 3.6). The 40 linkage groups were anchored to 21 wheat chromosomes based on their physical positions with the most SNPs (90) on 5A spanning 182.05 cM and the least SNPs (11) on chromosome 3D spanning 103.05 cM.

3.3.3 QTL for FHB resistance

Two QTL were mapped for FHB resistance on chromosomes 4AL and 6AL (Table 3.7). *QFhb-4AL* was a major QTL for FHB resistance on chromosome arm 4AL, flanked by *K4A685473955* and *GBS4A690563166* in two greenhouse experiments and one BLUP dataset. This QTL explained 10.34% to 15.79% of the phenotypic variation for PSS. *QFhb-6AL* with a minor effect on FHB resistance on chromosome arm 6AL between *GBS6A540881333* and *GBS6A543690537* was significant in one greenhouse experiment and one BLUP dataset, which explained 7.51% and 6.53% of PSS variation respectively. Jagger carries the resistance allele at *QFhb-4AL*, but susceptibility allele at *QFhb-6AL* (Table 3.7).

The effects of QTL *QFhb-4AL* and *QFhb-6AL* on FHB severity remained similar, 11.80% and 6.23% of the phenotypic variation for PSS, respectively, after HD and HT were used as covariate factors (Table 3.8). Interestingly, a new QTL (*QFhb-4DL*) with a minor effect (PVE= 6.34%) was discovered on chromosome arm 4DL after removal of the effects of HD and HT (Table 3.8). Jagger contributes the resistance allele at *QFhb-4AL*, but susceptible alleles at *QFhb-4DL* and *QFhb-6AL*.

3.3.4 QTL for yield-related traits

Four QTL were detected for SNS (Table 3.7). *QSns-4AL* showed the largest effect in four greenhouse experiments and the BLUP dataset, explained 20.66 to 43.65% of the phenotypic variation. *QSns-5DL* explained 9% to 15.60% of the SNS variation in two greenhouse experiments and the BLUP dataset. *QSns-7AL* explained 7.16% to 11.53% of the phenotypic variation in three greenhouse experiments and the BLUP dataset. *QSns-2BS* explained 4.05% and 6.47% of the phenotypic variation in one greenhouse experiment and the BLUP dataset. Jagger contributes increased SNS alleles at *QSns-4AL* and *QSns-5DL*, but decreased SNS alleles at *QSns-2BS and QSns-7AL*.

Two QTL were significant for TGW (Table 3.7). *QTgw-4BS* explained 8.74 to 29.12% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. Jagger contributes low TGW alleles at both loci. *QTgw-3AS* explained 8.08% and 10.36% of the phenotypic variation in one greenhouse experiment and the BLUP dataset.

Three QTL were detected for GA (Table 3.7). *QGa-4BS* showed the largest effect explaining 11.67 to 28.33% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. *QGa-5AL* explained 10.25% and 9.77% of the GA variation in one greenhouse experiment and the BLUP dataset. *QGa-3AS* explained 9.41% and 8.37% of the phenotypic variation in one greenhouse

experiment and the BLUP dataset, respectively. Jagger contributes small GA alleles at *QGa-4BS* and *QGa-3AS*, but large GA allele at *QGa-5AL*.

Two QTL were detected for GW (Table 3.7). *QGw-4BS* explained 9.69 to 23.87% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. *QGw-5AS* explained 12.66% and 13.69% of the phenotypic variation in one greenhouse experiment and the BLUP dataset. Jagger contributes the narrow grain allele at *QGw-4BS*, but the wide grain allele at *QGw-5AS*.

Five QTL were detected for GL (Table 3.7). *QGl-2DL* was a major QTL explaining 14.85 to 15.79% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. *QGl-4BS* explained 9.20 to 16.70% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. *QGl-4DS* explained 3.91 to 17.22% of phenotypic variation in two greenhouse experiments and the BLUP dataset. *QGl-5BL* explained 6.49 to 8.63% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. *QGl-1DL* explained 8.22% and 5.89% of the phenotypic variation in one greenhouse experiment and the BLUP dataset, respectively. Jagger contributes the short grain alleles at *QGl-1DL*, *QGl-4BS* and *QGl-4DS*, but long grain alleles at *QGl-2DL* and *QGl-5BL*.

3.3.5 QTL for other traits

Five QTL were mapped for plant HT on chromosome 3AL, 4AL, 4BS, 5AS and 5DL (Table 3.7). *QHt-4BS* close to *Rht-B1* showed the largest effect explaining 8.95 to 42.28% of the phenotypic variation and was significant in four greenhouse experiments and the BLUP dataset. *QHt-4AL overlapping with QFhb-4AL* was significant in two greenhouse experiments and the BLUP dataset, explaining 5.16 to 12.02% of the phenotypic variation. *QHt-3AL* explained 5.03 to 10.07% of the phenotypic variation in three greenhouse experiments and the BLUP dataset. *QHt-5DL* explained 5.60 to 11.43% of phenotypic variation in two greenhouse

experiments and the BLUP dataset. *QHt-5AS* explained 10.55% and 4.38% of phenotypic variation in a greenhouse experiment and the BLUP dataset, respectively. Jagger contributes the tall alleles at *QHt-4AL*, *QHt-5DL*, but the short alleles at the other three QTL.

Four QTL on chromosome arms 2BS, 2DL, 4AL and 5DL were significant for HD in at least two greenhouse experiments (Table 3.7). Among them, *QHd-4AL* overlapping with *QFhb-4AL* is a major QTL significant in four greenhouse experiments and the BLUP dataset, explaining 10.94 to 21.69 % of the phenotypic variation. *QHd-5DL* also showed major effect and explained 11.77 to 24.83% of the phenotypic variation. *QHd-2BS* showed minor effect and explained 5.29 to 6.37% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. *QHd-2DL* explained 4.96 to 6.09% of the phenotypic variation in two greenhouse experiments and the BLUP dataset. JagR1097 contributes the late heading allele at only *QHd-2BS* and Jagger contributes the late heading alleles at other three loci.

Five QTL for SL were mapped on chromosome arms 2BL, 3DL, 4AL, 4BS and 5AS (Table 3.7). Jagger contributes the long spike alleles at *QSl-4AL* and *QSl-3DL*, but the short spike alleles at *QSl-2BL*, *QSl-4BS* and *QSl-5AS*. Five QTL for SC were detected on chromosome arms 2AS, 3DL, 5AS, 5DL and 7AL (Table 3.7). Jagger contributes the compactness alleles at *QSc-5AS* and *QSc-5DL*.

3.3.6 QTL clusters for multiple traits

A total of nine QTL clusters were discovered on chromosome arms 2BS, 2DL, 3AS, 3DL, 4AL, 4BS, 5AS, 5DL and 7AL in the mapping population (Table 3.9). The 4AL cluster is flanked by *K4A685473955* (685.47 Mb) and *GBS4A690563166* (690.56 Mb) based on IWGSC RefSeq v2.1 (Zhu et al. 2021). It contains overlapping QTL for FHB severity (*QFhb-4AL*), HT, HD, SNS and SL. The 4BS cluster is flanked by *K4B24978862* (24.98 Mb) and *K4B40019304* (40.02 Mb) contains QTL for HT, SL, TGW, GA, GW and GL. The

2BS cluster contains QTL for HD and SNS. The 2DL cluster contains QTL for HD and GL. The 3AS cluster contains QTL for TGW and GA. The 3DL cluster contains QTL for SL and SC. The 5AS cluster contains QTL for HT, SL and SC. The 5DL cluster contains QTL for HT, HD, SNS and SC. The 7AL cluster contains QTL for SNS and SC.

Jagger carries alleles for lower PSS, taller plant, later HD, more SNS and longer SL at the 4AL cluster (Table 3.9). JagR1097 contributes alleles for taller HT, longer SL, higher TGW and larger grain size (GA, GW and GL) at the 4BS cluster without adverse effects on FHB resistance. Similarly, JagR1097 contributes alleles for higher SNS at the cluster 2BS, 5DL and 7AL. Moreover, JagR1097 contributes allele for higher TGW without impact on FHB resistance at 3AS cluster. The different QTL at the same locations might be due to pleiotropy or closely genetic linkage.

3.4 Discussion

Inheritance of FHB resistance is a complex and quantitative trait, which is usually controlled by multiple genetic loci. Accurate phenotyping of FHB resistance is time-consuming and labor-intensive due to its extensive interaction with environment factors and agronomic traits, such as temperature, humidity, HD and HT. Characterizing the native FHB resistance QTL and developing high-throughput markers for MAS could facilitate the improvement of FHB resistance in breeding programs.

3.4.1 Genetic architecture of type II FHB resistance in Jagger x JagR1097 RIL population

HD and HT had significant effects on expression of FHB resistance, which may result in overestimation of FHB severity. In this study, we first mapped two native QTL for type II FHB resistance on 4AL and 6AL. To removal the confounding effects of HD and HT, we conducted covariate analysis (Table 3.7). These two QTL (*QFhb-4AL* and *QFhb-6AL*) were still significant for FHB resistance

after using corrected PSS values (Table 3.8), confirming these QTL were real and not due to HT and HD variation. Interestingly, an additional new QTL was discovered on 4DL (*QFhb-4DL*) for FHB resistance after masking the HD and HT effects (Table 3.8).

QFhb-4AL showed a major effect on FHB resistance explaining up to 15.79% of the phenotypic variation for PSS and mapped in a 5 Mb interval (685.47 - 690.56 Mb) between *K4A685473955* and *GBS4A690563166* based on IWGSC RefSeq v2.1 (Table 3.7). Previously, several studies reported one FHB resistance QTL between 649.59 Mb and 713.54 Mb genomic region on 4AL in different populations derived from three Canadian cultivars, 'AC Foremost', '86ISMN 2137' and 'FL62R1' (Yang et al. 2005; McCartney et al. 2016; Zhang et al. 2020). In addition, Ágnes et al. (2014) mapped one FHB resistance QTL in Brazilian wheat Frontana between *wPt-800509* (674.84 Mb) and *wPt-2780* from (723.45 Mb) on 4AL. Buerstmayr and Buerstmayr (2015) detected one FHB resistance QTL in a Swiss cultivar 'Arina' in a region between *wPt-2345* (689.73 Mb) and *wPt-4828* (752.46 Mb) on 4AL. These QTL were located in similar physical location as *QFhb-4AL* in current study (Table 3.7), and they are probably the same. Consistent detection of *QFhb-4AL* in diverse genetic backgrounds of American and European wheat germplasm indicates that *QFhb-4AL* is a stable major QTL and have already been deployed in many wheat cultivars in these regions. Flanking KASP markers have been developed for *QFhb-4AL* (Table 3.10) and can be used to pyramiding it with native resistance QTL in wheat breeding.

QFhb-6AL had a minor effect on FHB resistance explaining up to 7.51% of phenotypic variation in a 3 Mb interval (540.88 - 543.69 Mb) flanked by *GBS6A540881333* and *GBS6A543690537* (Table 3.7). Holzapfel et al. (2008) reported one FHB resistance QTL between *IWB44265* and *IWB10928* from 358.75 Mb to 545.82 Mb on chromosome arm 6AL in a French wheat cultivar 'Apache'. The 6AL QTL for FHB resistance were also mapped in the same 6AL region from 288.91 Mb to 583.12 Mb in one U.S. wheat variety 'NC-

Neuse' and two Canadian wheat cultivars, 'AC Brio' and 'AC Cadillac', respectively (Petersen et al. 2016; Malihipour et al. 2017; Berraies et al. 2023). These QTL were all mapped in similar physical positions based on their flanking markers and they are likely the same QTL in diverse germplasm (Table 3.7). However previous studies mapped the QTL in large intervals of 187 and 294 Mb, which is too large interval for using flanking markers to select the QTL in breeding. In this study, we mapped *QFhb-6AL* to the 3 Mb interval, which will facilitate marker-assisted selection and map-based clonig of this QTL.

Additionally, a new QTL (*QFhb-4DL*) was discovered for FHB resistance on 4DL after masking HD and HT effects (Table 3.8). *QFhb-4DL* showed a minor effect on FHB resistance explaining 6.34% of the PSS variation and was mapped in a ~25 Mb interval (456.25 - 481.52 Mb) flanked by *GBS4D456253756* and *GBS4D481515282* (Table 3.8). Srinivasachary et al. (2008) reported one FHB resistance QTL on chromosome arm 4DL flanked by *Xgwm192* and *Xgwm265* from 412.60 to 499.47 Mb in a UK wheat variety 'Spark'. Ma et al. (2006) mapped an FHB resistance QTL on 4DL between *Xwmc331* and *Xcfd84* from 453.54 to 498.70 Mb in a Chinese wheat germplasm 'CS-SM3-7AD'. Clinesmith et al. (2019) detected an FHB resistance QTL flanked by snp5725 (~455.84 Mb) on chromosome arm 4DL in a US wheat variety Everest. Based on the physical locations of those QTL, *QFhb-4DL* identified in this study is likely the same QTL as reported in Spark, CS-SM3-7AD and Everest.

3.4.2 Association of QTL for FHB resistance and agronomic traits

In this study, *QFhb-4AL* was mapped in the same locations with QTL for plant HT (*QHt-4AL*) and HD (*QHd-4AL*) between *K4A685473955* and *GBS4A690563166*, which implies that wheat FHB resistant lines had later heading date and taller plants in greenhouse environment (Table 3.9; Figures 3.2 & 3.3). This result confirmed several previous studies that the overlapping QTL have

been mapped in the interval from 649.60 Mb to 723.45 Mb on 4AL for FHB resistance, plant HT and HD (Ágnes et al. 2014; McCartney et al. 2016; Zhang et al. 2020). Additionally, two 4AL QTL for spikelet number per spike (*QSns-4AL*) and spike length (*QSl-4AL*) were also mapped within *QFhb-4AL* interval (Table 3.9; Figure 3.2). Previously, QTL for SNS and SL were reported in the same region as *QFhb-4AL* in Chinese wheat cultivar 'J411' that carries the positive allele (Fan et al. 2019; Li et al. 2022). These results indicate that *QFhb-4AL* is likely a QTL with pleiotropic effects on or tightly linkage to the QTL for agronomic traits and the Jagger alleles contribute to higher resistance, later heading date, taller plant height, longer spike and more SNS.

Rht-B1b is the most predominant semi-dwarfing gene deployed into modern wheat cultivars to reduce lodging and improve grain yield since the Green Revolution (Hedden 2003). R*ht-B1* locus was reported to be associated with agronomic traits and FHB resistance in previous studies (Srinivasachary et al. 2009; Buerstmayr et al. 2012; Lu et al. 2013; Liu et al. 2013; Prat et al. 2017; Xu et al. 2019; Song et al. 2023a). Xu et al. (2019) reported one major QTL for TGW on 4BS flanked by *Rht-B1* and *AX-89323611* between 33.61 Mb and 35.64 Mb associated with an haploblock deletion carrying three genes. Recently, Song et al. (2023a) cloned the candidate genes for this 4BS QTL for TGW and validated the function of *Rht-B1b* on agronomic traits. The loss function mutant of *Rht-B1b* displayed significant increase in plant HT, SL, TGW and grain size (GW, GL). In this study, one 4BS QTL were detected for plant HT (*QHt-4AL*), spike length (*QSl-4BS*), TGW (*QTgw-4BS*), GW (*QGw-4BS*), GL (*QGl-4BS*) and GA (*QGa-4BS*) between 24.98 Mb and 49.39 Mb on 4BS. Based on the physical position, *QHt-4BS* is most likely *Rht-B1* (Xu et al. 2019; Song et al. 2023a). Surprisingly, FHB resistance QTL was not detected at *Rht-B1* locus in this JagR1097 x Jagger RIL population. Previously, Zhang et al. (2018b) reported a similar result that *Rht-B1* locus had little effect on FHB resistance in a doubled haploid population from FL62R1 x Stettler under greenhouse

conditions. However, several studies reported colocalization of *Rht-B1* locus with FHB resistance QTL in the region and association of the short allele *Rht-B1b* with increased FHB susceptibility (Buerstmayr et al. 2012; Lu et al. 2013; Liu et al. 2013; Prat et al. 2017). In contrast, Srinivasachary et al. (2009) reported association of *Rht-B1b* with increased FHB resistance. Based on these studies reported to date, the *Rht-B1* may not show a pleiotropic effect on FHB resistance and a tightly linked gene to *Rht-B1* more likely conditions FHB resistance in this region. Further investigations may further dissect the association between plant HT and FHB resistance in this region.

3.4.3 Other QTL for grain yield component traits

In the current study, *QSns-2BS* was physically mapped between *GBS2B69937043* and *GBS2B147654222* from 69.94 Mb to 147.65 Mb. Hu et al. (2020) reported two QTL for SNS on 2BS in the same region as *QSns-2BS* in two Chinese cultivars. *QSns-5DL* was detected between *GBS5D473603213* and *GBS5D508721978* in 473.60 Mb to 508.72 Mb, respectively. Liu et al. (2006) discovered one QTL for SNS flanked by *WMC215* (475.21 Mb) overlapped with *QSns-5DL* identified in this study. *QSns-7AL* was located between *GBS7A676634402* and *GBS7A682575058* from 676.63 Mb to 682.58 Mb. Kuzay et al. (2019) reported one QTL between *AX-111159341* and *AX-109360122* from 678.62 Mb to 678.70 Mb, which is most likely the sane QTL as *QSns-7AL* detected in this study. *QTgw-3AS* was physically mapped between *GBS3A131021938* and *GBS3A164943473* from 131.02 Mb to 164.94 Mb, which is likely the same QTL as reported by Rathan et al. (2023).

3.4.4 Deployment of *QFhb-4AL* in local wheat breeding

Jagger was an excellent hard winter wheat variety with wide adaptation in the Great Plains. It is well known for its extensive adaptability, early maturity, high yield and quality (Sears et al. 1997; Rawat et al. 2019). Jagger is moderate susceptible to FHB (Table

3.1). Previous report identified two minor QTL on 2DS and 6DL for FHB resistance in Jagger (Cai and Bai 2014). In the current study, one novel major QTL was discovered for FHB resistance on 4AL in Jagger. At this 4AL locus, Jagger provides positive alleles for FHB resistance, SNS, HT and HD, but has no effect on TGW. Selecting Jagger allele at *QFhb-4AL* may provide higher resistance, more SNS, but taller HT and later HD without improvement in TGW (Figures 3.2 & 3.3; Table 3.9). Whereas JagR1097 allele at *QTgw-4BS* contributed to increased TGW without decrease in FHB resistance (Table 3.9), and this QTL is most likely *Rht-B1a*. Pyramiding *QFhb-4AL* with *QTgw-4BS* may be able to develop FHB resistant high-yielding wheat cultivars that adapt to North America regions. This pyramiding scheme may result in slightly taller plants with later maturity, but higher FHB resistance, more SNS and greater TGW for higher yield potential (Table 3.11). The KASP markers flanking the two loci can be used for marker-assisted selection (MAS) in breeding programs (Tables 3.10 & 3.12). However, tall and late plants can be the disadvantages in some wheat growing areas (Table 3.11). Recently, one natural haploblock deletion carrying *Rht-B1b* has been proposed for shaping semi-dwarf trait with improved grain yield via deleting *ZnF-B* in the absence of *Rht-B1b* (Song et al. 2023a). Based on the antagonistic effects between *ZnF-B* and *Rht-B1b*, one specific haplotype which carries *znf-b* and *Rht-B1a* can be created by knocking-out of *ZnF-B* in the *QTgw-4BS* interval to reduce plant height without grain yield reduction to develop novel FHB resistant high-yielding wheat varieties.

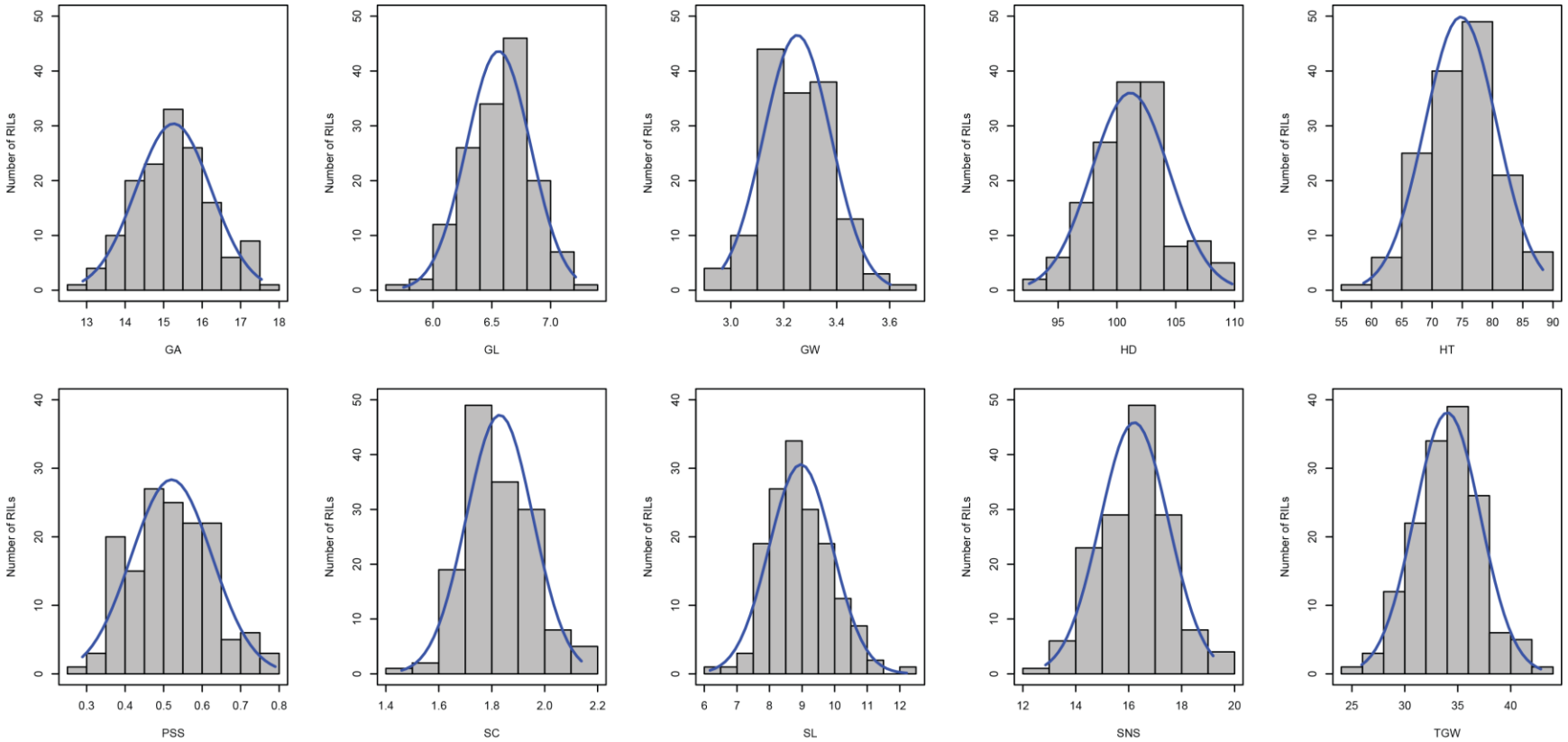

Figure 3.1 Distribution of best linear unbiased prediction (BLUP) values of Fusarium head blight (FHB) and agronomic traits in JagR1097 x Jagger population under greenhouse.

PSS, percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length.

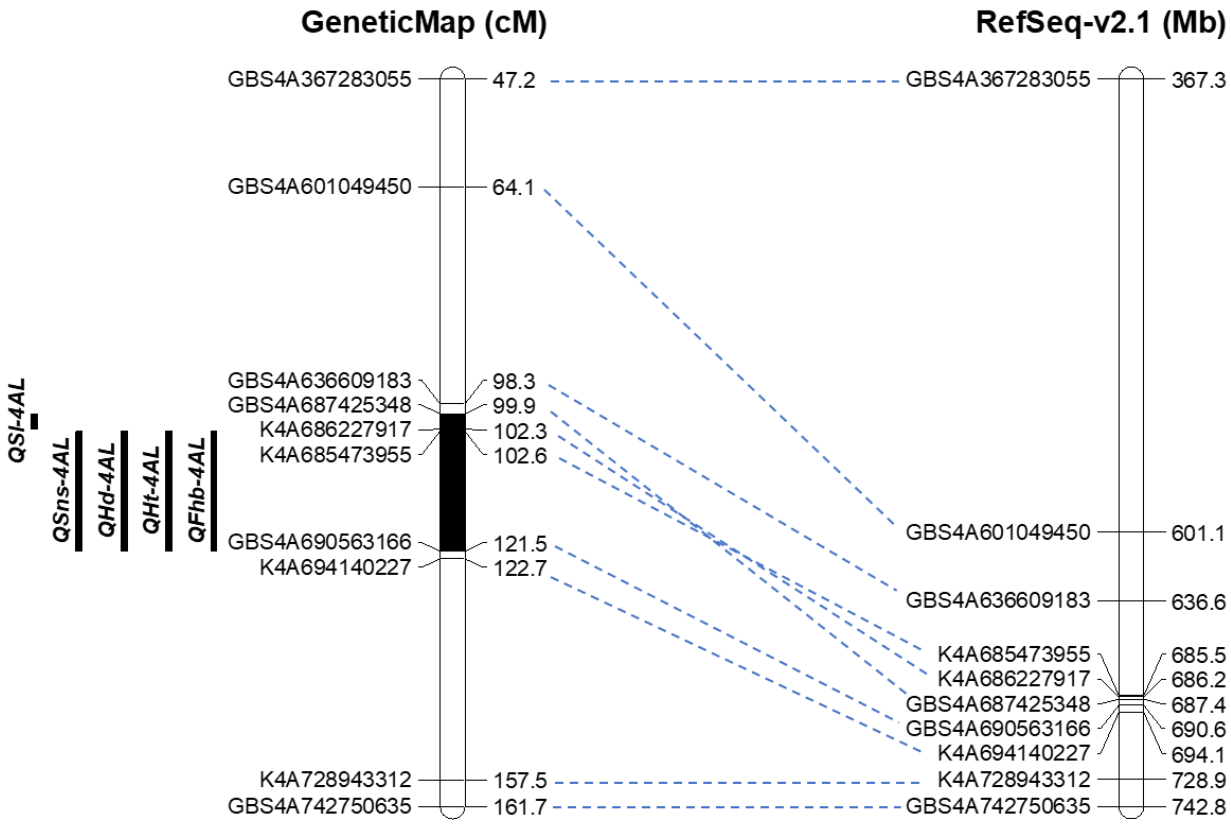

Figure 3.2 Partial genetic map (left) and physical map (right) based on IWGSC RefSeq v2.1 for chromosome 4A to show the quantitative trait locus (QTL) regions (black bars in the linkage map) for multiple traits (QTL names and intervals on the left).

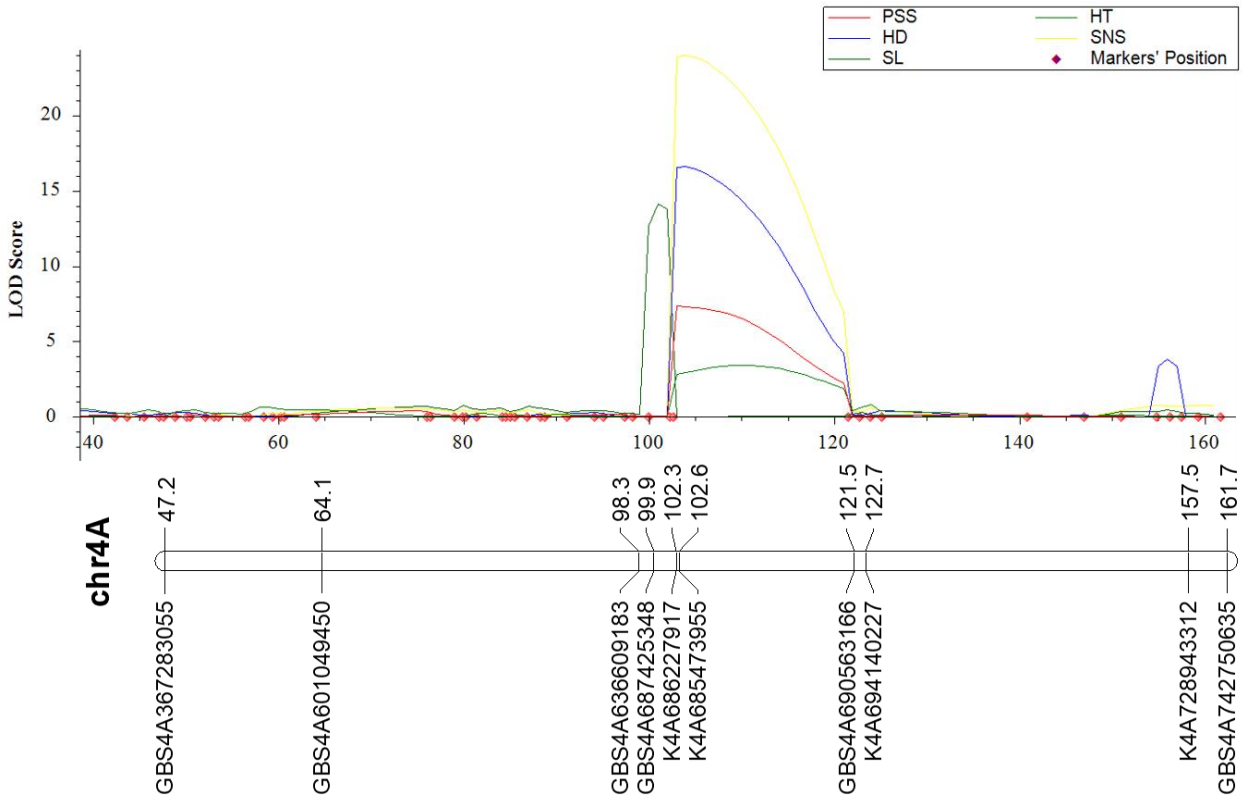

Figure 3.3 Logarithm of odds value (LOD) profiles of *QFhb-4AL* region for Fusarium head blight (FHB) and some agronomic traits on chromosome arm 4AL.

Table 3.1 Statistic summaries of Fusarium head blight (FHB) and agronomic traits using best linear unbiased prediction (BLUP) values in JagR1097 x Jagger population.

Trait	RILs		Jagger	JagR1097
	Range	Mean ± SD	Mean ± SD	Mean ± SD
PSS (%)	4.76 - 100.00	54.98 ± 24.35	69.00 ± 14.00	36.00 ± 10
HT (cm)	32.00 - 113.50	77.15 ± 12.35	77.00 ± 5.76	95.25 ± 7.59
HD	86.00 - 214.00	122.17 ± 41.84	96.13 ± 4.52	97.13 ± 4.29
SL (cm)	5.00 - 13.90	9.18 ± 1.36	8.54 ± 0.19	11.98 ± 1.25
SC	1.18 - 3.00	1.77 ± 0.24	1.83 ± 0.05	1.44 ± 0.17
SNS	9.60 - 21.80	16.00 ± 1.81	15.13 ± 0.52	14.15 ± 0.65
TGW (g)	9.32 - 50.94	34.01 ± 6.59	27.60 ± 4.23	36.44 ± 3.99
GA (mm^2)	8.90 - 19.30	15.26 ± 1.63	13.42 ± 0.76	16.80 ± 0.93
GW (mm)	2.30 - 3.80	3.25 ± 0.24	3.04 ± 0.13	3.30 ± 0.12
GL (mm)	5.40 - 7.60	6.56 ± 0.34	6.18 ± 0.10	7.01 ± 0.16

SD, standard deviation; PSS, Percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; mm^2, square of millimeter.

Table 3.2 The correlation coefficients between Fusarium head blight (FHB) and agronomic traits based on best linear unbiased prediction (BLUP) values in JagR1097 x Jagger population.

	HT	HD	SNS	SL	SC	TGW	GA	GW	GL
PSS	-0.13	-0.45**	-0.45**	-0.43**	0.13	-0.13	-0.11	-0.1	-0.1
HT		0.29**	0.34**	0.45**	-0.26	0.64**	0.6**	0.54**	0.3**
HD			0.57**	0.33**	0.11	0.21*	0.16*	0.16*	0.04
SNS				0.67**	0.12	0.2*	0.12	0.12	-0.02
SL					-0.64**	0.24**	0.21*	0.15	0.13
SC						-0.13	-0.18*	-0.1	-0.21*
TGW							0.95**	0.94**	0.49**
GA								0.87**	0.7**
GW									0.3**

PSS, Percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; * and ** are significant at p=0.05 and p=0.01, respectively.

Table 3.3 Analysis of variance (ANOVA) for Fusarium head blight (FHB) and developmental traits in JagR1097 x Jagger population.

Source of variation	PSS			HT			HD			SL			SC		
	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)
Replication	2	39.15**		2	77.15**		2	230.41**		2	27.52**		2	1.17	
Environment (E)	3	190.49**		3	872.52**		3	1170.21**		3	111.98**		3	135.27**	
Genotype (G)	148	5.73**	81	148	12.19**	90	148	13.05**	93	148	28.83**	95	148	9.63**	85
G x E	441	1.41**		441	1.76**		441	1.30**		441	2.08**		439	2.05**	
Residuals	580	NA		574	NA		588	NA		574	NA		566	NA	

PSS, percentage of symptomatic spikelet; HD, heading date; HT, plant height; SL, spike length; SC, spike compactness; df, freedom degree; H^2, broad-sense heritability; ** means significant at $p = 0.01$.

Table 3.4 Analysis of variance (ANOVA) for yield component traits in JagR1097 x Jagger population.

Source of variation	SNS			TGW			GA			GW			GL		
	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)	df	F-values	H^2 (%)
Replication	2	62.73**		1	18.05**		1	16.64**		1	10.49**		1	13.93**	
Environment (E)	3	310.11**		1	130.48**		1	193.36**		1	183.41**		1	168.41**	
Genotype (G)	148	26.50**	93	148	5.68**	85	148	7.77**	91	148	6.69**	88	148	16.91**	97
G x E	439	2.54**		148	2.09**		148	1.87**		148	2.02**		148	1.50**	
Residuals	580	NA		281	NA		281	NA		281	NA		281	NA	

PSS, percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; SL, spike length; SC, spike compactness; TGW, thousand grain weight; GA, grain area; GW, grain width; GL, grain length; df, freedom degree; H^2, broad-sense heritability; ** means significant at $p = 0.01$.

Table 3.5 Analysis of variance (ANOVA) for Fusarium head blight (FHB) severity corrected by masking confounding effects of heading date (HD) and plant height (HT).

Source of variation	PSS				
	df	Sum of Square	Mean of Square	F-value	H^2 (%)
Replication	2	2.31	1.16	40.68**	
Environment (E)	3	16.53	5.51	193.91**	
Genotype (G)	148	24.24	0.16	5.76**	
G x E	441	17.51	0.04	1.40**	57
HD	1	0.27	0.27	9.41**	
HT	1	0.5	0.5	17.57**	
Residuals	564	16.03	0.03	NA	

PSS, Percentage of symptomatic spikelet; HD, heading date; HT, plant height; NA, not available data; df, freedom degree; H^2, broad-sense heritability; ** means significant at $p = 0.01$.

Table 3.6 Summary of linkage groups in JaR1097 x Jagger population.

No.	Linkage groups	Number of loci	Length (cM)	Mean interval (cM)
1	1A1	33	70.96	2.15
2	1A2	7	30.3	4.33
3	1B1	28	83.17	2.97
4	1B2	14	13.07	0.93
5	1B3	8	8.79	1.10
6	1D	24	90.17	3.76
7	2A1	11	71.51	6.50
8	2A2	20	76.02	3.80
9	2B1	7	62.84	8.98
10	2B2	6	24.74	4.12
11	2B3	6	24.7	4.12
12	2D	15	97.67	6.51
13	3A1	3	1.33	0.44
14	3A2	17	63.51	3.74
15	3B1	3	7.73	2.58
16	3B2	26	96.46	3.71
17	3D1	3	31.8	10.60
18	3D2	8	71.25	8.91
19	4A	64	161.71	2.53
20	4B	45	108.68	2.42
21	4D	10	50.75	5.08
22	5A	90	182.05	2.02

23	5B	19	104.06	5.48
24	5D1	4	109.47	27.37
25	5D2	4	35.87	8.97
26	5D3	8	36.43	4.55
27	6A1	3	1.25	0.42
28	6A2	10	26.83	2.68
29	6A3	45	66.62	1.48
30	6B1	23	56.21	2.44
31	6B2	19	26.52	1.40
32	6D1	6	38.36	6.39
33	6D2	5	2.24	0.45
34	6D3	14	18.29	1.31
35	6D4	9	11	1.22
36	7A	59	143.28	2.43
37	7B	54	158.22	2.93
38	7D1	9	40.87	4.54
39	7D2	6	44.37	7.40
40	7D3	5	21.8	4.36
Total		750	2370.9	3.16

cM, centimorgan.

Table 3.7 Quantitative trait locus (QTL) mapping results of JagR1097 x Jagger population.

Trait	QTL	Experiment	Interval	LOD	PVE (%)	Add
FHB	*QFhb-4AL*	FHB_GH2018S	K4A685473955 - GBS4A690563166	3.79	10.34	-0.07
		FHB_GH2019S	K4A685473955 - GBS4A690563166	7.75	15.79	-0.09
		BLUP_PSS	K4A685473955 - GBS4A690563166	9.39	15.63	-0.05
	QFhb-6AL	FHB_GH2019S	GBS6A540881333 - GBS6A542602755	4.24	7.51	0.06
		BLUP_PSS	GBS6A542602755 - GBS6A543690537	4.62	6.53	0.03
HT	*QHt-3AL*	FHB_GH2018S	K3A544491394 - K3A625498368	5.19	10.07	2.59
		FHB_GH2018F	K3A544491394 - K3A625498368	3.92	5.03	2.55
		FHB_GH2019S	K3A544491394 - K3A625498368	3.91	7.61	1.99
		BLUP_HT	K3A544491394 - K3A625498368	5.55	6.64	1.52
	QHt-4AL	FHB_GH2018F	K4A685473955 - GBS4A690563166	4.14	6.29	-2.83
		FHB_GH2019W	K4A685473955 - GBS4A690563166	4.27	12.02	-2.68
		BLUP_HT	K4A685473955 - GBS4A690563166	3.27	5.16	-1.33
	QHt-4BS	FHB_GH2018S	K4B31175570 - K4B24978862	15.39	32.88	4.63
		FHB_GH2018F	K4B40019304 - K4B31175570	22.92	40.53	7.15
		FHB_GH2019S	K4B31175570 - K4B24978862	16.17	34.51	4.20
		FHB_GH2019W	K4B31175570 - K4B24978862	4.12	8.95	2.31
		BLUP_HT	K4B40019304 - K4B31175570	25.14	42.28	3.80
	QHt-5AS	FHB_GH2019S	GBS5A399501381 - GBS5A395580974	5.86	10.55	2.32
		BLUP_HT	GBS5A399501381 - GBS5A395580974	3.67	4.38	1.22
	QHt-5DL	FHB_GH2018S	GBS5D437279013 - GBS5D428329481	3.23	5.60	-1.91
		FHB_GH2019W	GBS5D444079953 - GBS5D437279013	4.38	11.43	-2.62
		BLUP_HT	GBS5D444079953 - GBS5D437279013	6.41	9.77	-1.84

HD	*QHd-2BS*	FHB_GH2018F	GBS2B69937043 - GBS2B147654222	4.94	6.37	1.54
		FHB_GH2019W	GBS2B69937043 - GBS2B147654222	3.74	5.29	1.05
		BLUP_HD	GBS2B69937043 - GBS2B147654222	4.51	5.56	0.89
	QHd-2DL	FHB_GH2018F	GBS2D620990763 - GBS2D623580344	4.88	6.09	-1.53
		FHB_GH2019W	GBS2D620990763 - GBS2D623580344	4.75	5.47	-1.08
		BLUP_HD	GBS2D620990763 - GBS2D623580344	4.22	4.96	-0.86
	QHd-4AL	FHB_GH2018S	K4A685473955 - GBS4A690563166	7.34	15.21	-1.82
		FHB_GH2018F	K4A685473955 - GBS4A690563166	12.45	19.23	-2.67
		FHB_GH2019S	K4A685473955 - GBS4A690563166	4.75	10.94	-1.21
		FHB_GH2019W	K4A685473955 - GBS4A690563166	14.83	21.69	-2.12
		BLUP_HD	K4A685473955 - GBS4A690563166	14.29	21.43	-1.75
	QHd-5DL	FHB_GH2018S	GBS5D473603213 - GBS5D485388092	12.33	24.83	-2.34
		FHB_GH2018F	GBS5D508721978 - GBS5D473603213	11.10	15.26	-2.39
		FHB_GH2019S	GBS5D473603213 - GBS5D485388092	6.29	14.59	-1.40
		FHB_GH2019W	GBS5D508721978 - GBS5D473603213	9.39	11.77	-1.57
		BLUP_HD	GBS5D508721978 - GBS5D473603213	13.89	19.02	-1.66
SNS	*QSns-2BS*	FHB_GH2019W	GBS2B69937043 - GBS2B147654222	3.32	6.47	0.41
		BLUP_SNS	GBS2B69937043 - GBS2B147654222	3.46	4.05	0.28
	QSns-4AL	FHB_GH2018S	K4A685473955 - GBS4A690563166	12.04	20.66	-0.73
		FHB_GH2018F	K4A685473955 - GBS4A690563166	18.26	26.05	-1.15
		FHB_GH2019S	K4A685473955 - GBS4A690563166	22.52	43.65	-0.99
		FHB_GH2019W	GBS4A687425348 - K4A686227917	18.97	36.40	-0.98
		BLUP_SNS	K4A685473955 - GBS4A690563166	26.02	39.83	-0.89
	QSns-5DL	FHB_GH2018S	GBS5D508721978 - GBS5D473603213	11.00	15.02	-0.63
		FHB_GH2018F	GBS5D508721978 - GBS5D473603213	12.15	15.60	-0.89

		BLUP_SNS	GBS5D508721978 - GBS5D473603213	6.76	9.00	-0.42
	QSns-7AL	FHB_GH2018S	GBS7A676634402 - GBS7A682575058	8.76	11.53	0.55
		FHB_GH2019S	GBS7A676634402 - GBS7A682575058	5.07	7.17	0.40
		FHB_GH2019W	GBS7A676634402 - GBS7A682575058	4.56	7.16	0.44
		BLUP_SNS	GBS7A676634402 - GBS7A682575058	6.85	7.87	0.40
SL	*QSl-2BL*	FHB_GH2019S	GBS2B781640361 - GBS2B786902172	5.76	5.63	0.27
		FHB_GH2019W	GBS2B781640361 - GBS2B786902172	6.56	10.39	0.45
		BLUP_SL	GBS2B781640361 - GBS2B786902172	5.61	7.57	0.28
	QSl-3DL	FHB_GH2019S	GBS3D436623518 - GBS3D495293169	6.35	6.66	-0.29
		BLUP_SL	GBS3D436623518 - GBS3D495293169	4.12	5.91	-0.24
	QSl-4AL	FHB_GH2018F	GBS4A687425348 - K4A686227917	15.51	27.42	-0.71
		FHB_GH2019S	GBS4A687425348 - K4A686227917	18.46	22.58	-0.53
		FHB_GH2019W	GBS4A687425348 - K4A686227917	18.18	27.70	-0.72
		BLUP_SL	GBS4A687425348 - K4A686227917	16.31	26.61	-0.51
	QSl-4BS	FHB_GH2018F	GBS4B49389906 - GBS4B43868168	4.98	7.40	0.37
		FHB_GH2019S	K4B40019304 - K4B31175570	8.25	8.89	0.33
		BLUP_SL	GBS4B43868168 - K4B40019304	4.62	6.13	0.25
	QSl-5AS	FHB_GH2019W	GBS5A399501381 - GBS5A395580974	7.38	9.38	0.42
		BLUP_SL	GBS5A399501381 - GBS5A395580974	6.29	8.75	0.29
SC	*QSc-2AS*	FHB_GH2019S	GBS2A82633071 - GBS2A74091161	4.16	6.28	0.05
		BLUP_SC	GBS2A82633071 - GBS2A74091161	3.43	5.49	0.03
	QSc-3DL	FHB_GH2018S	GBS3D436623518 - GBS3D495293169	4.38	10.76	0.06
		FHB_GH2018F	GBS3D436623518 - GBS3D495293169	3.50	9.34	0.08
		FHB_GH2019S	GBS3D436623518 - GBS3D495293169	5.20	7.71	0.05
		BLUP_SC	GBS3D436623518 - GBS3D495293169	5.42	9.79	0.04

	QSc-5AS	FHB_GH2019S	GBS5A399501381 - GBS5A395580974	3.33	4.93	-0.04
		FHB_GH2019W	GBS5A399501381 - GBS5A395580974	3.77	9.43	-0.07
	QSc-5DL	FHB_GH2018S	GBS5D508721978 - GBS5D473603213	3.46	7.90	-0.06
		FHB_GH2018F	GBS5D473603213 - GBS5D485388092	3.90	9.67	-0.08
		BLUP_SC	GBS5D473603213 - GBS5D485388092	3.52	6.28	-0.03
	QSc-7AL	FHB_GH2018S	GBS7A676634402 - GBS7A682575058	4.34	11.24	0.07
		FHB_GH2019W	GBS7A676634402 - GBS7A682575058	4.03	10.48	0.07
		BLUP_SC	GBS7A676634402 - GBS7A682575058	8.03	15.19	0.05
TGW	*QTgw-3AS*	Yld_GH2018F	GBS3A131021938 - K3A164943473	3.68	8.08	1.93
		BLUP_TGW	GBS3A131021938 - K3A164943473	5.34	10.36	1.03
	QTgw-4BS	Yld_GH2018F	GBS4B43868168 - K4B40019304	3.92	8.74	1.99
		Yld_GH2019S	K4B40019304 - K4B31175570	11.05	29.12	2.37
		BLUP_TGW	K4B40019304 - K4B31175570	10.84	23.51	1.54
GA	*QGa-3AS*	Yld_GH2018F	GBS3A131021938 - K3A164943473	4.17	9.41	0.47
		BLUP_GA	GBS3A131021938 - K3A164943473	5.14	8.37	0.29
	QGa-4BS	Yld_GH2018F	GBS4B43868168 - K4B40019304	5.07	11.67	0.52
		Yld_GH2019S	K4B40019304 - K4B31175570	14.00	28.33	0.76
		BLUP_GA	GBS4B43868168 - K4B40019304	13.71	25.71	0.51
	QGa-5AL	Yld_GH2018F	GBS5A491620424 - GBS5A486718824	4.55	10.25	-0.49
		BLUP_GA	GBS5A538018691 - GBS5A491620424	5.34	9.77	-0.31
GW	*QGw-4BS*	Yld_GH2018F	K4B40019304 - K4B31175570	4.96	9.69	0.08
		Yld_GH2019S	K4B40019304 - K4B31175570	8.64	23.87	0.08
		BLUP_GW	K4B40019304 - K4B31175570	8.05	18.92	0.05
	QGw-5AS	Yld_GH2019S	GBS5A78753070 - GBS5A49359680	5.16	12.66	-0.06
		BLUP_GW	GBS5A61593246 - GBS5A78753070	6.14	13.69	-0.05

GL	*QGl-1DL*	Yld_GH2019S	GBS1D466011842 - GBS1D436753692	5.45	8.22	0.10
		BLUP_GL	GBS1D466011842 - GBS1D436753692	5.99	5.89	0.07
	QGl-2DL	Yld_GH2018F	GBS2D622524713 - GBS2D577769273	9.77	14.85	-0.14
		Yld_GH2019S	GBS2D558875120 - GBS2D622524713	11.19	15.79	-0.14
		BLUP_GL	GBS2D622524713 - GBS2D577769273	13.30	15.42	-0.12
	QGl-4BS	Yld_GH2018F	GBS4B49389906 - GBS4B43868168	6.74	9.20	0.10
		Yld_GH2019S	K4B40019304 - K4B31175570	12.47	16.70	0.14
		BLUP_GL	GBS4B49389906 - GBS4B43868168	12.01	13.08	0.10
	QGl-4DS	Yld_GH2018F	GBS4D1646274 - GBS4D60616324	8.59	17.22	0.14
		Yld_GH2019S	GBS4D1646274 - GBS4D60616324	3.37	3.91	0.07
		BLUP_GL	GBS4D1646274 - GBS4D60616324	8.65	9.05	0.09
	QGl-5BL	Yld_GH2018F	GBS5B38058313 - GBS5B273021492	4.42	7.41	-0.09
		Yld_GH2019S	GBS5B38058313 - GBS5B273021492	7.07	8.63	-0.10
		BLUP_GL	GBS5B273021492 - GBS5B400687853	6.27	6.49	-0.07

FHB, Fusarium head blight; SNS, spikelet number per spike; TGW, thousand grain weight; HD, heading date; HT, plant height; SL, spike length; SC, spike compactness; GA, grain area; GW, grain width; GL, grain length; GH, greenhouse; BLUP, best linear unbiased predictions; Yld, yield; S, spring; F, fall; W, winter; LOD, logarithm of odds; PVE, the phenotypic variation explained by a QTL; Add, additive effect in which a positive value indicates beneficial allele contributed by JagR1097.

Table 3.8 Quantitative trait locus (QTL) for Fusarium head blight (FHB) severity corrected by masking confounding effects of heading date (HD) and plant height (HT).

Trait	QTL	Experiment	Interval	LOD	PVE (%)	Add
FHB severity	*QFhb-4AL*	Corrected_BLUP	K4A685473955 - GBS4A690563166	7.45	11.80	-0.04
	QFhb-4DL	Corrected_BLUP	GBS4D456253756 - GBS4D481515282	3.69	6.34	0.03
	QFhb-6AL	Corrected_BLUP	GBS6A542602755 - GBS6A543690537	4.21	6.23	0.03

BLUP, best linear unbiased predictions; LOD, logarithm of odds value; PVE, the phenotypic variation explained by a QTL; Add, additive effect in which a positive value indicates beneficial allele contributed by JagR1097.

Table 3.9 Quantitative trait locus (QTL) clusters in JagR1097 x Jagger population.

Cluster	Trait	QTL	Dataset	Interval	Donor
2BS	HD	*QHd-2BS*	3	GBS2B69937043 - GBS2B147654222	JagR1097
	SNS	*QSns-2BS*	2	GBS2B69937043 - GBS2B147654222	JagR1097
2DL	HD	*QHd-2DL*	3	GBS2D620990763 - GBS2D623580344	Jagger
	GL	*QGl-2DL*	3	GBS2D577769273 - GBS2D622524713	Jagger
3AS	TGW	*QTgw-3AS*	2	GBS3A131021938 - K3A164943473	JagR1097
	GA	*QGa-3AS*	2	GBS3A131021938 - K3A164943473	JagR1097
3DL	SL	*QSl-3DL*	2	GBS3D436623518 - GBS3D495293169	Jagger
	SC	*QSc-3DL*	4	GBS3D436623518 - GBS3D495293169	JagR1097
4AL	FHB	*QFhb-4AL*	3	K4A685473955 - GBS4A690563166	Jagger
	HT	*QHt-4AL*	3	K4A685473955 - GBS4A690563166	Jagger
	HD	*QHd-4AL*	5	K4A685473955 - GBS4A690563166	Jagger
	SNS	*QSns-4AL*	5	K4A685473955 - GBS4A690563166	Jagger
	SL	*QSl-4AL*	4	K4A686227917 - GBS4A687425348	Jagger
4BS	HT	*QHt-4BS*	5	K4B24978862 - K4B40019304	JagR1097
	SL	*QSl-4BS*	3	K4B31175570 - GBS4B49389906	JagR1097
	TGW	*QTgw-4BS*	3	K4B31175570 - GBS4B43868168	JagR1097
	GA	*QGa-4BS*	3	K4B31175570 - GBS4B43868168	JagR1097
	GW	*QGw-4BS*	3	K4B31175570 - K4B40019304	JagR1097
	GL	*QGl-4BS*	3	K4B31175570 - GBS4B49389906	JagR1097
5AS	HT	*QHt-5AS*	2	GBS5A399501381 - GBS5A395580974	JagR1097
	SL	*QSl-5AS*	2	GBS5A399501381 - GBS5A395580974	JagR1097
	SC	*QSc-5AS*	2	GBS5A399501381 - GBS5A395580974	Jagger

5DL	HT	*QHt-5DL*	3	GBS5D428329481 - GBS5D444079953	Jagger
	HD	*QHd-5DL*	5	GBS5D473603213 - GBS5D508721978	Jagger
	SNS	*QSns-5DL*	3	GBS5D473603213 - GBS5D508721978	Jagger
	SC	*QSc-5DL*	3	GBS5D473603213 - GBS5D508721978	Jagger
7AL	SNS	*QSns-7AL*	4	GBS7A676634402 - GBS7A682575058	JagR1097
	SC	*QSc-7AL*	3	GBS7A676634402 - GBS7A682575058	JagR1097

FHB, Fusarium head blight; SNS, spikelet number per spike; TGW, thousand grain weight; HD, heading date; HT, plant height; SL, spike length; SC, spike compactness; GA, grain area; GW, grain width; GL, grain length; Donor, parent providing resistance or high phenotypic value allele; Dataset indicates the number of repeated QTL.

Table 3.10 Kompetitive allele specific polymerase chain reaction (KASP) primers for quantitative trait locus (QTL) in JagR1097 x Jagger population.

Marker	Physical position (IWGSC RefSeq v2.1, bp)	FAM primer	HEX primer	Common primer
K4A685473955	chr4A:685473955	ATGGAGTAGATCGGCGGCT	ATGGAGTAGATCGGCGGCG	TGCTCCAGCTCCGCTACC
K4A686227917	chr4A:686227917	GAAATGTCTGTTTCTCCAACAAGTA	GAAATGTCTGTTTCTCCAACAAGTG	TATGTACTCTCTCTTCGTCCGA
K4A694140227	chr4A:694140227	ACACTACCGAGCCTAGTGAGA	ACACTACCGAGCCTAGTGAGC	GCAGGGCTAGTTAGAACTGGTAG
K4A728943312	chr4A:728943312	CCTGTAAGACGGCAGAACCTAT	CCTGTAAGACGGCAGAACCTAC	TGAACCCTGTACATGGTCCG
K3A164943473	chr3A:164943473	AGCATATTCTCCGACGTGCT	AGCATATTCTCCGACGTGCC	CTCTGATGATGCGCGGTCT
K3A544491394	chr3A:544491394	GCCGAGGGAGGTGAACAGT	GCCGAGGGAGGTGAACAGC	CATAGTTTGAACTCCATCACTTCTT
K3A625498368	chr3A:625498368	GATGATAGCCGAACGTGAGAGT	GATGATAGCCGAACGTGAGAGC	ACAGAGACCATGAACCTTCGA
K4B24978862	chr4B:24978862	CGGTGATTTACTGTTTCTGCTCA	CGGTGATTTACTGTTTCTGCTCG	CTCTGCACCATGCCTGTCAT
K4B31175570	chr4B:31175570	ACTTCCAACTGCCACACCTA	ACTTCCAACTGCCACACCTC	GGTTGCCTTCAGTCTCTGATACA
K4B40019304	chr4B:40019304	CCGTTCATTGTTCAGACTGATTGT	CCGTTCATTGTTCAGACTGATTGC	AGAATATGCTCCCTGTCTCCTA

Table 3.11 Comparison of phenotypic values based on best linear unbiased prediction (BLUP) between different recombinant inbred line (RIL) groups with contrasting alleles at *QFhb-4AL* and *QTgw-4BS*.

Group ID	Genotype	PSS (%)	TGW(g)	SNS	HT	HD
1	4AL(+)4BS(+)	47.62	35.97	17.17	80.35	102.2
2	4AL(+)4BS(-)	46.19	32.75**	16.79	72.1**	102.53
3	4AL(-)4BS(+)	57.91**	35.06	15.28**	77.38**	99.85**
4	4AL(-)4BS(-)	56.5**	32.56**	15.33**	69.24**	100.06**

PSS, Percentage of symptomatic spikelet; HD, heading date; HT, plant height; SNS, spikelet number per spike; TGW, thousand grain weight; 4AL(+) and 4AL(-) refer to the Jagger allele and JagR1097 allele, respectively, at *QFhb-4AL*; 4BS(+) and 4BS(-) refer to, JagR1097 allele and Jagger allele, respectively, at *QTgw-4BS*; All the statistical analysis were compared to the group 1 with genotype 4AL(+)4BS(+). ** is significant at $p < 0.01$.

Table 3.12 Physical positions of quantitative trait locus (QTL) flanking single nucleotide polymorphisms (SNPs) in IWGSC RefSeq v2.1 reference genome.

SNP name	Flanking sequence	Physical location (bp)
GBS1D436753692	TTACCTATTTCCGTTATGCC[G/A]TTTTTATTTTCTAGC	chr1D:436753692
GBS1D466011842	ATGGAGCCAAAACAGGTGGA[C/G]AGATGGAAGCCACCC	chr1D:466011842
GBS2A74091161	ACTCAAGACCTCTTTTTTTA[A/G]AACTGAGCAACCGAA	chr2A:74091161
GBS2A82633071	GAAAATATCTGTGAACGGAC[T/C]AATAACGGAGCCCAA	chr2A:82633071
GBS2B147654222	TTGTTCAGACATCATTGACG[T/C]CGAATGCTCGGAGTG	chr2B:147654222
GBS2B69937043	AAACCAAACTGATTCAGAAG[A/G]CACAAACAGGGAAAC	chr2B:69937043
GBS2B781640361	ATTGATTGATTGCCATTTGC[T/C]AGTGATTTTTACTCT	chr2B:781640361
GBS2B786902172	AGCGATTCTTCCGCGTCTCT[T/C]TCGTCGTCGACAGAT	chr2B:786902172
GBS2D558875120	GACAACGACTACGACGGCGG[T/C]CGCCAGAGTTCGCCT	chr2D:558875120
GBS2D577769273	TGCAGCTGCAGGCAATAATA[C/T]GTGCGTGAATGAAGC	chr2D:577769273
GBS2D620990763	CTGAGAATGAGTAAGCAGAG[A/G]ACAAAGGAGGATTGT	chr2D:620990763
GBS2D622524713	GAACCAAATCATCACATTCG[G/A]TACAAGTTGTAGGAC	chr2D:622524713
GBS2D623580344	CCTGGTTGCACGCACAGCTA[G/A]TATCTTAGCCTTTGC	chr2D:623580344
GBS3A131021938	CACCTACAAAGGCTACAACA[A/G]AAGACAAACGAAGAC	chr3A:131021938
GBS3D436623518	TTGGGCTGAATTGTTGGATG[A/G]TACTCTTTTTGTTAG	chr3D:436623518
GBS3D495293169	CGAATAAAAGCAAGTATAAT[A/T]AGATACAGTCAGCAA	chr3D:495293169
GBS4A687425348	TCTGCGACCCTGCCCTGGCC[T/A]CTGCATCATGTAGGG	chr4A:687425348
GBS4A690563166	CAAGGCCAATGGCACAGTAC[T/A]GGCATGCAGCGCCAT	chr4A:690563166
GBS4B43868168	CGGAGCTGCAGCGTATCGCC[A/T]CTAGGACGGAAGAGG	chr4B:43868168
GBS4B49389906	CGCCGAAGTACACTGCAAGG[C/G]TGCTGCACCCAACGT	chr4B:49389906
GBS4D1646274	AGTAGCTTGCTTAGGGAAGG[G/A]ATCACACGGCTGTCC	chr4D:1646274
GBS4D456253756	CCGAGGACCACCACCTGCAG[C/T]TTTCCTCCGACGATC	chr4D:456253756

GBS4D481515282	TAATAACCAACGTCGGTAAA[A/G]AGTGTCAGACATCGG	chr4D:481515282
GBS4D60616324	ATATGGAGTAGTATTCTGGA[A/G]CGCTGCAGAGTTCCC	chr4D:60616324
GBS5A395580974	CTCAGCGTGAATGGCGGCTT[G/T]GCCGTGCCCCTGCCT	chr5A:395580974
GBS5A399501381	ACTCGCTGCTGTACCGCACC[T/A]ACCCGCGGGACAGGG	chr5A:399501381
GBS5A486718824	GGTTGAGGCAAGGCCCACTT[T/G]AATCGGCCTACTGTA	chr5A:486718824
GBS5A491620424	CAGTGATGATGTAATCAGGC[T/C]GATTGGTGTAGTTCG	chr5A:491620424
GBS5A49359680	GCTGTCGCTTCCTCCGTCGC[T/C]GCCGTAGAGAGAGGC	chr5A:49359680
GBS5A538018691	GACCAGACAGACCCACCTTT[T/G]GCCGAGTTTACTTTC	chr5A:538018691
GBS5A61593246	ATCGGGCCAGCGGAAGCAGC[A/G]GCTGCGTTCGGAACC	chr5A:61593246
GBS5A78753070	GCGCTTATGGTCTCTGGGTG[T/C]AACCTACCAGAAACG	chr5A:78753070
GBS5B273021492	CAAGGCCTAGAAGATGAGGA[T/C]TGAGTGGTACGACAC	chr5B:273021492
GBS5B38058313	GACGACAGCAGGCAGTGCGC[T/C]GCCGCCGCCGCCGCC	chr5B:38058313
GBS5B400687853	TGGTACGTCAGCGTACTCTC[T/G]GGAAAAAAACACACA	chr5B:400687853
GBS5D428329481	TGACGGCGCCTGAAATGCCG[T/C]GTCCTCTGGTCACCA	chr5D:428329481
GBS5D437279013	AGAATGGTGGCAGAGGTGCC[G/A]CTTGGTAGTATTTTA	chr5D:437279013
GBS5D444079953	GACACCATGGGCTCCACCCA[C/T]GACACGTCTGCAGCT	chr5D:444079953
GBS5D473603213	CTGCAGGACTAGAAAAACTG[G/A]CGGTTTCTTCTAAAA	chr5D:473603213
GBS5D485388092	TTCAACTAGTGATTTCTTTG[T/C]GTTTCATAGAAAGAT	chr5D:485388092
GBS5D508721978	CTTTTGAGGCAAATATTATG[C/A]ATGACGGGATCGAGT	chr5D:508721978
GBS6A540881333	AGGTGTTTTGACATGGATTG[G/C]AGCGTTCGGTGGCTG	chr6A:540881333
GBS6A542602755	CTCGCCTGGCCCTGGCTGTG[G/C]CCTGTGGGTGGTCCT	chr6A:542602755
GBS6A543690537	CAGTTCATTCAGAAACCATA[A/C]ATAGCACAATTCTGC	chr6A:543690537
GBS7A676634402	CAGATGTTGTCGACGCCACC[A/G]AGAACAGCTGCAGCC	chr7A:676634402
GBS7A682575058	TCCTGATTATGGCAAGTTCC[G/A]AAATCCCCTCCGCCT	chr7A:682575058

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