Genetic mapping of quantitative trait loci conditioning Fusarium head blight resistance in hard winter wheat

by

Umara Sahar Rana

B.S., University of Agriculture Faisalabad, 2010
M.S., University of Agriculture Faisalabad, 2012

AN ABSTRACT OF A DISSERTATION

submitted in partial fulfillment of the requirements for the degree

DOCTOR OF PHILOSOPHY

Department of Agronomy
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Abstract

Fusarium head blight (FHB), also known as 'scab', incited by *Fusarium graminearum* (Schw), is one of the most damaging fungal diseases in wheat. FHB reduces grain yield drastically, but also grain quality due to shriveled kernels, protein damage, and mycotoxin contamination caused by the fungal infection. Host plant resistance is the most effective and environmentally safe approach to combat this disease. To identify resistance genes from locally adapted cultivars, a population of 178 recombinant inbred lines (RILs) from Overland × Everest was genotyped using single nucleotide polymorphism (SNP) markers derived from genotyping-by-sequencing (GBS). The RIL population was phenotyped for resistance to the initial infection (type I), fungal spread within a spike (type II), mycotoxin (DON) accumulation in grains (type III) and *Fusarium* damaged kernel (type IV) in repeated greenhouse and field experiments. Seven QTLs were identified on chromosome arms 1AL, 3BL, 4BS, 4BL, 6AL, 6BL 7AS and 7BL for type I resistance. Hard winter wheat cultivar Everest contributes all the resistance alleles except two on chromosome arms 4BS and 6BL, which are contributed by hard winter wheat cultivar Overland. Six QTLs on chromosome regions of 1BL, 4A, 4BS, 5AL, 6BL and 7AS confer type II resistance with the resistance QTLs on 1BL, 4BS, 6BL and 7AS from Everest and on 4A, 4BS, and 5AL from Overland. The type II QTL on chromosome 4BS is overlapped with the reduced height gene *Rht-B1*. QTLs for type III resistance were mapped on 4BS and 5AL while QTLs for type IV resistance were mapped on chromosome 4BS, 5AL and 7AS and they overlapped with type II resistance in the corresponding chromosome regions. The haplotype analysis showed that genotypes containing multiple QTLs showed significantly higher resistance than those with fewer or no QTLs, indicating that these QTLs have additive effects on FHB resistance. Type I FHB resistance was poorly characterized in the literature. The current study

demonstrated that Everest carries several QTLs for type I resistance, thus is a useful native source for type I resistance. Some SNP markers tightly linked with the QTLs for different types of resistance were successfully converted into Kompetitive allele-specific polymerase chain reaction (KASP) assays and could be used in marker-assisted breeding for FHB resistance in wheat.

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Chapter 1 - Literature Review

Origin and domestication of wheat

The origin of modern-day agriculture traces back to 12,000 years ago when humans made a transition from nomadic to an agriculture-based sedentary lifestyle and started raising plants and animals for food and feed, an event called the Neolithic revolution. The first deliberate efforts of selection from wild natural stands of cereals took place in the fertile crescent, a region spanning from modern-day Jordan to Southeast Turkey. These farming practices led to the development of domesticated varieties of cereals, which are still cultivated in some parts of the world (Salamini et al., 2002). Alteration of key traits of wild plants through artificial selection is called domestication, e.g. large kernel size and non-brittle ears were the principal morphological traits of wild progenitors (Zohary, 2004). Three main cereals: einkorn (*T. monococcum* L.), emmer wheat (*T. dicoccum*), and barley (*Hordeum vulgare*), were the important domesticated crops of the Neolithic period due to their nutritive values and high content of starch and proteins. Soon after their establishment, the rapid expansion of wheat to Asia, Europe, and Africa made wheat one of the staple food crops worldwide and it is widely grown in more than 70 countries (FAO, 2019).

Wheat belongs to the genus *Triticum*, which encompasses three sub-groups based on the ploidy level. The diploid einkorn wheat (*Triticum monococcum*), the tetraploid emmer wheat (*Triticum dicoccum*), and hexaploid bread wheat (*Triticum aestivum*). Bread wheat is an allohexaploid with three genomes, A, B and D. Each genome consists of seven chromosomes that result in a total of 42 chromosomes (2n = 6x = 42). The A genome originated from *Triticum urartu* (2n =14, AA), while the D genome was from *Aegilops tauschii* (2n =14, DD). The origin

of the B genome is still debatable, but it is thought that *Aegilops speltoides* (2n = 14, SS) is the closest species to the B genome in the modern wheat.

Two independent events of hybridization led to the development of bread wheat. The allopolyploidization followed by the chromosome doubling between the A and B genomes lead to the development of tetraploid wheat *Triticum turgidum* spp. *dicoccoides* (2n =28, AABB). Domestication of *Triticum dicocoides* and its rapid expansion in Europe and Asia lead to a second independent hybridization event with *Aegilops tauschii* to form the hexaploid wheat *Triticum aestivum* (2n = 6x = 42, AABBDD) (Luo et al., 2007; Sorrells & Yu, 2009).

Hexaploid wheat behaves as a diploid with 21 bivalents that can be observed during metaphase I. Chromosome pairing is largely genome-specific, i.e., homologous pairing controlled by a major locus (*Ph1*) on chromosome 5B, a trait responsible for hybrid fertility within and between species.

Production and market classes of wheat in the United States

Wheat covers the largest acreage among all commercial crops. The current global wheat production is 764.32 million tons, making it the third most-produced cereal after maize and rice (FAO, 2020). United States is the third-largest producer of wheat (49.3 million tons) after China (122.0 million tons) and India (101.2 million tons) (USDA Wheat outlook, 2020). In the U.S. wheat acreage and production are slightly behind corn and soybean. In the 2019-2020 growing season, wheat was planted on 18.2 million hectares with an average yield of 3.07 metric tons per hectare in 15.6 million hectares harvested (USDA Wheat data, 2020). This represents the lowest acreage on record since 1919, which is due to the relative lower return of wheat than corn and soybean, and increased competition in the global wheat market.

The two important commercial wheat species are *Triticum aestivum* L. (common wheat) and *Triticum turgidum* var. durum L. (durum wheat) with common wheat occupied 95% global wheat production. Wheat has three types based on their growth habits, namely winter wheat, spring wheat and facultative wheat. Winter wheat that is planted in fall and harvested in summer (10-months cycle) requires a vernalization period of below 10 ºC temperature before flowering. Facultative wheat is planted in winter months, may or may not require vernalization before flowering, but it cannot withstand the prolonged period of freeze stress. Spring wheat is planted in spring or early summer and harvested in later summer or early fall (4-5 months cycle) without requirement of vernalization for flowering and cannot even withstand freezing temperature (Baenziger, 2016). Durum wheat is mainly spring type.

Wheat in the United States is further subdivided into six classes based on grain color, grain hardness and growth habits. These are hard red winter (HRW), hard red spring (HRS), soft red winter (SRW), soft white (SW), hard white (HW), and durum wheat (Vocke & Ali, 2014). Winter wheat contributes 70-80% of total wheat production in the United States. Being versatile and having excellent milling and baking qualities, HRW wheat is used in making bread and related products like hard rolls, flatbread and croissants (U.S. wheat associates, https://www.uswheat.org/working-with-buyers/wheat-classes/). The states of Great Plains, namely Colorado, Kansas, North Dakota, South Dakota, Nebraska, Oklahoma, Texas, and some neighboring states, such as Montana and New Mexico grow HRW wheat, which accounts for 40% of total wheat production in the United States (Carver et al., 2001). The HRS wheat is grown in the northern plains, mainly in Montana, Minnesota, South and North Dakota. The HRS wheat has high protein content and strong gluten, and is perfect for making bread, pizza crust, bagels, pretzels and related products. Internationally, HRS wheat is often used to improve the

flour strength by blending it with domestic wheat supplies or other classes of wheat to achieve the desired charactersitics. The SRW wheat is mainly grown in Arkansas, Georgia, Indiana, Illinois, Kentucky, Louisiana, Michigan, Maryland, North and South Carolina, Ohio, Pennsylvania, Tennessee and Virginia (Bacon et al., 2001). It usually has low protein content and less gluten strength, which makes it ideal for making cakes, cookies, pastries, pie crust and crackers etc. The SW wheat (spring and winter types) is primarily grown by farmers in Pacific Northwest States namely Idaho, Washington, Oregon. Because of the white color and low protein content, SW wheat is mainly used in making Asian style bakery products, cakes and pastries.

Durum wheat is the only tetraploid in commercial production, and it has the hardest texture among all six wheat classes. It is mainly grown in two areas of the United States, hard amber durum in the Northern Plains and desert durum in the desert Southwest under irrigation in California and Arizona. It comprises 3-5% of total wheat production and is used mainly to make spaghetti and pasta products due to its high gluten content and amber color (Shewry, 2009; Gwirtz et al., 2007).

Kansas is the leading wheat producing state in the United States throughout 20th century due to its well-suited climate for winter wheat. During 2019-2020 growing season, wheat was planted on 2.75 million hectares in Kansas with a total production of 80,122 metric tons (www.nass.usda.gov). Hougham was the first person who started research on spring and winter wheat in 1868 in Kansas (Paulsen, 2001). T. C. Henry was the one who tested winter wheat in Kansas in 1870 and he encouraged other farmers to grow wheat instead of corn (https://www.kshs.org/kansapedia/wheat/12235).

Factors affecting the wheat production

Wheat production around the world is important for the food security of the human population. The Great Plains of the United States is the main region of wheat production. The wheat production in the Great Plains is mainly influenced by a combination of temperature and precipitation. Hatfield & Dold (2018) analyzed the yield data from 1950 to 2016 and found a decreasing trend in production since 2000 until with a yield recovery to a record level in 2016. They observed that the main cause of variation in yield was attributed to insufficient precipitation during the grain-filling period. On the other hand, prolong wet-period during harvesting can also cause wheat head sprouting which ultimately leads to reduce crop yield (Lin et al., 2016). Besides abiotic stresses, many biotic stresses affect wheat yield which includes diseases and insect pests. Viruses and fungi are mainly responsible for wheat diseases. Hollandbeck et al. (2018) estimated the average yield losses of 10.7% in the past five years due to diseases including stripe rust, leaf rust, wheat streak mosaic virus, barley yellow dwarf, Fusarium head blight (FHB), powdery mildew, tan spot and bunt. Moreover, unsustainable human population growth leads to global climate disruption due to human-generated greenhouse gas emissions in the atmosphere. The challenges such as rising grain consumption and global warming require further improvement in the yield potential of wheat while not compromising food safety (Curtis & Halford, 2014). To ensure food security, it is the dire need to breed wheat varieties with a higher yield, resistance to multiple diseases, adapted to the changing environmental patterns.

Pathogens, epidemics and impact of FHB

Fusarium head blight (FHB) was first described in the UK in the late 19th century (Champeil et al., 2004a; Goswami & Kistler, 2004). At present, it has been widely recognized as

the most devastating disease in wheat grown in warm and humid environments, worldwide (Kubo et al., 2013; Russell et al., 2010). FHB, also known as scab, has received great attention during the past decades and caused significant yield and economical losses (Magan et al., 2011). Mycotoxins that are produced during pathogen infection contaminate wheat grain and cause the major concerns of human and animal health (Gilbert & Tekauz, 2011; Hornick, 1992; Pestka & Smolinski, 2005).

F. graminearum is the predominant species in most wheat-growing regions of the world. The fungus can survive in crop residues and transmits through the wind and splash water. It can cause not only head blight, also seedling blight, root rot, and foot rot (Walter et al., 2010). Accumulation of deoxynivalenol (DON) mycotoxin in grain causes quality loss, which may result in the rejection of sale or reduced price in domestic and export markets (Schaafsma et al., 2001).

Several *Fusarium* species including *F. poae, F. avenaceum, F. culmorum, Microdochium nivale* and *F. graminearum* have been associated with FHB. *F. graminearum* is the prevalent causal species of wheat head blight in most of wheat-growing area worldwide (Kosová, Chrpová, & Šíp, 2009). *Gibberella. zeae* is a facultative saprophyte; serves as the main reservoir for inoculum that leads to the initial infection (Del Ponte et al., 2005). Other *Fusarium* species have also been found to be predominant in some wheat-growing areas (Osborne & Stein, 2007). Infected crop residue is the principal source of *F. graminearum* inoculum for FHB infection. The ascospores, conidia, chlamydospores and hyphal fragments of *Fusarium* sp. can all serve as the inocula to spread the disease (Russell et al., 2010). Climate or environmental variations such as precipitation, humidity, temperatures and other environmental factors all affect the disease

epidemics (Wu et al., 2011). Prolonged wet weather along with warm temperatures will facilitate FHB outbreaks.

Many epidemics of FHB have been reported globally including Asia, Europe, South and North America. Yield reduction due to shrunk or undeveloped kernels in infected wheat may reach up to 50-60% (Champeil et al., 2004a; Champeil et al., 2004b). During the last decade of the 20th century, China has suffered more than one million tons of yield losses in wheat due to FHB (Bai & Shaner, 2004). While in the United States, several epidemics have been reported in different wheat-producing regions such as the SRW wheat region (Arkansas, Indiana, Michigan, Missouri, Ohio etc.), HRS wheat region (Minnesota, North and South Dakota) (De Wolf et al., 2003). The direct yield losses of wheat and barley from 1991 to 1997 were $1.3 billion (Johnson et al., 1998). Average wheat yield losses between 20 and 50% were recorded during the last 60 years in Argentina (Stenglein, 2009). The FHB infected seeds significantly reduced the germination rate and seedling vigor (Matarese et al., 2012).

FHB disease cycle and symptoms

Up to 20% of *Fusarium* species are known to go through the sexual stage during the disease cycle (Ma et al., 2013). *F. graminearum* is haploid and belongs to the phylum Ascomycota. The homothallic (capable of self-fertilization) nature of *F. graminearum* is due to the subsistence of genes in the haploid genome associated with *Mat1-1* and *Mat1-2* mating types in the fungus (Ma et al., 2013). During the brief sexual stage of the fungus development, bluish-black perithecia that appear as black "dots" on the infested plant debris are reproduced under favorable weather conditions. The spores are forcibly discharged into the air from the surface of these residues (Gilbert & Haber, 2013; Trail, 2009) and land on flowering spikelets. The fungus produces asexual spores (macroconidia) on sporodochia (hyphal structures) on the infected plant

debris, which may be dispersed to plants by rain-splash or wind to plants. These spores germinate and enter inside the plant tissues through degenerating anther or natural opening such as palea and lemma base to start the disease cycle of FHB. At the infection site, the fungus grows intracellularly and asymptomatically, proliferating through pith and xylem. This infection strategy is classified as biotrophic where the pathogen lives on a living host. Underneath the infection site, the *F. graminearum* spreads gradually, the necrosis begins and immediately colonizes the spikelet tissues to show water soaking symptoms as the fungus grows intracellularly, and late the tissues become bleached. After the floret infection, *F. graminearum* spreads into uninfected florets through the rachis, eventually damage the kernels by contaminating them with DON (Ma et al., 2013). Overall, spike tissue bleaching in the field is a visual symptom that occurs about three weeks of floret infection of FHB pathogen (Leonard & Bushnell, 2003), and visual salmon-pink mycelium of *Fusarium* grown at the base of spikelets can be observed. Due to the DON accumulation and colonization, the developing seeds may stop filling and can be bleached or shriveled and small-sized as 'tombstones' or *Fusarium*-damaged kernels (FDK) (Gilbert & Haber, 2013; Wegulo, 2012; Loughman et al., 2008).

Deoxynivalenol (DON) toxicity and it's health concern

DON, also known as vomitoxin, is a kind of trichothecene, anepoxy-sesquiterpenoid. It occurs mainly in grains of wheat, barley, oats, rye, and maize, and in a smaller amount in rice, sorghum, and triticale. The DON contamination in infected grains has primarily consorted with *F. graminearum* (*Gibberella zeae*) and *F. culmorum*, both of which are responsible for FHB in cereal crops (Gautam, 2010). The DON is a potent mycotoxin having a low molecular weight (MW = 296 Da) and acts as an inhibitor of protein biosynthesis (Gratz et al., 2013). Accumulation of DON and the plant metabolite DON-3-β-D-glucoside (D3G) are usually

observed in infected kernels of wheat, maize and barley (Gunnaiah & Kushalappa, 2014). However, the mechanisms involved in metabolizing these compounds by human microbia has not been fully understood (Gratz et al., 2013). Studies on human intestinal cells revealed that DON can adversely affect nutrient absorption efficiency of the intestine and disrupts intestinal barrier function (Pinton et al., 2009). The DON may cause emetic effects in humans ranging from vomiting, anorexia and nausea (gastrointestinal dysfunction) to immunotoxicity (Wu et al., 2010). While in animals, it can cause feed refusal, weight gain reduction, diarrhea, vomiting, weakness (Pestka, 2007; Wegulo, 2012).

Several countries have set an advisory level of DON in grains. The United States has set a up limit of 1 $\mu g\ g^{-1}$ for human consumption while Canada limits DON content < 2 $\mu g\ g^{-1}$ in cereal grains for human consumption (Bianchini et al., 2015). The presence of DON in barley can lead to uncontrolled gushing in beer that significantly affects the brewer industries (Loughman et al., 2008). From 1993 to 1997, only 19% barley produced in the U.S. met the standards of the brewing industry (< 0.5 ppm of DON) and the remaining 81% had up to 41 ppm of DON and could not be used for beer production. Barley with < 3 ppm of DON can be sold as feed grain at a very low price and the rest need to be discarded (Windels, 2000). Therefore, grain quality is affected by FHB through direct damage from low yield, and indirect damage from DON produced during disease development (Aldred & Magan, 2004). Mycotoxin accumulation makes contaminated cereal grains unsuitable for human consumption.

Strategies for management of FHB in wheat

The FHB disease triangle includes available susceptible hosts, pathogens, and an appropriate environment for an epidemic to take place. Many management attempts have been made to reduce the severity of the disease such as disease forecasting, agronomic management

practices include crop rotation and deep ploughing to reduce levels of initial pathogen inoculum, biological and chemical control, growing cultivars with resistance (Bai & Shaner, 2004; Bergstrom et al., 2011; Surendra & Cuperlovic-Culf, 2017). None of these strategies could control the disease when they are used singly. However, integrated management practices that combine multiple strategies have recently received great acceptance (Mcmullen et al., 2008).

Disease forecasting

The epidemic risk forecasting model was set up online for the first time in Minnesota during the 2004 growing season. The model defined different categorical risk designations (e.g., low, moderate, high) and provided predicted information to the producers for managing their crop production and personal aversion to risk (Hollingsworth et al., 2006). Computational resources for building a mathematical description of multilevel biological processes are available to help explain not only the complexity of biological systems also their interactions with surrounding environments and agro-ecosystem (El-Sharkawy, 2011). Presently, many plant disease simulation models are being employed as disease prediction tools to provide strategic options for improvement of crop productivity and for optimization of the crop management system to minimize disease risk. Disease forecasting models can also aid producers to evaluate the disease risks and fungicide types and the amount required for control. Several forecasting models have been available for predicting FHB disease severity, disease incidence, disease index including both incidence and severity, and DON content in infected kernels based on the environmental variables such as temperature, rainfall and relative humidity. The prediction methods may include empirical analysis (regression types of analysis), mechanistic approaches (process-based analysis) and simulation approaches. Mechanistic approaches use linked-differential equations to follow through the different aspects of the life cycle of disease and host

phenology (De Wolf et al., 2003; Del Ponte et al., 2005; Dexter et al., 2003; Rossi et al., 2003). Model performance is determined by the accuracy and precision of different measurements (R-squared, RMSE, d-index, etc.). Some prediction models are based on laboratory studies describing the impacts of environmental conditions on the production of mycotoxins while other are developed by validating the results from laboratories in the field to predict occurrence of DON, and other toxins. Variables for prediction models typically include weather variable data, and agronomic and economic factors. Most of the models intend to provide an early warning for unacceptable risk of disease and mycotoxin levels, but mainly depends on weather data (within season). Pre-planting management options require stochastic model approaches based on long-term weather forecast, historical data and agronomic practices on mycotoxin occurrence. However, it is likely that these prediction models may become widely available in the near future as risk assessment tools to assist in the management of diseases (Wu et al., 2010).

Cultural practices, biological and chemical control

The rotation of different crops, manipulating planting time, and burying crop residues through tillage have been found affecting the incidence of FHB. No-tilling may contribute to recent regional FHB epidemics by providing more inoculum for infection. Deep plough buries Fusarium-infested residues that produce initial inoculum.

An alternate disease control strategy is the deployment of eco-friendly and cost-effective bio-control agents such as microbes to avoid disease epidemics (Khan et al., 2006). Numerous studies were conducted to exploit various bacterial species to reduce FHB disease severity and DON accumulation in wheat. Several bacterial species belonging to genus *Bacillus* and *Pseudomonas;* yeast species belonging to genus *Cryptococcus;* and fungal species belonging to *Trichoderma* have been investigated for potential biological control (Khan et al., 2001; Matarese

et al., 2012; Schisler et al., 2006). Several microbial antagonists of *F. graminearum* have been identified with the potential to reduce FHB (Gilbert & Fernando, 2004; da Luz, 2000). Effective control of seedling blight caused by *F. graminearun*, *F. culmorum*, and *Microdohium nivale* has to be reported from a seed treatment with the bacterial stains, *Pseudomondas fluorescens*, *Pantoea* and *Bacillus cereus*, and the fungus *Trichoderma harzianum* in both glasshouse and field experiments (Dal Bello et al., 2002; Johansson et al., 2003). Biological agent, *Trichoderma* spp. is known to be involved in the competition for nutrient and secreting antifungal compounds to parasitize pathogens (Baffoni et al., 2015; Whipps & Lumsden, 2009). Active research efforts are being carried out to identify effective bio-control agents against FHB and DON, however, no biological organism has been registered for field applications to date.

Several fungicides have been registered in different countries for FHB control in wheat and barley (Bai & Shaner, 2004; Blandino et al., 2006; Mcmullen et al., 2008). However, application times, treatment costs, and weather conditions for application are the major factors determining the effectiveness of the fungicide applications. However, most of these fungicides are effective only when they are applied to cultivars with some levels of resistance (Magan et al., 2002; Paul et al., 2008; Simpson et al., 2001).

FHB resistance mechanisms

Breeding for FHB is a complex task that requires long term investment and dedicated work for years. The major goal of wheat breeding programs is to develop FHB resistant genotypes through recombination among different sources followed by selection for a combination of desired agronomic traits. The success of any breeding program depends upon 1) the availability of genetic variations for the traits of interest in the gene pool and 2) reliable tools to identify the desirable genotypes and move the desirable traits into new cultivars (Buerstmayr

et al., 2019). To explore the genetic variation, screening of a large number of genetic resources for FHB resistance is required. The germplasm can be evaluated for FHB resistance using artificial inoculation in the greenhouse and applying appropriate disease pressure with a mixture of aggressive isolates in a field nursery (Buerstmayr et al., 2009; Mesterhazy, 2003).

Based on plant responses to pathogen infection, FHB resistance in wheat is governed by either active or passive processes. Active resistance refers to plant resistance to fungus through activation of the defense-related genes during early infection of the wheat spike. There are many physiological or chemical processes that may involve limiting the colonization or spread of the pathogen inside plants. Reduction in the propagation of the pathogen has been observed by the inhibiting translocation of DON (Mitterbauer & Adam, 2002), strengthening cell membranes and intensing and rapidly depositing lignin and other compounds surrounding infected cells (Ban et al, 2000, Kang & Buchenauer, 2003). Lignification and cell wall thickening prevent nutrient movement from host to pathogen, arrest the mycelium growth to the neighboring cells, starve pathogen to death and hence reduce disease development (Siranidou et al., 2002; Walter et al., 2010). Whereas passive resistance refers to morphological and developmental features of plants such as plant height, flowering time and anther extrusion that indirectly affect the disease establishment in plants (Anderson, 1948; Buerstmayr et al., 2019; Champeil et al., 2004).

FHB resistance is more likely controlled by a combination of physiological, biochemical, and environmental factors in wheat. Based on the inoculation methods, resistance to FHB was classified into two types, types I and II (Schroeder & Christensen, 1963). Later, Miller et al. (1985) proposed type III, and Mesterházy (1995) elaborated two additional resistance types (types IV and V).

Type I is resistance to the initial infection. Type I resistance in fields is usually more easily affected by plant morphology and environmental interactions than type II resistance, resistance to disease symptom spread within a spike (Buerstmayr et al., 2019). Morphological traits such as plant height, anther extrusion, and days to heading showed a strong association with type I FHB resistance (Lu et al., 2013; Mao et al., 2010; Skinnes, Semagn, Tarkegne, Marøy, & Bjørnstad, 2010). A negative association was observed between plant height (PH), anther extrusion (AE) and FHB susceptibility (He et al., 2016; Lu et al., 2013). Dwarfing genes *RhtB1b* found to be co-localized with anther extrusion QTL and showed increased severity for FHB (Lu et al., 2013). European wheat exhibited a strong association between *RhtD1b* and type I FHB susceptibility (Miedaner & Voss, 2008; Srinivasachary et al., 2008). In general, type I resistance can be evaluated by spraying the conidial spore suspension on flowering spikes (Bai & Shaner, 2004), and later counting the percentage of infected spikelets after seven days of inoculation (Rudd et al., 2001). Inoculated plants are incubated in a chamber at 22 ºC with 100% relative humidity for 60 h and then transferred to benches in a greenhouse experiment. The inoculated spike can be covered individually with a misted polythene bag when a moist chamber is not available (Miedaner et al., 2003). In a field, high relative humidity can be maintained by sprinkler irrigation twice a day for 3-4 days after each inoculation. The disease could be scored starting at 10^{th}-day post-inoculation. Type I resistance is considered an relatively unstable type of resistance and more difficult to be characterized than other types of resistance due to multiple factors (Buerstmayr et al., 2019). Difficulty in accurate quantification of inoculum concentration (Cai, 2016) and interference of type II resistance may all confound type I resistance (Rudd et al., 2001). Type I and type II resistances are not genetically related. Schroeder and Christensen

(1963) assessed seven genotypes for type I and type II resistance and observed that each genotype has only one of the two types of resistance.

Type II resistance has been the most widely studied type of resistance in wheat due to its relatively stable effect and easy evaluation procedure (Bai & Shaner, 2004). This type of resistance relies mainly on active mechanisms of resistance. In greenhouse, Injecting a conidial spore suspension in the single floret of wheat head and assessing the disease severity of the wheat head for FHB symptom spread within the spike allows the estimation of type II resistance (Miedaner et al., 2003). Grain-spawn inoculation is generally used in the field to screening large numbers of germplasm (Rudd et al., 2001). Disease symptoms can be measured as a percentage of an infected spikelets that displays visual symptoms after point inoculation with a conidial spore suspension. Visual observations of symptoms indicate the expension of the disease from the initial infection point, however, a low level of FHB symptoms is correlated with a low level of fungal biomass or toxin accumulation (Rudd et al., 2001).

Type III resistance refers to wheat genotypes that degrade or accumulate low levels of deoxynivalenol (DON). The resistant genotypes have the ability to detoxify the mycotoxin (DON) in the kernels (Miller et al., 1985) and thus have reduced DON concentration than susceptible genotypes. Acetyltransferase enzyme may play a role in the detoxification of trichothecenes (Mitterbauer & Adam, 2002). Physiological changes such as permeability of cell wall and signaling cascades in the cell occurring due to thaumatin-like proteins (*tlp*) may enhance tolerance to trichothecenes (Chen et al., 1999). Lemmens et al. (2005) proposed that resistance of *Fhb1* may be related to toxin degradation and DON can be converted into non-toxic DON-3-glucoside (D3G) as a non-toxic product in the resistant genotypes. However, cloning of *Fhb1* gene did not find any detoxification gene in the QTL region, suggesting that *Fhb1* may not

associate with type III resistance (Su et al., 2019). Several factors may affect the accurate measurement of type III resistance. Combine harvesting may blow out lightweight infected kernels during threshing. Type III resistance is evaluated based on the measurement of DON concentration in the harvested grains. Toxin content can be measured using thin-layer chromatography, high-pressure liquid chromatography (HPLC), gas chromatography-mass spectrometry (GC-MS), ELISA and NIR etc. (Dowell et al., 1999; Peiris et al., 2010; Warth et al., 2015; Lemes da Silva et al., 2019). Different methods vary in toxin measurement accuracy.

Type IV resistance is to measure the proportion of kernels damaged by *Fusarium* in a grain sample, also called *Fusarium* damaged kernels (FDK) (Mesterházy, 1995). Commonly visual scoring method is used to identify genotypes having type IV resistance, in which a grain sample is divided into two fractions: sound kernels (healthy infected) and FDK including lightweight, shriveled, discolored and pinkish kernels (Argyris et al., 2003). The resistant genotypes show higher kernels weight (1000 grain weight) and fewer whitish and shriveled kernels than susceptible genotypes (Jin et al., 2013). Yield components (1000 grain weight and total yield) and the percentage of FDK are the most efficient way of identifying resistant cultivars with low infection rates (Klahr, et al., 2007). Many studies demonstrated significant positive correlations between type II, type III and type IV resistance (Bai et al., 2001; Jin et al., 2013; Lemmens et al., 2004; Paul et al., 2005; Wegulo et al., 2011), thus can be concluded that type II resistance is a useful estimate for types III and IV resistance. Normally type III and type IV resistances are correlated because FDK also have higher DON concentration (Jin et al., 2013).

Type V is FHB tolerance that is determined by measuring the reduction of grain yield due to FHB (Mesterházy et al., 1999). The tolerant genotypes exhibiting type V resistance show a minimum reduction in grain yield and quality under FHB epidemics (Zhou et al., 2002). The

tolerant genotypes may have less yield reduction than the susceptible genotypes, but may still high mycotoxin content due to the large number of toxin contaminated grains (Foroud et al., 2012; Mesterházy et al., 1999). The screening method for FHB tolerance may be inconsistent across environments and years (Mesterházy et al., 1999). Grain yield of field plots infected with FHB compared to control plots having no disease allows the assessment of type V resistance, however, this type of experiments are hard to conducted because it is not possible to put the both experiments in the same field that has the same growing conditions. Thus, more accurate, effective, quick and inexpensive evaluation methods are required to screen germplasms, identify sources of resistance and select elite materials for breeding programs.

Genetics for FHB resistance

FHB resistance in wheat is a complex trait that is controlled by multiple genes and affected by environments (Cai et al., 2016; Ruckenbauer et al., 2001). Resistance to FHB in wheat is non-race-specific (horizontal) that provides resistance against all species of *Fusarium.* (Tóth et al., 2008). Significant variations in genetic resistance have been observed among the wheat and its relatives (Bai et al., 2000; Cai et al., 2016; Miedaner et al., 2003; Cuthbert et al. 2007). Some studies reported transgressive segregation and demonstrated additive effects when several QTLs were moved from different sources to a single genotype (Bai et al., 2018; Liu et al., 2005; Rudd et al., 2001; Skinnes et al., 2010; Somers et al., 2006). Liu et al. (2005) estimated that a minimum of two to four genes were involved in disease resistance in ‘Ernie’ while another study reported that resistance to FHB might be regulated by at least one to three genes depending on the sources of resistance, gene pools and its origins (Bai et al., 2000). To date, QTLs for FHB resistance have been located on all chromosomes of different sources (Gervais et al., 2003; Kolb et al., 2001; Rudd et al., 2001; Snijders, 1990; Yu, 1982; Liu et al. 2009).

Recently, Su et al. (2019) and Li et al. (2019) cloned a histidine-rich calcium-binding protein as the candidate for *Fhb1* that showed a major effect on FHB resistance. However, Su et al. (2019) reported that the resistance allele with a large sequence deletion in the start codon of the gene resulted in the loss-of-function mutation to confers FHB resistance, whereas Li et al. (2019) reported the gain-of-function mutation of *Fhb1* by creating a new start codon in the upstream region of the gene. In addition, Rawat et al. (2016) cloned a pore-forming toxin like (*PFT*) gene at *Fhb1* locus from Chinese cultivar 'Sumai 3'. They suggested that *PFT* may have a broad-spectrum resistance against multiple isolates of *Fusarium*. Further studies may be needed to solve the discrepancy among the three studies (Lagudah & Krattinger, 2019).

Genetic variation for FHB resistance

In hexaploid wheat (AABBDD, 2n=6x=42), highly resistant sources to FHB have been reported early in China, Japan and Brazil. In China, Sumai 3 and its derivatives are the most commonly used source of resistance (Table 1.1) (Bai et al., 2003; He et al., 2014; Rudd et al., 2001). A QTL, designated as *Fhb1,* on chromosome arm 3BS has been reported repeatedly from Sumai 3 and its derivative Ning7840, and explained the largest phenotypic variation for type II resistance among the QTLs reported to date (Anderson et al., 2001; Bai et al., 1999; Waldron et al., 1999), thus have been a major source of resistance to FHB for improving FHB resistance worldwide (Zhu et al., 2019; Humphrey et al., 2001). Chinese landraces Wangshuibai, Huangcandou, Haiyanzhong, Baishanyuehuang and Huangfangzhu also show a higher level of type II resistance (Cai & Bai, 2014; Cai et al., 2019; Li et al., 2011). In addition, Japanese wheat cultivars Nyu Bai, Yumechikara, and Nobeokabouzu-komugi also possess resistance to FHB (Rudd et al., 2001; Urrea et al., 2002, Nishio et al., 2016). However, the direct use of unadapted germplasm as resistant parents in breeding programs is not very successful due to the linkage

drag of poor agronomic traits (Bai et al., 2018). A Brazilian wheat cultivar Frontana (Mardi et al., 2006; Steiner et al., 2004a), a Korean wheat cultivar Chokwang (Yang et al., 2005), and Ernie, Roane and Freedom from the United States (Jin et al., 2013; Liu et al., 2005; Rudd et al., 2001) have also been reported to have a high level of FHB resistance.

In European winter wheat, a large number of QTLs with minor to moderate effects were found controlling FHB resistance; however, a single common resistance QTL near *Rht-D1* locus for FHB was found in cultivars Biscay, Cansas, History, Renan, Arina and Pirat together with different minor QTLs in each cultivar (Häberle et al., 2009; Paillard et al., 2004; Schmolke et al., 2008; Somers et al., 2003; Srinivasachary et al., 2008; Steiner et al., 2004). Resistant sources obtained from the Asian countries may carry different QTLs for type II resistance (Chen et al., 2006; Jiang et al., 2007; Jin et al., 2013; Shen et al., 2003). QTL mapping studies which used Asian resistant sources have identified *Fhb1* with a large effect (Chen et al., 2006) and a marker *Xbarc133* was found to be tightly associated with the QTL (Bernardo et al., 2012; Hao et al., 2012; Liu et al., 2008). Populations developed from resistant parents W14 (Chen et al., 2006), Huapei 57-2 (Bourdoncle & Ohm, 2003), Ning 894037 (Shen et al., 2003), CJ 9306 (Jiang et al., 2007a) with different susceptible parents displayed the same haplotype as Sumai 3 at five SSR marker loci around *Fhb1* locus (Liu & Anderson, 2003a, 2003b), suggesting that all these resistant sources possess the same FHB resistance allele as that of Sumai 3, although Ning 894037 was reported to be derived by somaclonal variation from susceptible cultivar Yangmai 3 (Shen et al., 2003). Similarly, Cuthbert et al. (2007) mapped an additional resistance gene *Fhb2* on chromosome 6BS using a RIL population derived BW278 (AC Domain*2/Sumai 3).

Many researchers have evaluated tetraploid wheat for FHB resistance. New sources of resistance were not found due to the narrow genetic base for FHB resistance in durum wheat

germplasm (Prat et al., 2017). In other tetraploid wheat, Miller et al. (1998) reported that 10 out of 282 accessions of wild emmer evaluated for FHB resistance showed better type II resistance (spread of infection) than existing best durum wheat. Buerstmayr et al. (2003) tested 151 accessions of wild emmer *T. turgidum* from a geographical area close to the Mediterranean center of wheat origin and identified eight accessions with moderate resistance to FHB. Similarly, 16 accessions of *T. turgidum* subspecies carthlicum and dicoccum displayed moderate resistance to FHB, (Oliver et al., 2008). Huhn et al. (2012) evaluated a collection of Tunisian durum genotypes and reported five genotypes having type II resistance. Cluster analysis based on genetic markers deciphered the uniqueness of Tunisian lines from the known genetic resources of resistance. Those lines were genetically distant, particularly Tunisian 7 was the most resistant among all, indicating the presence of genetic diversity and their potential to be used as a novel source of resistance for FHB. Therefore, after extensive screening of tetraploid wheat germplasm for FHB resistance, only a small number of genotypes showed resistance and none of the resistance sources have a comparable level of resistance as Sumai 3 and its derivatives (Zhao et al., 2018). Incorporation of alien resistance fragments from related wheat species such as bread wheat (*Triticum aestivum* L.), wild emmer (*Triticum dicoccoides*), cultivated emmer (*Triticum dicoccum*) into durum wheat has been attempted (Prat et al., 2015), and recently *Fhb1* has been successfully introgressed into durum wheat. Advance lines with a high level of FHB resistance has been obtained (Prat et al., 2017). More recently, Zhao et al. (2018) successfully introgressed FHB resistance from hexaploid wheat PI 277012 into durum wheat.

Wheat wide relatives are important sources of FHB resistance for wheat breeding. To date, a large number of accessions from wild relatives of common wheat have been tested for FHB resistance. Mujeeb-Kazi et al. (1983) reported a highly resistant accession of *Elymus*

giganteus L (2n=4x=JJNN). Two *Roegneria* species*, R. kamoji* (syn. *Agropyron tsukushiense*, 2n=6x=42) and *R ciliaris* (syn. *A. ciliare*, 2n=4x=28), were identified to have highly resistant germplasm (Weng and Liu, 1989; Weng and Liu, 1991). The resistant accessions were also identified in the genera *Roegneria, Hystrix*, *Elymus, Kengyilia* and *Agropyron* collected from different geographical regions (Wan et al., 1997) and some of the accessions have been utilized in wheat breeding programs around the globe to develop FHB resistant wheat cultivars (Table 1.1). Wheat wild relatives such as *Thinopyrum elongatum*, *Thinopyrum ponticum*, *Thinopyrum intermedium*, *Aegilopes speltoides*, *Elymus tsukushiensis* and *Leymous racemosus* have been used to improve the FHB resistance in wheat by a cytogenetic modification to create deletion, substitution and translocation lines (Cai et al., 2008; Cainong et al., 2015; Guo et al., 2015; Oliver et al., 2005; Qi et al., 2008; Wang et al., 2020), and the alien segments have been introgressed from these wild species to elite wheat lines. Recently, Wang et al. (2020) successfully cloned *Fhb7* by assembling the genome of *Thinopyrum elongatum* and revealed that *Fhb7* encodes glutathione S-transferase (GST) that detoxify mycotoxin produced by different species of *Fusarium* to provide broad-spectrum resistance against *Fusarium* species. However, most of these alien resistant fragments possess poor agronomic traits, their effects in different wheat backgrounds need to be evaluated before they can be used as resistant parents in wheat breeding programs (Bai et al., 2018).

QTL mapping for FHB resistance

High-density genetic maps that show the order and relative genetic distance of genome-wide markers are required for QTL mapping (Collard et al., 2005). The genetic maps can be constructed using different types of markers such as restriction fragment length polymorphism (RFLP), simple sequence repeat (SSR), diversity arrays technology (DArT), and single

nucleotide polymorphism (SNPs) (Li et al., 2015; Lin et al., 2015). The recombination frequencies among markers and genes in the segregating mapping population are used to calculate genetic distances (cM) among markers or genes (Peterson, 1996). The positions of markers and QTL intervals can be located in the maps. Integration of genetic information from multiple genetic maps from the same species allows the construction of a unique consensus map to generate more accurate estimation of marker positions for QTL mapping and map-based cloning (Somers et al., 2004; Wen et al., 2017). Many genetic maps have been constructed using SSR markers in wheat (Röder et al., 1998; Somers et al., 2004). However, due to limitations in the number of available SSR markers and high cost for marker development, SSR is not suitable for the construction of high-density genetic maps required for QTL fine mapping and gene cloning (Wang et al., 2014). The quick development of next-generation sequencing technologies makes it possible for rapid identification of an unlimited number of genome-wide SNP markers at an affordable cost for fine mapping, genome-wide association study, genomic selection and gene cloning. Particularly, genotyping-by-sequencing (GBS) is a robust and cost-effective technique to sequence complex genome like wheat (Poland et al., 2012). GBS uses restriction digestion and PCR amplification to reduce genome complexity (He et al., 2014). To date, GBS has been widely used to wheat QTL mapping, genomic selection and genetic diversity study (Elshire et al., 2011), and also QTL mapping for FHB resistance (Marcio et al., 2016; Sun et al., 2016).

RILs and DH populations developed from two parents are often used to discover QTLs in wheat (Buerstmayr et al., 2019) since these are permanent populations and can be evaluated phenotypically over multiple environments and years to get reliable data for QTL mapping. A classic QTL mapping experiment is to use a genetic map and phenotypic data of the trait of

interest to detect the linkage between the marker and trait data through a whole-genome scan. This method has been extensively used to discover QTLs associated with FHB resistance. The parents selected for developing the mapping population should have contrast in the trait of interest with one as the resistant parent and another as the susceptible parent to FHB. A relatively large mapping population (>150 lines) should be used to reduce false positive QTLs (Mohan et al., 1997) and a high-resolution map is essential to ensure most of the QTLs being covered in the map (Collard et al., 2005).

Several methods can be used to map QTLs, including single marker analysis (SMA), simple interval mapping (SIM), composite interval mapping (CIM) and multiple interval mapping (MIM) (Tanksley, 1993). Among these methods, SMA is the easiest method to identify QTL utilizing an association between a single marker and the QTL. The statistical methods such as t-test, analysis of variance (ANOVA) and linear regression all can be used for the analysis (Collard et al., 2005; Young, 1996). However, SMA can not provide QTL locations in a chromosome. SIM however can determine a QTL interval in a chromosome. However, SIM does not consider the possible effects of other QTLs in backgrounds, it can not separate closely linked QTLs and estimate the interaction between QTLs. CIM uses the background markers as cofactors when conducting QTL mapping, which can detect multiple loci and overcome the limitation of SIM (Manly & Olson, 1999). Finally, MQM adds higher statistical power to map QTL in a three-step procedure. In the first step, missing data is augmented, then important markers are selected by multiple regression and backward elimination and finally, QTL is moved along the chromosome using these pre-selected markers as a cofactor leaving the markers in the interval under study. It maps QTLs using the most informative model of ‘maximum likelihood’ and internally control the false discovery rates. It keeps into account the multiple QTLs and

genotype by environment interactions (Broman, 2009; Wang et al., 2006, Arends et al., 2014). All the QTL mapping methods except SMA use the logarithmic of odds (LOD) (Lander & Kruglyak, 1995) or likelihood ratio statistics (LRS) (Haley & Knott, 1992) to determine the significance of QTLs. The LOD and LRS thresholds can be generated using 1000 permutation tests with 95% confidence interval to claim significant QTLs. Based on the phenotypic variation explained, QTLs can be categorized into major and minor QTLs. The major QTL explains a larger percentage of the phenotypic variation, (usually $R^2 > 10$) while minor effect QTL explains a relatively smaller proportion of the phenotypic variation (usually $R^2 < 10$) (Collard et al., 2005).

One of the main objectives of QTL mapping is to identify tightly linked markers to resistance genes or QTLs to monitor the movement of these QTL or genes in progeny when they are transferred into well-adapted cultivars in breeding. Usually, a QTL can be identified using the biparental population to identify the QTL associated with a target trait and then the tightly linked markers can be converted into breeder-friendly markers for breeding application. However, these markers were developed based on a single biparental population and may be population specific and not be diagnostic in other populations. Thus, these markers need to be further validated in different populations or in a diversity panel for association with target QTLs.

Buerstmayr et al. (2009) summarized QTL information from 52 QTL mapping studies and then Buerstmayr et al. (2019) reviewed 101 additional QTLs studies conducted in the recent decade including biparental mapping population, association mapping panels, validation of already reported QTLs, fine mapping and cloning of QTLs. Since the first QTL study for FHB resistance in wheat published by Waldron et al. (1999) 500 resistant loci have been reported on all wheat chromosomes (Buerstmayr et al., 2019) with varying effects based on different genetic

backgrounds. Among hundreds of resistant loci claimed by authors as major effect QTLs, seven QTLs have a large effect on FHB resistance and validated across multiple populations and have been formally named as *Fhb1, Fhb2, Fhb3, Fhb4, Fhb5, Fhb6, Fhb7.*

Fhb1 on chromosome 3BS derived from 'Sumai 3' and 'Ning7840' was the first mapped major QTL for FHB resistance (Bai et al., 1999; Waldron et al., 1999). This QTL was validated to confer type II resistance (Anderson et al., 2001b; Liu et al., 2008b; Yang et al., 2005) and is the most widely used QTL in wheat breeding programs (Bai et al., 2018; Bernardo et al., 2012; Lu et al., 2011; McMullen et al., 2012; Xie et al., 2007). *Fhb1* has been incorporated through MAS in elite lines in the U.S.A., Canada, Germany and Australia (Del Blanco et al., 2003; McCartney et al., 2007; Miedaner, 1997; Xie et al., 2007). Cuthbert et al. (2006) formally named it as *Fhb1*. Liu et al. (2008b) further identified tightly linked marker *UMN10* to *Fhb1* by positional cloning and this marker has been widely used to transfer *Fhb1* QTL into elite cultivars and varieties in U.S. wheat breeding programs with great success (Bai et al., 2018). Shi et al. (2008) analyzed DNA marker polymorphism on already developed pyramided lines and three donor parents to identify the number of QTLs in WSY with FHB resistance. The marker analysis results indicate that four FHB resistance QTLs from Sumai 3, four QTLs from WSB, and one QTL from 'Nobeokabouzu' pyramided to WSY wheat line. Several independent research attempts have been made to clone *Fhb1* and decipher the underlying mechanisms of *Fhb1* resistance (Bernardo et al., 2012; Lagudah & Krattinger, 2019; Liu et al., 2008a). Recently two candidate genes have been cloned for *Fhb1* (Lagudah & Krattinger, 2019; Li et al., 2019; Rawat et al., 2016; Su et al., 2019). Highly diagnostic markers for *Fhb1* have been developed using the candidate gene sequence and validated in a worldwide wheat collection, thus are valuable tools for pyramiding *Fhb1* with other genes/QTLs in marker-assisted breeding (Su et al., 2018).

Another large effect QTL for FHB resistance originated from Sumai 3 is *Fhb2* on 6B chromosome. This QTL explained up to 21% of the phenotypic variance for type II FHB resistance and DON accumulation (Cuthbert et al., 2007; Jia et al., 2018a; Li et al., 2012; Semagn et al., 2007; Yang et al., 2005; Yang et al., 2003). The loci were localized in the interval between *Xgwm508* and *Xbarc79* (Liu et al., 2009b). The putative candidate genes of *Fhb2* were found to be associated with cell wall reinforcement and detoxification of DON (Dhokane et al., 2016).

Two major effect QTLs for type I resistance were mapped on the long arm of chromosome 4B and the short arm of chromosome 5A of WSB, designated as *Fhb4* and *Fhb5,* respectively (Jia et al., 2005; Jia et al., 2018b; Xue et al., 2010, 2011). These QTLs explained 17.5% and 30% of the phenotypic variation, respectively. Fine mapping of the QTL on 4B chromosome revealed that the resistant NILs have significantly lower infection than the succeptible NILs and *Xhbg226* and *Xgwm149* were the flanking markers that can be used in marker-assisted selection (Xue et al., 2010). Another QTL for type I FHB resistance on 5AS chromosome was finely mapped between *Xgwm304* and *Xgwm415,* and the resistance class showed significantly less infectin compared to the succeptible class (Xue et al., 2011). Liu et al. (2009b) found an association of 5AS QTL with type II resistance in multiple mapping studies. Meta-analysis of data from 11 independent QTL-mapping studies for FHB resistance located the QTL in one interval on chromosome 5AS from the different studies, thus this is repeatable QTL for improvement of FHB resistance in wheat breeding programs through marker-assisted selection (Buerstmayr et al., 2009).

The other three major QTLs are *Fhb3*, *Fhb6* and *Fhb7* identified from alien species. These QTLs have been transferred into wheat (Bai et al., 2018). Qi et al. (2008) reported the

Fhb3 from *Leymus racemosus* and then introgressed the alien fragment to the long arm of chromosome 7A of wheat as a Robertsonian translocation T7AL.7Lr#1S. This QTL showed a similar level of resistance as Sumai 3 in some wheat backgrounds. Pyramiding of *Fhb1, Fhb2* and *Fhb3* through marker-assisted backcrossing scheme revealed the additive effects of QTLs (Brar et al., 2015). Another major effect QTL *Fhb6* has been mapped to the subterminal region of the short arm of chromosome 1Ets#1S *Elymus tsukushiensis*. It has been successfully introgressed to 1AS chromosome of wheat via *ph1b*-induced homoeologous recombination and the selected lines had percentage of symptomatic spikelets ranging from 7 to 35% (Cainong et al., 2015). Another alien gene fragment is *Fhb7* derived from *Thinopyrum ponticum.* This gene has been successfully introgressed through 7DS.7el2L Robertsonian translocation. The QTL is flanked by *XsdauK66* and *Xcfa2240* and explains 15-32.5% of the phenotypic variation for resistance against FHB (Guo et al., 2015). Recently, *Fhb7* has been cloned to encode a glutathione S-transferase (GST) that is involved in the detoxification of trichothecenes and confers resistance against multiple species of *Fusarium* (Wang et al., 2020).

Association of morphological traits with FHB resistance

Previous studies have repeatedly shown an association of FHB severity with several morphological traits. Plant height (PH) is one of the most frequently reported traits associated with FHB resistance under both field and greenhouse conditions. Usually, taller genotypes are more resistant to FHB compared to short one (Hilton et al.,1999; Rudd et al., 2001; Steiner et al., 2004a). This may be due to that the spikes of short plants are closer to the soil surface where the *Fusarium* spores come from and receive more spores from soil debris than taller plants (Buerstmayr et al., 2019). Besides, microclimate such as humidity, spike temperature and duration of leaf wetness of plants are different between the two types of plants, which creates

different environments on them for FHB initiation. The shorter plants near the soil surface may have a lower temperature, higher humidity, reduced air circulation that results in more disease infection (Buerstmayr & Buerstmayr, 2016; Hilton et al., 1999; Jones et al., 2018). A number of QTLs for type II resistance have been colocalized with reduced height (*Rht*) loci (Draeger et al., 2007; Gervais et al., 2003; Häberle et al., 2009; Paillard et al., 2004). Srinivasachary et al. (2009) observed that lines carrying semi-dwarf allele *Rht-B1b* were more resistant to FHB. Another study reported the ability of dwarf lines to detoxify DON accumulation (Saville et al., 2012). The gibberellin-insensitive semi-dwarfing alleles *Rht-B1b* on 4B and *Rht-D1b* on 4D chromosomes to increase the FHB susceptibility have been linked to low anther extrusion in plants (He et al., 2016). *Rht-B1b* and *Rht-D1b* are more frequently present in modern germplasm than in landraces (Buerstmayr et al., 2019). These genes are also involved in the development of stiff and compact spike structure by interfering cell elongation (Boeven et al., 2016; Buerstmayr & Buerstmayr, 2016; Okada et al., 2019). Overall, compact spikes strongly influence FHB severity (Giancaspro et al., 2016; Yi et al., 2018).

Heading date (HD) may also have an influence on FHB resistance as the temperature and humidity at the time of anthesis play a critical role in disease development and is necessary to understand the role of HD with disease severity. The plant growth habit is controlled by *Vrn* genes and together with *ppd* and earliness per se loci control the HD. Several studies observed the overlapping of these genes with FHB resistance QTL (Grogan et al., 2016; Shcherban & Salina, 2017; Würschum et al., 2018). Although multiple studies reported overlapping QTLs for HD and FHB resistance but no systematic association was observed between HD and disease infection, thus this association may be controlled by environmental factors especially weather conditions at the time of anthesis (Buerstmayr et al., 2019). He et al. (2016) observed that a

significant QTL for FHB resistance at the locus of *Vrn-A1* disappeared when heading date and plant height were used as a covariate in the analysis. More critical evidences are required to understand the genetic role of heading date and flowering time on FHB.

Conventional breeding approaches for FHB resistance

Conventional breeding for FHB resistance comprises of three major phases (i) identifying resistance sources from available elite breeding lines, landraces or wild relatives, (ii) creating variations by crossing resistant and susceptible parents (iii) selecting progenies with acceptable combinations of FHB resistance and other agronomic traits such as yield and quality. Conventional breeding mostly uses either pedigree or bulk selection based on early and advanced generation performance in multi-location and multi-years field trials (Caligari, 2001; Mwadzingeni et al., 2016). Several FHB resistant hard red spring wheat cultivars such as Alsen, Steele-ND and Glenn have been developed and grown in the Northern Great Plains through marker-assisted selection since 2000 (ElDoliefy et al., 2020; Ma et al., 2009; Mergoum et al., 2011). In 1960s, China initiated conventional breeding programs to develop FHB resistant cultivars. As a result, the cultivar 'Sumai 3' was developed in 1970s, and has been widely used as a resistant parent in many wheat breeding programs in China and globally. Subsequently, many resistant lines such as Ning series, Fan 60096, Fu 5125 and Fu 5114 were developed using 'Sumai 3' as a resistant parent (Bai & Shaner, 2004; Zhu et al., 2019). Since then, 'Sumai 3' and its derivatives have been widely used for the improvement of type II resistance in wheat breeding programs in the U.S.A., Canada and many other countries (Zhu et al., 2019). Although, classical breeding is the base for new cultivar developments, many challenges associated with it including pleiotropic effect, linkage drag, and adaptation of elite lines. Moreover, selection of lines with multiple FHB resistance genes is very difficult to evaluate through conventional approaches.

Some of these challenges can be overcome by implementing new advanced technologies like marker-assisted selection in conjunction with conventional strategies.

Molecular and genomic breeding to improve FHB resistance

In addition to classical breeding, molecular and genomic approaches have also been used to hasten FHB resistance breeding in wheat. Modern tools for genetic research include genomics, phenomics, proteomics, metabolomics, chromosome engineering and gene editing etc, all aid plant breeders and geneticists in dissecting the genetic architecture of complex traits. Over the past two decades, marker-assisted selection has been widely used for indirect selection owing to the availability of reliable, co-dominant, closely linked, and cost-effective molecular markers for quick selection of critical genes/QTLs for beneficial traits. MAS can be performed at any stage in a breeding cycle and allows the pyramiding of multiple resistance loci in a single resistant plant (Buerstmayr et al., 2019). In a breeding program, QTLs with stable major effect on a specific trait of interest are desired for gene pyramiding, so MAS need only handle a minimum number of QTLs to yield higher gain (Poland & Rutkoski, 2016). To date, several hundreds of QTLs have been mapped, markers associated with these QTLs have been reported and some of the markers have been used in MAS to enhance FHB resistance (Table 1.2). Pyramiding QTLs through MAS leads to the development of single cultivar having resistance QTLs from different genetic backgrounds. Some QTLs from non-adapted germplasm may have linkage drag that may affect yield and end-user quality of new cultivars developed from these sources. Recently, Bakhsh et al. (2019) evaluated the agronomic performance of 21 Wesley BC_2F_2 hard red winter wheat lines with *Fhb1* introgressed and reported that seven of those lines showed similar performance to the recurrent parent Wesley for most of the traits including grain yield. Some

lines have the potential to be released as a FHB resistant cultivar for production and used as a parent in wheat breeding programs.

Recently, genomic selection (GS) along with double haploid technology have boosted the genetic gain by shortening the breeding cycle. GS is a predictive approach that utilizes all available marker information to capture genetic variation for even undetected minor QTLs (Heffner et al., 2009). It has the potential to enhance the breeding efficiency of complex traits such as yield and disease resistance by utilizing the genome-wide marker data to predict FHB resistance (Buerstmayr et al., 2019). In the era of next-generation sequencing, an urgent need has arisen for large-scale data analyses, advanced statistical techniques, proper population designs and high-throughput and precised phenotyping to enhance the breeding efficiency and genetic gain (Nadeem et al., 2018; Rasheed et al., 2017; Schrag et al., 2018; Tardieu et al., 2017; Xu et al., 2017). To control FHB in farmer's fields, an integrated approach including growing resistant cultivars, improvement of cultural practices, application of fungicides and disease forecasting need to be used. Among them, the use of resistant cultivars is the most sustainable and environmental friendly approach to control the disease.

Table 1.1. Resistant sources of FHB along with their pedigrees, origins and resistance reactions

Accession	Pedigree	Origin	FHB resistance
Sumai 3	Funo/Taiwanmai	China	R
Ning 7840	Aurora/Anhui 11//Sumai 3	China	MR
Ning 8331	Yangmei 4/(Aurora/Anhui 11//Sumai 3)	China	MR
Ning 8026	Aurora/Sumai 3//Yangmai 2	China	MR
Ning 8428	75-6711/Lovrin//Ning7840	China	MS-MR
Ning 894037	Somaclonal variant from Yangmai 3	China	MR
Wangshuibai	Landrace from Jiangsu Province, China	China	R
Hongheshang	Landrace from Zhejiang Province, China	China	R
Yangmai158	Yangmai 4/St 1472/506	China	R
Yangangfangzhu	Unidentified	China	R
Yangmai 5	(Nanda 2419/Triumph//Funo)/St 1472/506	China	MR
Yangmai 4	Nanda 2419/Triumph//Funo	China	MR

Fanshanmai	Unidentified	China	MR
Zheng	Fusuihunag/Yougimai	China	MR
Xinzhongchang	Unidentified	China	MR
Funo	Duecentodieci/Demiano	Italy	R
Nobeokabozu	Landrace	Japan	R
Nyu Bai	Unidentified	Japan	R
Frontana	Fronteira/Mentana	Brazil	R
Blackbird	Unidentified	France	MR
Yumechikara	Satsukei 159/KS 831957//Kitanokaori	Japan	MR
Haiyanzhong (HYZ)	Landrace	China	R

*R: Resistant; MR: Moderate resistant; MS: Moderate succeptible

Table 1.2. Quantitative trait loci (QTLs) detected/identified in various studies for FHB resistance

Parent cross (Population type)	**FHB trait*** **(Inoculation method)****	**Resistant parent (Chr. displayed QTLs)**	**Marker type**	**Phenotypic variance (%)**	**Reference**
Sumai 3 × Stao (RIL)	Type II resistance (SFI in GH)	Sumai 3 (3BS, 6BS), Stao (2AL, 4B)	*Xcdo981*	15.4	(Waldron et al., 1999)
Ning 7840 × Clark (RIL)	Type II resistance (SFI in GH)	Ning 7840 (3BS)	AFLP	60	(Bai et al., 1999)
ND2603 × Butte56 (RIL)	Type II resistance (SFI in GH)	ND2603 (3BS, 6AS, 3AL)	*Xgwm493/Xgwm533*	24.8 - 41.6	(Anderson et al., 2001)
Sumai 3	Type II resistance	3BS, 6BL	RFLP	63	(Kolb et al., 2001)
Langdon Dicc-3A × Lagdon (RIL)	Type II resistance (SFI in GH)	*Triticum dicoccoides* (3AS)	*Xgwm2*	37	(Otto et al., 2002)
CM-82036 × Remus	Type II resistance	3BS, 5A	*Xgwm533-Xgwm493*	60	(Buerstmayr et al., 2002)
Ning 894037 × Alondra (RIL)	Type II resistance (SFI in GH&FE)	Ning 894037 (3BS, 6BS), Alondra (2DS)	*Xbarc133–Xgwm493*	42.5	(Shen et al., 2003b)
Patterson × F201R (RIL)	Type II resistance (SFI in GH)	F201R (1B, 3A, 5A), Patterson (3D)	*Xbarc76, Xgwm674*	3.6 to 13.4	(Shen et al., 2003c)
Wuhan 1 × Nyu Bai (DH)	Type II, III resistance (SFI in GH & SI in FE)	Wuhan (2DL, 4BS), Nyu Bai (2D, 4BS, 5AS, 2 QTL on 3BS)	*Xgwm539, Xwmc238, Xgwm533*	4 to 13	(Somers et al., 2003)

Sumai 3 × ND Lines	Type II resistance (SFI)	*Fhb1*	*Xgwm533-Xgwm274*	-	(del Blanco et al., 2003)
Huapei 57-2 × Patterson (RIL)	Type II resistance (SFI in GH & FE)	Huapei 57-2 (3BS, 3BL, 3AS), Patterson (5BL)	*Xbarc59-Xbarc133*	7.1 to 23.6	(Bourdoncle and Ohm, 2003b)
CM-82036 × Remus (DH)	Type II resistance (SI in FE)	CM-82036 (3BS, 5A)	*Xgwm533–Xgwm293*	20.5 to 29.1	(Buerstmayr et al., 2003a)
Renan × Recital (RIL)	Type II resistance (SI in FE)	Renan (2BS, 5AL, 2A)	*Xgwm639b, Xgwm374*	14-19.2	(Gervais et al., 2003)
Arina × Forno (RIL)	Type II resistance (SI in FE)	Arina (6DL, 3BL, 5AL, 4AL, 2AL), Forno (5BL, 3AL, 3DS)	*Xcfd19a–Xcfd47, Xgwm371*	6.3 to 22.1	(Paillard et al., 2004)
Remus × Frontana (DH)	Type I, II resistance (SI in FE)	Frontana (3A, 5A, 2B, 6B), Remus (1B, 2A)	*Xdupw227–Xgwm720*	5.5 to 16.2	(Steiner et al., 2004)
Nanda2419 × Wangshubhai (RIL)	Type II resistance (SFI in FE)	Wangshubhai (3BS, 6B)	*Xgwm533.1-Xwmc539*	13.6-17.8	(Lin et al., 2004)
Wangshubhai × Wheaton (RIL)	Type II resistance (SFI in GH)	Wangshubhai (3BS, 1B, 7A, 3BS)	*Xbarc147-Xgwm759*	3-37.3	(Zhou et al., 2004c)
Langdon × T.dicoccides	Type II resistance	3A	SSR	60	(Hartel et al., 2004)
Seri82 × Wangshubhai (RIL)	Type II resistance (SI in FE)	Wangshubhai (3BS, 2DL)	*Xwmc96–Xgwm304*	8.14-16.7	(Mardi et al., 2005)
Seri82 × Frontana (RIL)	Type II resistance (SI in FE)	Seri82 (1BL), Frontana (3AL, 7AS)	*Xe38m50.10 -Xgwm233*	7.6 to 7.9	(Mardi et al., 2006)

Wangshubhai × Alondra (DH)	Type II resistance (NI in FE)	Wangshubhai (2D, 3BS, 4B, 5B, 7A)	*Xgwm443–Xbarc32*	9.9 to 13.3	(Jia et al., 2005)
Dream × Lynx (RIL)	Type II resistance (SI in FE)	Dream (6AL, 2BL, 7BS), Lynx (1B)	*Xs25m15.187-Xs23m21.497*	11 to 21	(Schmolke et al., 2005),
HsTm4A × Hobbit-sib (DH)	Type I resistance (SI in polytunnel exp.)	T. macha (4AS)	*Xgwm165*	-	(Steed et al., 2005)
Chokwang × Clark (RIL)	Type II resistance (SFI in GH)	Chokwng (5DL, 4BL, 3BS)	*Xbarc239, Xbarc1096*	4.7 to 10.5	(Yang et al., 2005a)
DH181 × AC Foremost (DH)	Type I, II, IV (SFI in GH & SI in FE)	DH181 (2DS, 3BS, 6BS, 7BL, 3BC, 4DL, 5AS, 1DL), AC Foremost (3A)	*Xwmc397, Xwmc264–Xwmc428*	5.8 to 24	(Yang et al., 2005b)
Patterson × Goldfield (RIL)	Type I resistance (GP in FE)	Goldfield (2BS, 2B, 7B)	*Xbarc200–Xgwm210*	7 to 29	(Gilsinger et al., 2005)
Ning 7840 × Clark (RILs	Type II resistance	Ning 7840 (1AL, 3BS)	*Xsrst.3B1*	5.5-12.7%	(Guo et al., 2003)
CS-SM3-7ADS × Annong8455 (RIL)	Type II resistance (SFI in GH & SI in FE)	CS-SM3-7ADS (3BS, 2D, 4D)	*Xgwm533–Xgwm493*	10.8 to 30.2	(Ma et al., 2006a)
Wangshubhai × Anong8455 (RIL)	Type II, Type III (SFI in FE)	Wangshubhai (3BS, 2A, 5A)	*Xgwm533.1 – Xbarc133*	6 to 17	(Ma et al., 2006b)
Seri82 × Frontana (RIL)	Type II resistance (SI in FE)	Seri82 (1BL), Frontana (3AL, 7AS)	*Xgwm720–Xgwm1121*	7.6 to 7.9	(Mardi et al., 2006)
Blackbrid × Strongfield (DH)	Type II resistance (SFI in GH)	*T. durum* cv. Strongfield (2BS), *T. carthlicum* cv. Blackbrid (6BS)	*Xwmc474–Xwmc175, Xgwm518*	23-26	(Somers et al., 2006a)

Sumai 3 × Stao (RIL)	Type II resistance (SFI in GH)	Sumai 3 (3BS, 6BS), Stao (2AL, 4BS)	SSR	7.2 to 41.6	(Hill-Ambroz et al., 2006)
W14 × Pion2684 (DH)	Type II resistance (SFI in GH & SI in FE)	W14 (2 QTL on 3BS, 5A)	*Xgwm493–Xgwm533*	10 to 33	(Chen et al., 2006b)
Ning 7840 × Clark (RIL)	Type II resistance (SFI in GH)	Ning 7840 (3BS, 2BL, 2AS)	SSR	3 to 52	(Horsley et al., 2006b)
Sumai 3 × Fundulea 201R	Type II resistance	3A	SSR	-	(Ittu et al., 2006)
Thatcher × 5Sumai 3 (BC4F2)	Type II resistance (SFI in GH)	Fine mapping of 3BS	*Xsts3B.80–Xsts3B.142*	-	(Cuthbert et al., 2006)
HC374 × 3*98B69-L47 (BC2F3)	Type II resistance (SFI in GH)	Fine mapping of 3BS	*Xsts3B.80–Xsts3B.66*	-	(Cuthbert et al., 2006)
Nanda2419 × Wangshubhai (RIL)	Type I resistance (SI /spawn in FE)	Wangshubhai (5A, 4B, 2D)	*Xwmc96–Xgwm304*	16.6-20.3	(Lin et al., 2006)
F7 Heterozygous derivatives of Sumai 3	Type II resistance (SFI in GH)	Sumai 3 (3BS fine mapping)	*Xsts3B.189 Xsts3B.206*	-	(Liu et al., 2006)
Wangshuibai × Alondra (RIL)	Type II resistance (FE & GH)	Wangshuibai (3BS), Alondra (1B)	*Xbarc147–Xgwm493*	13.7-23.8	(Zhang et al., 2004)
ARz × Yangmai 158F (RIL)	Type I resistanceNI in FE, SI in FE	2DL, 3BL, 7DL	SSR	5.8-8.7	(Wu et al., 2006)
K2620 × K11463 (RIL)	Type II resistance (SFI in GH)	*Thinopyrum ponticum* (7e12)	*Xpsr121–Xcfa2240*	15 to 30	(Shen and Ohm, 2007)
Wangshubai × Alondra (RIL)	Type II resistance (SFI in FE)	Wangshubhai (3BS), Alondra (1B)	SSR	13.7 to 23.8	(Haeberle et al., 2007)

KY 93-1238-17-2 × VA01W-476	Artificial Inoculation (Field)	2D	STS, SSR	25-28%	(Ansari et al., 2007)
LDN × LDN-D1C 3A	Type II resistance, (SFI in GH)	*Triticum dicoccoides* (3AS)	*Xgwm2*	-	(Chen et al., 2007)
BW278 × AC Foremost (RIL)	Type II resistance (SI in FE)	Sumai 3 (6BS fine mapping)	*Xgwm133–Xgwm644*	-	(Cuthbert et al., 2007)
Arina × Riband (DH)	Type II resistance (SI in FE)	Arina (4DS, 6BL)	*Xpsp3131, Rht-D1*	9.9 to 23.9	(Draeger et al., 2007)
QTL-NIL for *Fhb1* locus from 13 different pop.	Type II resistance (SFI in GH)	Validation of *Fhb1* locus	SSR	-	(Draeger et al., 2007)
Wangshuaibi × Seri 82	Type II resistance	6BS	ESTs	18%	(Browne and Brindle, 2007)
Dream × Lyn×	Type II resistance	6AL, 7BS	AFLP	27%	(Haeberle et al., 2007a)
Wangshubai × Alondra (RIL)	Type II resistance (SFI in FE)	Wangshubhai (3BS), Alondra (1B)	SSR	13.7 to 23.8	(Haeberle et al., 2007b)
Veery × CJ 9306 (RIL)	Type II & III resistance (SFI in GH & FE)	CJ 9306 (3BS, 2DL, 1AS, 7BS, 5AS)	*Xgwm533b–Xgwm493*	5.9 to 30.7	(Jiang et al., 2007a)
CM-82036 × Remus (RIL)	Type III resistance (SFI in GH)	CH-82036 (3BS)	SSR	92.6	(Gosman et al., 2007)
Ernie × MO94-317 (RIL)	Type II resistance (SFI in GH)	Ernie (2B, 3B, 4BL, 5A)	*Xgwm285, Xbarc165*	4.2 - 17.4	(Liu et al., 2007b)
K2620 × K11463 (RIL)	Type II resistance (SFI in GH)	Thinopyrum ponticum (7e12)	SSR	15 to 30	(Shen and Ohm, 2007)

Sumai 3 × Stoa	Type II resistance (FE)	2B, 3B, 7A	SSR	-	(Zhang and Mergoum, 2007)
G16-92 × Hussar (RIL)	Type II resistance (SI in FE)	Hussar (1A), G16-92 (2BL)	*Xgwm501–Xgwm47*	9.7 to 14.1	(Schmolke et al., 2008)
Spark × Rialto (DH)	Type II resistance (SI in FE)	Spark (4DS)	*Rht-D1*	50.9	(Srinivasachary et al., 2008a)
Ning 7840 × Clark	Type II resistance (FE)	3BS fine mapping	SSR, SSCP EST	-	(Yu et al., 2008a)
Wangshuibai × Wheaton (RIL)	Type I, II, III resistance (SFI & SI in GH)	Wangshuibai (3BS, 3BSc, 3DL, 3AS, 5DL)	*Xbarc147-Xgwm292*	6.8 to 34	(Yu et al., 2008b)
Fukuho-Komugt × Oligo Culm	-	-	RAPD	-	(Holzapfel et al., 2008)
Dream × Lynx (RIL)	Type II resistance (SI in FE)	Dream (6AL, 7BS)	*Xgwm82, Xgwm46*		(Häberle et al., 2008)
Sumai 3 × Gamenya	-	2DS	SSR	26%	(Ban and Handa, 2008)
Ben, Maier, Lebsock, Mountrail	Type II resistance	5B, 2A	DArT	18.10	(Ban et al., 2008)
Apachi × Biseay, History × Rubens Romus × Pirat	Type II resistance	6A	AFLP	0.3-35	(Holzapfel et al., 2008)
Sincron × F1054W (RIL)	Type II resistance (SFI in FE)	Sincron (1BL, 1DS)	SSR	-	(Hongxiang et al., 2008)

Cansas × Ritmo	Type II resistance	Cansas (1BS, 3DL, 5BL), Ritmo (3B, 1DS, 7AL)	*Xe35m52.331,XsS25m20.245*	9.9-20	(Klahr et al., 2007)
Langdon × Langdon (Dicc-7A) (RIL)	Type II resistance (SFI in GH)	T. dicoccoides (PI478742)(7AL)	SSR	19	(Diethelm et al., 2008)
Nanada 2419 × Wangshuibai	Type II, IV resistance	2B, 3B, 4B	SSR	7.4-23.4	(Li et al., 2008a)
Sumai 3 × Gamenya (DH)	Type I, II and III (SFI in GH, SI in FE)	Gamenya (2DS)	*Xgwm261–MRP*	14-25	(Handa et al., 2008)
SHA 3 / CBRD × Na×os	Type II resistance	2D	SSR, DArT	-	(Abate et al., 2008b)
Ning894037× Alondra Wangshuibai × Alondra Sumai3 × Alondra	Type II resistance (FE and GH)	3B	SSR	10-43	(Hamzehzarghani et al., 2008)
Chinese Spring×Sumai 3 3B substitution line	Type II resistance (GH)	3BS	STS STM	45	(Brown-Guedira et al., 2008)
Chinese Spring × CS-Sumai 3-7ADSL (RIL)	Type II, III & IV resistance (SFI in GH)	Sumai 3-7ADSL (3BS, 7AC)	SSR/STS	18 to 35	(Bernousi et al., 2009b)
Soissons × Orvantis (DH)	Type I & II resistance (SI in FE)				(Srinivasachary et al., 2009)
1L94-1653 × Patton (RIL)	Type II, III & IV resistance (SI in GH & FE)	IL94-1653 (2B, 4B, 6B)	SSR	2.3-12.3%	(Bonin and Kolb, 2009)

Pelikan//Bussard/Ning 8026	Type II resistance	5BL, 6BS	-	24-30%	(Haeberle et al., 2009)
Frontana × Falat	Type II resistance	3AS	-	-	(Hosseini et al., 2009)
C93-3230-24 × Foster	-	1H, 2H, 4H, 5H, 6H, 7H	RFLP	-	(Lamb et al., 2009)
Bankuti 1201 × Mv Magvas	-	-	RFLP	-	(Laszlo et al., 2009)
Huangfangzhu × Wheaton	Type II resistance	1AS, 1B, 5AS, 7AL	SSR	18-35%	(Hosseini et al., 2009)
Wangshuibai × Falat ($_{F2:2}$)	Type II resistance	2A, 3B	SSR	9.1-16	(Golkari et al., 2009)
Tokai 66 × Jagalene (RIL)	Type II, III & IV resistance (FE)	5B, 3BS, 3D,	SSR	-	(Buerstmayr et al., 2009)
Arina × NK93604 (DH)	FHB severity (SI in FE)	Arina (1BL, 6BS), NK93604 (1AL, 7AL)	SSR	7.8 to 27.9	(Du et al., 2010)
Nanda2419 × Wangshubai (NIL)	Type II resistance (SSEG in FE)	Wangshubai (4B fine mapping)	*Xhbg226-Xgwm149*	Fine mapping *Fhb4*	(Xue et al., 2010)
Wangshubai × Sy95-7 (F2:3)	Type II resistance (SFI in FE)	Wangshubai (7A, 6B, 3B, 2D, 1B)	SSR	4.98 to 22.37	(Zhang et al., 2010)
Thatcher × 5*Sumai 3 (BC4F2)	Type II resistance (SFI in GH)	Sumai 3 (3BS fine mapping)	SSR	-	(Ghaffari et al., 2010)

HC374 × 3*98B69-L47-66 (BC2F3)	Type II resistance (SFI in GH)	Nyu Bai (3BS fine mapping)	SSR	-	(Ghaffari et al., 2010)
Fundulea 201R ×	Type II resistance	1B, 3A, 3D, 5A,	-	32.7-43%	(Coster, 2010)
U 24 × Saiki 165	Type II & IV resistance	3BS	SSR	-	(Kubo et al., 2010)
T. macha × Furore (BC_2 RILs)	Type II resistance (SI in FE)	*T. macha* (2AS, 2BL, 5AL, 5B)	AFLP, SSR	-	(Buerstmayr et al., 2011)
71Ning 7840 × Clark BC 7F7	Type II resistance	3BS	ESTs	45-54%	(Bernardo et al., 2011)
Wheaton × Haiyanzhong (RIL)	Type II resistance (SFI in GH)	Haiyanzhong (7D, 2 QTL on 6B, 5A, 1A)	SSR	5.4 to 22.6	(Li et al., 2011)
Avle × Line 685 (DH)	Type II resistance (SFI, SI in FE)	3BS (*Fhb1*), 5AL, 2BL, 4D (*Rht-D1*)	SSR, DArT	38%	(Lu et al., 2011)
8 different biparental crosses + Tun34 × Lebsock	Type II resistance (SFI in GH)	Lebsock (5BL)	SSR	-	(Ghavami et al., 2011)
445 European soft winter wheat lines for association mapping	Type II resistance (FE)	1B, 1D, 2D, 3A	SSR	36 GV	(Miedaner et al., 2011)
Chinese Spring × CS-Sumai 3-7ADSL (7ACRIL)	Type II, III & IV resistance (SFI in GH)	Sumai 3 (3BS same as *Fhb1*) CS-Sumai 3-7ADSL (7AC named *Fhb7*AC)	SSR (*Xwmc17)*	22 and 24%	(Jayatilake et al., 2011)
PI 277017 WB-Grandin (DH)	Type II, III & IV resistance (SFI in GH, Spawn in FE)	PI277017 (4B, 5AS, 5AL)	*Xbarc40, Xcfd39*	20-32%	(Chu et al., 2011)

Trego' × Heyne (RIL)	Type II resistance	Tregi (3AS, 4DL, and 4AL)	SSR	13-23	(Zhang et al., 2012a)
Baishanyuehumg × Jagger (RIL)	Type II resistance (SFI in GH)	Baishanyuehumg (3BS, 3A, 3BSc)	SSR	3.8 to 15.7	(Zhang et al., 2012b)
Sumai 3 × Y1193-6 (RIL)	Type I, II & IV resistance (SI/Spawn in GH & FE)	Sumai 3 (2DS, 3BS, 6BL)	SSR & DArT	6.5 to 25.8	(Basnet et al., 2012)
T. dicoccum-161 × DS-131621	Type II resistance, (SI)	Floradur (3B) *T. dicoccum* (4B (*Rht-B1*) 6B, 7B)	AFLP, SSR	-	(Buerstmayr et al., 2012)
T. marcha × Furore (BC2F3)	Type II resistance (SI in FE)	T. march inbred (2A, 2BS, 2BL, 5B), Furore (2D)	SSR/AFLP	3.2 to 22.7	(Basnet et al., 2012)
Ning 7840 × Clark (RIL)	Type II resistance (SFI in GH)	Ning 7840 (3BS)	SSR	25 to 56	(Hao et al., 2012)
Sumai 3 × Gamenya (DH)	Type I, II & III resistance (SFI in GH & SI in FE)	Gamenya (2DS)	SSR	14 to 25	(Chen et al., 2012)
Frontana × Remus (DH)	Type II & Type IV resistance (SI in FE)	Frontana (7B)	SSR	-	(Szabo-Hever et al., 2012)
Kukeiharu 14 × Sumai 3 (DH)	Type II resistance (FE)	3BS, 5AS, 6BS, 2DL, 4BS	SSR	1-5	(Suzuki et al., 2012)
BGRC3487/2*DT735 (BCRIL)	Type II & Type III resistance (FE & GH)	2A, 3B, 5A, 5B, 7A, 7B	DArT SSR	53.5-86.2	(Agostinelli et al., 2012)
Maier × Tunisian 7	Type II resistance	3A, 3B, 5A, 1B	SSR	-	(Huhn et al., 2012)
VA00W-38 × 26R46 (RIL)	Type I, III & IV resistance (SI in FE)	VA00W (2DL, 5B, 6A)	SSR, DArT	6.5-21.3%	(Liu et al., 2012)

Baishanyuehumg × Jagger (RIL)	Type II resistance (SFI in GH)	Baishanyuehumg (3BS (*Fhb1*), 3BSc)	SSR	3.8 to 15.7	(Zhang et al., 2012b)
Trego' × Heyne (RIL)	Type II resistance (SFI in FE and GH)	Tregi (3AS, 4DL, 4AL)	SSR	13-23	(Zhang et al., 2012a)
T. dicoccum-161 × Austrian *T. durum* recipient varieties (DS-131621, Floradur and Helidur)	Type I & Type II resistance (FE)	3B, 4B, 6A, 6B, 7B,	SSR AFLP	2-18	(Buerstmayr et al., 2012)
Huangcandou (HCD) × Jagger (RILs)	Type II resistance (SFI in GH)	HCD (3BSc, 3BSd, 3AS) Jagger (2D and 6D)	SSR	10-26.1% 6.7-9.5%	(Cai and Bai, 2014)
Neixiang188 × Yanzhan1 (RILs)	Type I & Type II resistance	Neixiang188 (4D, 5B and 5D) Yanzhan1 (2D and 4B)	*Xgwm292–Vrn-D1, Xpsp3007–DFMR2*	9.3 - 12.8 4.7 to 5.6-	(Lv et al., 2014)
Capo × Arina (RIL)	Type II resistance (SI in FE)	Arina (2AS, 4AL, 6BL) Capo (5AL)	*wPt-2903-wPt-4828*	4.3-8.6	(Buerstmayr & Buerstmayr, 2015)
02-5B-318 × Saragolla (RILs)	Type I & Type II resistance (NI in FE; SI in GH)	02-5B-318 (2AS, 3AL, 5BS) Saragola (2BS and 7AL)	SNPs *(IWB63138)*	7-12% 8-12%	(Giancaspro et al., 2016)
Haiyanzhong × (HYZ) Wheaton (RIL)	Type II resistance (SFI in GH)	HYZ (5AS , 6BS, 7DL)	SSR, SNPs	5.59-15.98%	(Cai et al., 2016)
Yumrchikara × Kitahonami (DH)	Type II resistance (Spawn in FE)	Yumechikara (1BS) Kitahonami (3BS)	*GluB3-barc32*	36.4% and 11.2%	(Nishio et al., 2016)

Sou#1 × Naxos (RILs)	Type I (SI/spawn in FE) Type II (SFI in FE)	Soru#1 (2DLc, 5AL, 4DS, and 5DL for type I) (2DS, 3AS, 4AL, 5AL for typeII)	SNPs, SSR and STS	15-22% for 2DLc	(He et al., 2016)
NC-Neuse × AGS2000 (RILs)	Type I, II, III & IV resistance (Spawn in FE)	NC-Neuse (1A, 1B)	*IWA3805-IWA6152*	8.7-10.8	(Petersen et al., 2016)
Kenyon × 86ISMN (RILs)	Type I (SI/Spawn in FE)	Kenyon (7D, 2D)	SArT SSR SNPs	-	(McCartney et al., 2016)
INW0412 (Huapei 57-2) × 992060G1 (RILs)	Type I resistance (SI in GH & FE)	INW0412 (1AS, 1BL, 2BL)	*TP126266, TPI88538*	8.8-11.7	(Sun et al., 2016)
DBC-480 (*Fhb1* introgression from Sumai 3) × Karur (RILs)	FHB severity (SI inoculation in FE) FHB spread (SFI in GHE	DBC-480 (2BL, 3BS, 4BS)	SSR, SNPs	4.3-64.2%	(Prat et al., 2017)
DBC-480 × Durobonus (RILs)	Type II resistance (SI in FE)	DBC-480 (3BS, 4BS, 5AL)	SSR, SNPs	6.2-38.4%	(Prat et al., 2017)
DBC-480 × SZD1029K (RILs)	Type II resistance (SI in FE)	DBC-480 (3BS, 4AL,4BS, 6AS)	SSR, SNPs	5-24.9	(Prat et al., 2017)
AC Brio × TC 67 (RILs)	Type II resistance (SFI in GHE), Type I & III & IV resistance (SI in FE)	TC 67 (5AL1, 5AL2, 6A)	*cfd39-cfa2185, gwm132.1-wmc621*	5.2-20.6%	(Malihipour et al., 2017)
Joppa × 10Ae546 (RILs)	Type II resistance (SFI in GH & FE)	Joppa (2A) 10Ae546 (5A, 7B)	90K SNP array	9-15% 7-19%	(Zhao et al., 2018)
ND2710 × Bobwhite (RILs)	Type II resistance (SFI in GH & FE)	ND2710 (3BS, 6B, 2A and 6A	90K SNP array	5-20% 5-12%	(Zhao et al., 2018)

DT707 × DT969 (DH lines)	Type II resistance (FE)	DT696 (5A)	90K SNP array(*Ex_c6 161_335)*	3.8-25.7%	(Sari et al., 2018)
Strongfield × Blackbird (DH lines)	Type II resistance (FE)	Blackbird (1A)	90K SNP array	11.3-26.8%	(Sari et al., 2018)
C615 × Yangmai 13 (RILs)	Type II resistance (SFI/ SI in FE)	C615 (2AL, 2DS, 2DL)	SNPs	-	(Yi et al., 2018)
Everest × WB-Cedar (DH)	Type II, III & IV resistance (SI in FE)	Everest (5AS, 1BS, 3DS, 4BL) WB-Cedar (1AS)	SNPs	5.51-13.86%	(Lemes da Silva et al., 2019)
AQ24788-83 × Luke 1652RILs (RILs)	Type I & Type II resistance (SFI in GHE, SI in FE)	AQ24788-83 (7DL)	*gwm428*	22-32%	(Ren et al., 2019)
Tunisian108 × Ben (BC1F7)	Type II, III & IV resistance (SFI in GH, Spawn in FE)	Tunisian108 (3BL, 2B)	SSR, DArT	6-11%)	(Pirseyedi et al., 2019
Glenn × MN0026`-4 (RILs)	Type I, II & IV resistance (SFI in GE; Spawn in FE)	MN0026-4 (5BL), Glenn (1AS,1BL, 6BS, 7AS)	*wPt8168-Xwmc728*	10.2-23.8	(ElDoliefy et al., 2020)

*PSS: Percentage of symptomatic spikelets; Type I: FHB incidence; Type II: FHB severity/spread; Type III: Deoxynivalenol (DON); Type IV: Fusarium damaged kernels (FDK)

*Inoculation methods: SFI: Single floret infection; SI : SI/Spawn inoculation; GE: Greenhouse experiment; FE: Field experiment; NI: Natural infection

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Chapter 2 - Mapping of quantitative trait loci for resistance types II, III and IV to Fusarium head blight in Overland × Everest population

Introduction

Wheat is subjected to various biotic and abiotic stresses, which can cause losses of more than half of the potential crop yield (Wang & Qin, 2017). Among the major biotic stresses, Fusarium head blight (FHB), incited by *Fusarium graminearum* (Schwabe), is the most widespread and devastating disease of wheat in humid and semi-humid areas of the world (Bai & Shaner, 2004; Goswami & Kistler, 2004). FHB can affect all classes of wheat and barley (Nganje et al., 2004). Yield losses are mainly from reduced grain size and test weight. Accumulation of mycotoxins, especially deoxynivalenol (DON), in grain lowers grain quality and market value on grain sale and is also a major health concern for humans and animals. DON showed negative effects on eukaryotic cells including inhibiting protein synthesis, DNA and RNA synthesis and cell division, impeding the mitochondrial functions, and altering the structure of cell membrane (Brar et al., 2019; Cetin & Bullerman, 2005; Rocha et al, 2005). Severe epidemics of the FHB disease may occur when high rainfall coincides with the anthesis stage of susceptible wheat cultivars in the presence of sufficient pathogen inocula (Cai & Bai, 2014). FHB disease epidemics have occurred in several hard winter wheat producing states in the Great Plains of the U.S.A. in recent years and DON has been a major concern in these regions (Wegulo, 2012).

There are many control strategies to minimize FHB damage such as cultural practices to destroy the primary inoculum source and foliar spray at anthesis to reduce the initial fungal

infection (Windels, 2000). The uneven flowering time in a field may need multiple applications of fungicides, which can be costly and also cause severe grain contamination that can be harmful to human health (Wegulo et al., 2015). The most effective and economical method for control of the disease is to grow FHB resistant cultivars (Bai & Shaner, 2004; Rudd et al., 2001). However, multiple minor QTLs governing FHB resistance make the breeding for highly FHB resistant cultivars a challenging task. Further understanding of the complex genetic mechanisms of FHB resistance will facilitate the effective deployment of the available FHB resistant sources.

Five types of FHB resistances have been reported: type I for resistance to initial infection; type II for resistance to fungal spread within a spike; type III for resistance to toxin accumulation in infected kernels; type IV for resistance to *Fusarium* damaged kernels (FDK) and type V for tolerance to FHB (Mesterházy et al., 1999). Type I and Type II resistance were first reported in wheat and have received wide attention (Dweba et al., 2017; Schroeder and Christensen 1963). Type II resistance has been extensively studied in wheat because it is more stable and less affected by environments than other types of resistance (Bai & Shaner, 2004).

Recent progress in the development of high-throughput sequencing technology and genome mapping tools has accelerated the identification of resistant QTLs and genes (Liu et al., 2014). Quick developments of next-generation sequencing (NGS) technologies further reduces the cost of NGS-based marker discovery including sequencing of reduce representation genome libraries, restriction site-associated DNA sequencing (RAD-seq), genotyping-by-sequencing (GBS) and multiplex shotgun sequencing (Chapman et al., 2015; Poland et al., 2012; Rife et al., 2015) which make it possible to construct high-resolution maps at acceptable costs for mapping of the genomic regions associated with FHB resistance in wheat. To date, several hundreds of QTLs associated with FHB resistance have been reported on all wheat chromosomes

(Buerstmayr et al., 2009; Liu et al., 2009). Exotic sources of resistance have been used in U.S. wheat breeding programs to improve FHB resistance in locally adapted wheat cultivars. However, significant yield penalties from the incorporation of exotic genes of poorly adapted materials prevent the direct use of these sources in a breeding program (Lemes da Silva et al., 2019). A Chinese landrace Wangshuibai (WSB) was found to possess an excellent level of FHB resistance because of major QTLs conferring FHB resistance on 5A, 3B, 4B, and 6B chromosomes of wheat (Jia et al., 2018; Lin et al., 2004; Xue et al., 2010, 2011). WSB has been utilized as a resistant parent in many breeding programs, but the linkage drag with poor agronomic traits has been the main concern to use it as a resistance source. Fortunately, some locally adapted cultivars have been identified with some levels of FHB resistance (Eckard et al., 2015). Breeding using native FHB resistant sources has an advantage of avoiding introgression of large chromosome fragments from an unadapted material which may result in yield reduction (Lemes da Silva et al., 2019). Therefore, integrating multiple native minor QTLs from adapted backgrounds can improve FHB resistance with a good balance of disease resistance, high yield potential and end-user quality.

Biparental mapping populations developed using parents with contrast in the target trait have been popular for mapping the chromosome regions associated with the traits of interest. However, most of the previous mapping studies used the low-density linkage maps to map QTLs. Wheat is polyploid and has a large genome size of ~17 Gb, which makes the construction of high density maps a challenging task (Lin et al., 2015). The availability of next-generation sequencing (NGS) technologies makes it possible to develop such maps using genome-wide single nucleotide polymorphism (SNPs) markers in wheat (Akhunov et al., 2009). Genotyping-by-sequencing (GBS) is a new NGS-based genotyping platform that reduces the genome

complexity using restriction digestion (Poland et al., 2012) and lowers the sequencing cost by multiplexing samples using barcodes (Spindel et al., 2013). With the availability of a wheat reference genome of Chinese Spring, high-density SNP markers can be easily located to chromosomes and used to map QTLs.

Using a population of recombinant inbred lines (RILs) developed from Overland × Everest, the objective of this study were to 1) map novel QTLs associated with FHB resistance types II, III, and IV in Overland and Everest; 2) identify markers tightly linked to the QTLs; and, 3) convert the GBS-SNP markers into Kompetitive Allele-Specific PCR (KASP) assays for marker-assisted selection.

Materials and Methods

Plant materials

A population of 178 $F_{2:7}$ RILs derived from Everest × Overland by single seed descent. Everest (PI 659807) is a hard red winter wheat cultivar developed by Kansas State University in 2009 (Fritz et al., 2011) with the pedigree of HBK1064-3/JaggerW//X960103 (NPGS, 2017). Everest showed moderate resistance to FHB with a designation of 4 on a 1-9 scale in fields. It is widely adopted in eastern and central Kansas where FHB frequently occurs (Bockus et al., 2016). Overland (PI 647959) is a hard red winter wheat cultivar developed from Nebraska Agriculture Experimental Station in 2007 with moderate resistance to FHB in the yield nurseries (Baenziger et al., 2008). Overland was derived from a single cross Millennium sib x ND8974. ND8974 was derived from a three-way cross Millennium sib//Seward/Archer.

Experimental design

Everest and Overland, and their RIL population were evaluated for type II FHB resistance in both spring and fall greenhouse experiments in 2017 and 2018, at Kansas State

University (KSU), Manhattan, KS. Each experiment was arranged in a randomized complete block design with two replicates. Both the parents and RILs were planted in 128-cell germination trays (Hummert International Topeka, KS) filled with Berger BM1 growing mix (Hummert International Topeka, KS) and placed in the vernalization chamber at 6 ^{o}C for seven weeks of vernalization. Plant tissues were collected at the three-leaf stage from each plant for DNA extraction. Six seedlings per line were transplanted into a '4 x 4' Dura pot filled with Berger BMI growing mix (Hummert International, Topeka, KS). All pots were fertilized with Osmocote® classic 19-06-12 slow-released fertilizer (The Scotts Company LLC, Marysville, OH) immediately after transplanting. The plants were watered with the Miracle-Gro® water-soluble all-purpose plant food (The Scotts Company LLC, Marysville, OH) once a week for the first four weeks. Marathon® 1% granular insecticide was applied one week after transplanting to control insects. The greenhouse was set at 17 ± 5 °C with 12 h photoperiod from 07:00 to 19:00. The daytime temperature was raised to 22 ± 5°C before heading.

FHB evaluation in greenhouses

Conidial inocula of *Fusarium graminearum* were prepared using a Kansas field isolate GZ3639 (Desjardins et al., 1996). For a decade, this isolate showed consistent pathogenicity on the same set of wheat cultivars tested in different years (Jin et al., 2013). At the anthesis stage, a single floret of a central spikelet of a spike was injected with a 10 µL spore suspension (~1000 spores/spike) using a Hamilton PB600-1 syringe (Hamilton Company, Reno, NV). Five spikes per pot were inoculated. After inoculation, plants were moved into a moist chamber with 100% relative humidity for 48 h at 21±5 °C for initial infection. About 48 h after inoculation when the dark brown disease symptoms appeared on the inoculated spikelets, the plants were moved to greenhouse benches for disease development. About 14 d post-inoculation, when the inoculated

spikes of the susceptible parent were completely blighted, numbers of infected and total spikelets of each inoculated spike were recorded to calculate the percentage of symptomatic spikelets (PSS).

$$PSS\ (\%) = \frac{Number\ of\ symptomatic\ spikelets\ per\ spike}{Total\ number\ of\ spikelets\ per\ spike} \times 100 \qquad (2.1)$$

Meantime, the heading date of primary tillers in each pot was recorded when more than 50% of spikes emerged from flag leaves. Plant height was measured before harvesting by measuring the height from soil surface to the tip of the spike excluding awns in each pot.

FHB evaluation in fields

Both the parents and their RILs population were evaluated for FHB resistance in the Rocky Ford FHB Nursery of the Department of Plant Pathology, Kansas State University, Manhattan, KS during the 2017-18 and 2018-19 growing seasons. The experiments were conducted in a randomized complete block design (RCBD) with two replications. About 40 seeds per line were planted in a 1-m long, single-row plot. The FHB nursery was inoculated corn grain spawn by scattering *F. graminearum* infected corn (*Zea mays* L.) kernels twice on the soil surface with one at the booting stage and another two weeks afterward to facilitate initial infection. The field was misted using mist sprinklers running 3 min per hour between 21:00 and 6:00 h daily from anthesis to early dough stage. The heading date was recorded per plot basis when 50% of spikes emerged from flag leaves. Visual evaluation of PSS was done on the basis of the overall performance of a row about 20 ± 5 days after heading based on the symptoms developed on control cultivars. Data were rechecked three days after the first note. At maturity, plant height was measured from the soil surface to the tips of spikes excluding awns.

After manual harvesting, spikes from each plot were threshed using a small Vogel thresher (Almaco, Nevada, IA) for evaluation of *Fusarium*-damaged kernels (FDK) and

deoxynivalenol (DON). Visual FDK was scored by estimating the percentage of FDK in total kernels harvested from each row. A 5 g kernel sample was randomly taken from the total harvested kernels for the DON test using gas chromatography-mass spectrometry (GC-MS) at the University of Minnesota, St. Paul, MN (Mirocha et al., 1998). The DON concentration was reported in parts per million (ppm) for each sample.

DNA extraction and marker genotyping

Leaf tissues from seedlings of the RIL population and its parents were collected into 96-deep-well plates at the three-leaf stage, then dried at -51 ºC for 72 h using a freeze dryer (ThermoSavant, Holbrook, NY). The dried tissues were then ground to a fine powder at 25 cycles/sec in a Mixer Mill (MM 400, Retsch, Germany). A modified cetyltrimethyle ammonium bromide (CTAB) protocol (Saghai-Maroof et al., 1984) was used to extract the genomic DNA of both the parents and RILs (https://hwwgenotyping.ksu.edu/protocols/). DNA was checked for quality using a 1% agarose gel, quantified using the Quant-iT™ PicoGreen® dsDNA Assay (Life Technologies Inc., NY), and normalized to a final concentration of 25 ng/µL for construction of Genotyping-by-Sequencing (GBS) library following the protocol of Poland et al. (2012). Briefly, DNA was digested using HF-*PstI* and *MspI* restriction enzymes (New England BioLabs Inc., Ipswich, MA) to reduce wheat genome complexity. The barcoded adapters and Y common adapters were ligated to digested DNA using T4 DNA ligase (New England BioLabs Inc., Ipswich, MA). The ligated samples were cleaned up using the QIAquick PCR purification kit (Qiagen Inc., Valencia, CA). Polymerase chain reaction (PCR) was performed using primers complementary to both adapters. The PCR products were cleaned up again using the same purification kit. Amplicons in the range of 250-300 bp were selected using an E-gel system (Life Technologies Inc., NY) and quantified using the Qubit 2.0 fluorometer and Qubit dsDNA HS

Assay kit (Life Technologies Inc., NY) for sequencing on an Ion Proton sequencer (Life Technologies Inc. NY).

The GBS sequence data with 80% of the bases having high-quality scores of >15 were processed to call SNP using a reference-based pipeline of TASSEL (Glaubitz et al., 2014; Liu et al., 2013; Poland et al., 2012). The Ion proton sequencer produced the raw sequence reads of variable lengths. Before TASSEL 5.0 analysis, 80 poly-A bases were added to the 3' end of all sequence reads so that TASSEL could use reads shorter than 64 bp in the analysis. To reduce false-positive SNP calls, only SNPs with < 10% heterozygotes across the RIL population and < 30% missing data were used for mapping.

Genetic map construction and QTL analysis

A genetic linkage map was constructed with SNP data from GBS using the 'regression' mapping algorithm in JoinMap version 4.1 (Van Ooijen, 2006). A logarithm of odds (LOD) value of 7.0 was used as grouping criteria to place markers into different linkage groups and the Kosambi mapping function was used to transform the recombination frequencies into the centiMorgan genetic distance (cM), (Kosambi, 1994). The linkage map with 21 linkage groups was visualized using the "DrawChr" function of Window QTL Cartographer v2.5 (Wang et al., 2012). The QTLs for FHB and related traits were analyzed by composite interval mapping (CIM) using Windows QTL Cartographer v2.5 (Wang et al., 2012). The genome-wide LOD thresholds used to declare the significance of QTLs for each trait were estimated using a permutation test with 1000 permutations (Doerge et al., 1996; Nettleton and Doerge, 2000).

Conversion and validation of KASP markers

GBS-SNPs tightly linked to the QTLs were further converted into KASP assays. A KASP primer mix consists of three primers with two allele-specific forward primers and one common

reverse primer. The two forward primers were attached to a unique tail sequence that corresponds to a universal fluorescence resonant energy transfer-cassette: one labeled with FAM™ dye and the other with HEX™ dye. The KASP primers were designed using PolyMarker (Ricardo et al., 2015). The designed primers were evaluated for polymorphism between two parents before genotyping the mapping population. For KASP analysis, a 6 µL KASP reaction mix including 2.92 µL 2X KASP master mix, 0.08 µL KASP primer mix and 3 µL genomic DNA at ~25 ng/µL. Polymerase chain reactions (PCRs) were performed following the manufacturer's protocol (LGC Genomics, https://www.biosearchtech.com/). Briefly, a PCR was initially incubated at 94 ºC for 15 min., followed by 10 cycles of 94 ºC for 20 sec., and 65 ºC for 60 sec., with a decrease of 0.8 ºC in each subsequent cycle, then went through an additional 36 cycles of 94 ºC for 20 sec. and 57 ºC for 60 sec. The PCR products were scored using a FLUOstar® Omega microplate reader (BMG Labtech Inc. Cary, NC) and SNPs were analyzed using KlusterCaller™ software (LGC, Middlesex, UK). KASP marker data were used to substitute the corresponding GBS SNP data to reconstruct the linkage map and reanalyze QTL as described before.

Statistical analysis of phenotypic traits

All phenotypic trait data were analyzed using SAS statistical software version 9.4 (SAS Institute Inc. 2011). PROC UNIVARIATE in SAS was used to check the data normality and distribution. The statistical analyses of greenhouse and field data were performed separately using PROC GLM procedure with the model $Y_{ijk} = u + g_i + t_j + gt_{ij} + r(t)_{ik} + e_{ijk}$ where Y_{ijk} is the trait, g_i states the fixed effect of the i^{th} genotype, t_j states the random effect of the j^{th} experiment, gt_{ij} states the random interaction of the i^{th} genotype with the j^{th} experiment, $r(t)_{ik}$ defines the random effect of the k^{th} replicate nested within the j^{th} experiment, and e_{ijk} is the

random error term. Similarly, variance components were estimated for field experiments following model: $Y_{ijk} = u + g_i + y_j + gy_{ij} + r(y)_{ik} + e_{ijk,}$ where Y_{ijk} is the trait, g_i states the fixed effect of the i^{th} genotype, y_j states the random effect of the j^{th} year, gy_{ij} states the random interaction of the i^{th} genotype with the j^{th} year, $r(y)_{ik}$ defines the random effect of the k^{th} replicate nested within the j^{th} year, and e_{ijk} is the random error term. Plant height (PH) was included as a covariate with a fixed effect in the analysis in order to avoid the confounding effect on FHB resistance caused by plant height. PROC VARCOMP was used to estimate the variance components to estimate broad-sense heritability for all disease-related traits using the following formula:

$$H^2 = \sigma_G^2 / (\sigma_G^2 + \frac{\sigma_{GE}^2}{e} + \frac{\sigma_E^2}{re}) \qquad (2.2)$$

where, σ_G^2 defines the genetic variance, σ_{GE}^2 defines genotype x experiment interaction variance and σ_E^2 is the error variance, r is the number of replicates and e is the number of experiments (Nyquist & Baker, 1991). Adjusted mean comparisons between parents and RIL lines were performed using a Tukey-Kramer test (Tukey, 1949). Pearson correlations for all traits were estimated using a corrplot and ggplot2 package in RStudio v 4.0.2 (RStudio, Boston, MA).

Results

Variations and correlations among phenotypic traits in the greenhouse experiments

Overland showed moderate FHB resistance in all the four-greenhouse experiments with an average PSS of 31.09%, ranging from 12.23 to 36.51%, whereas Everest had a mean PSS of 68.35 (46.76 to 80.83%), showing moderate susceptibility (Fig. 2.1). The RIL population showed significant variation in PSS after point inoculation and PSS showed continuous, but skewed distribution towards Everest in each individual experiment except fall 2018 which was skewed

right towards Overland (Fig. 2.1). Obvious transgressive segregation was observed in the fall 2017 experiment. The mean PSS of RILs across four-experiments ranged from 4.62 (highly resistant) to 100% (highly susceptible). Overall, the disease pressure was lowest in the fall 2018 experiment and the highest in the spring 2018 experiment. The mean PSS were 53.33% (spring 2017), 60.89% (fall 2017), 65.15% (spring 2018) and 34.59% (fall 2018) with the mean PSS of 53.66% from all the four experiments.

Significant differences were observed between the two parents for heading date (HD) but not for plant height (PH) in individual greenhouse experiments (Fig. 2.2a and 2.2b). To test the presence of *Rht-B1* and *Rht-D1* genes for reduced height, diagnostic KASP assays for *Rht-B1b* and *Rht-D1b* were analyzed between parents and in the population. *Rht-B1b* marker was polymorphic between parents and segregated among RILs, but not *Rht-D1*. Pairwise Pearson correlation coefficients of PSS were significantly positive among four greenhouse experiments ($P < 0.05$), ranging from 0.28 to 0.52 (Fig. 2.3). A significantly negative correlation ($P < 0.05$) was observed between PSS and HD, ranging from -0.13 to -0.50 in each individual experiment. In general, a higher disease score was associated with early headed plants. The plant height showed a significantly negative correlation ($P < 0.05$) with PSS only from the spring 2017 greenhouse experiment, but not significant in the other three greenhouse experiments. Analysis of variance (ANOVA) showed significant variations in genotypes and genotype-by-experiment in the joint analysis of greenhouse experiments (Table 2.1). The broad-sense heritability estimate for PSS was very high (0.79) (Table 2.1). Plant height was included as a covariate with a fixed effect in the analysis and it appeared to be not significant.

Variations and correlations among the phenotypic traits in the field experiments

The ANOVA on data across two experiments showed significant genotypic effects (G), environment (E), and genotype-by-environment (G × E) effects for the three disease traits, PSS, FDK and DON (Table 2.2). The broad-sense heritabilities of the three FHB resistance traits estimated from the two field experiments were relatively higher for FDK and DON than PSS (Table 2.2).

In the 2017 field experiment, the mean PSS values were not significantly different between Everest and Overland, while in the 2018 field experiment, Everest showed significantly lower mean PSS (53.8%) than Overland (63.8%) (Fig. 2.4). The DON was not significantly different between the two parents but Overland showed lower FDK than Everest (Fig. 2.5; Fig. 2.6). The RIL population showed a normal distribution for PSS, FDK and DON in both field experiments. However in the 2018 field experiment, the DON distribution skewed right (Fig. 2.6). Logarithm transformed DON data were used for the analysis of variance. Overall, the range of DON values from the 2018 field experiment was narrower than that from the 2017 experiment (Fig. 2.6). Transgressive segregations for both resistance and susceptibility were observed for all the three FHB related traits. Although the two parents did not show a significant difference in plant height in both field experiments, their RILs showed significant height variation with a normal distribution in both experiments because Everest and Overland carry contrasting alleles at *Rht-B1* locus (Fig. 2.7a). The heading date differed significantly between the two parents in the 2018 experiment but not significant in the 2017 experiment. On average, Everest headed six days earlier than Overland (Fig. 2.7b). Positive pairwise-Pearson correlations were significant among the three disease traits in both years ($r = 0.21$ to 0.70, $P < 0.05$), except that between 2017 DON and 2018 PSS data (Fig. 2.8). Significantly negative correlations were observed between the

plant height and three FHB-related traits evaluated in both years (r = -0.11 to -0.51, $P < 0.05$). Heading dates were significantly negatively correlated with PSS and FDK, but not with DON (Fig. 2.8). Also, significantly positive correlations were observed between FDK and DON content in both years (r = 0.31 and 0.61, $P < 0.05$) (Fig. 2.8). Strong positive correlations were observed between plant height and heading date in both years (0.30 and 0.42, $P < 0.05$).

Construction of linkage map

The GBS-derived SNPs from 158 RILs were analyzed after removing 20 RILs that had excessive missing data. A total of 53,717 GBS-SNPs were called with 80% missing data (Fig. 2.9a). After removing the SNPs with >30% missing data, 2,593 polymorphic SNPs between parents were used for mapping (Fig. 2.9b). After removal of these markers mapped to the same locations, the remaining 2,018 (76.5%) markers were mapped to 32 linkage groups with 3 to 160 markers per linkage group. The total map length was 1,819.05 cM with an average genetic distance of 0.90 cM between the two adjacent markers. The highest marker density was observed on 7D with an average density of 0.32 cM per marker, while the lowest was 2D with an average marker interval of 4.10 cM. The B genome carried most of the markers (44.8%), followed by A genome (35.8%) and the D genome (19.3%) (Fig. 2.10).

QTL mapping for FHB resistance

Composite interval mapping identified six significant QTLs on chromosomes 7AS, 4BS, 5AL, 4AS, 1B and 6BL for different types of FHB resistance in Overland × Everest RIL population. The QTLs on chromosome arms 5AL, 4BS and 7AS were detected on the same genomic regions in at least two experiments, suggesting they are stable QTLs.

Among the seven significant QTLs, *Qfhb.hwwgr.4BS* showed the largest effect on type II FHB resistance and was significant in three greenhouse experiments, one field experiment and a

mean from the four greenhouses and two field experiments (Table 2.3). In the greenhouse experiments, the QTL was flanked by SNPs *4B_SNP26787* and *4B_SNP26830,* and explained 9.64 to 16.22% of the phenotypic variation for PSS in fall 2017, spring 2018 and fall 2018 experiments and mean from the four greenhouse experiments. Marker locus *4B_SNP26830* was positioned at 31.17 Mb on the short arm of chromosome 4B. The resistance allele at the 4BS QTL was from Overland.

In the two field experiments, the resistance allele for PSS, FDK and DON was contributed by Everest at the same genomic region (Figure 2.11, Table 2.3). The QTL *Qfhb.hwwgr.4BS* explained 17.23%, 20.46% and 8.65% of the phenotypic variation for mean PSS, FDK and DON, respectively, and was located between 16.01 to 31.21 cM interval where plant reduced height gene (*Rht-B1*) was mapped (Fig. 2.11). The *Rht-B1b* allele for shorter plant height was from Overland and explained 18.19 to 44.38% of phenotypic variation in the population. The plant height showed a significant negative correlation with PSS (-0.14 to -0.51, $P < 0.05$), FDK (-0.35 to -0.51, $P < 0.05$), and DON (-0.11 to -0.24, $P < 0.05$) in individual field experiments (Fig. 2.8), indicating that the taller plants had less FHB symptoms. However, no significant correlation between PSS and plant height was observed in the greenhouse using artificial inoculation except spring 2017 experiment which showed a significantly negative correlation between PSS and PH (-0.19 to -0.36, $P < 0.05$).

The QTL on chromosome 5AL, *Qfhb.hwwgr.5AL* also showed a significant effect on type II resistance in spring and fall of 2017 and spring 2018 greenhouse experiments and the mean of all four greenhouse experiments. This QTL was mapped to 119.37 to 130.78 cM interval between the SNP markers *5A_SNP32131* and *5A_SNP32237* and explained 9.49 to 11.10% of the phenotypic variation (Table 2.3, Fig. 2.12). The peak marker was positioned at 595.96 Mb on

the long arm of chromosome 5A. The resistance allele was from Overland. This QTL also had a significant impact on low FDK in both field experiments and the mean from the two field experiments, which explained 11.86% and 7.54% phenotypic variation in the 2017 and 2018 field experiments, respectively. In spring 2018, the peak of *Qfhb.hwwgr.5AL* was positioned away from 119 cM and mapped to 68 cM on the long arm of the chromosome and overlapped with plant height QTL which explained up to 10.17% of the phenotypic variation for plant height (Table 2.3, Fig. 2.12). The resistant allele for PSS and plant height at the QTLs was also from Overland.

The QTL on 7AS, *Qfhb.hwwgr.7AS,* showed the largest effect on type II FHB resistance in spring 2017 and mean from the four greenhouse experiments. This QTL was flanked by *7A_SNP45705* and *7A_SNP45970* and explained 12.50% ~19.40% of the phenotypic variation (Table 2.3, Fig. 2.13). The QTL was mapped between 44.26 to 80.38 cM interval with the peak marker *7A_SNP836* at 72.20 Mb.

One QTL on chromosome 4A, *Qfhb.hwwgr.4A,* showed a relatively large effect on type II FHB resistance only in spring 2017 greenhouse experiment. This QTL was flanked by markers *4A_SNP24282* and *4A_SNP24370* and explained 12.11% of the phenotypic variation with resistance allele from Overland. The closest marker to the peak was *4A_SNP25098* positioned at 383.49 Mb (Table 2.3, Fig. 2.14).

Another QTL on the short arm of chromosome 6B, *Qfhb.hwwgr.6BL,* was mapped between *SNPs 6B_SNP43575* and *6B_SNP43639* in one greenhouse and one field experiment. This QTL from Everest explained 13.47% and 8.73% of the phenotypic variation and was delimited between 13.39 and 18.52 cM interval in fall 2017 greenhouse and 2017-18 field

experiments, respectively (Table 2.3, Fig. 2.14). The peak markers for this QTL were *6B_SNP43575* and *6B_SNP43611* located at 716.39 and 721.71 Mb, respectively.

The QTL on 1BL was mapped between *1B_SNP1584* and *1B_SNP3688* and explained 8.25% and 12.56% of the phenotypic variation for fall 2017 greenhouse and 2018-19 field experiments (Table 2.3, Fig. 2.14). The QTL detected in the field 2018 experiment was positioned between 16.60 cM and 20.06 cM, while the QTL peak for the fall 2017 experiment was near *1B_SNP4586* at 23.66 cM. The resistance allele is contributed by Everest for PSS from both experiments.

Conversion and validation of KASP markers

To verify the accuracy of GBS derived SNPs data and fill up the missing GBS data for these markers mapped in the QTL region, 22 KASP assays were designed for six GBS-SNPs in the *Qfhb.hwwgr.7AS* region, six in the *Qfhb.hwwgr.5AL* region, five in the *Qfhb.hwwgr.4BS* region and five in the *Qfhb.hwwgr.4A* region using GBS sequence data. Among them, 10 KASP assays showed polymorphism between parents and segregated in the RIL population (Fig. 2.15). Out of 12 KASP markers, eight markers showed identical allele calls with the GBS-SNP data across the RIL population and were remapped to the 5AL, 4BS, 4A and 7AS QTL regions (Table 2.3). Seven of the KASP markers (RILGBS363, RILGBS836, RILGBS153, RILGBS798, RILGBS1229, RILGBS150, RILGBS752, RILGBS1120) were close to these QTLs and can be used for marker-assisted breeding.

Effects of QTL combinations on FHB type II resistance

To investigate the effect of three resistance QTLs on type II FHB resistance, the three repeatable and significant QTLs on chromosomes 4BS, 5AL and 7AS were analyzed for interaction among the QTLs. All alleles from Overland were represented by capital letters

whereas all alleles from Everest were represented by lowercase letters. Eight possible allelic combinations were designated as AABBCC, AABBcc, AAbbCC, aaBBCC, AAbbcc, aaBBcc, aabbCC, aabbcc (Fig. 2.16). The average PSS of eight genotypic groups of the RIL population ranged from 41.92% to 63.61%. The KASP markers that were closely linked to the QTL peaks were used to estimate the allelic effects. They were *KASP_5A_836* for the *Qfhb.hwwgr.5AL* *KASP_4B_1209* for the *Qfhb.hwwgr.4BS* and *KASP_7A_292* for the *Qfhb.hwwgr.7AS*. The mean PSS value of the genotypic group that carried all susceptible alleles at the three QTLs was 63.60% compared to 41.92% for the group with all the resistance alleles at the three QTLs, suggesting all the three QTLs reduced the FHB severity. The mean PSS values for genotypic groups that carry a single QTL were 58.76% for the *Qfhb.hwwgr.5AL*, 50.76% for *Qfhb.hwwgr.4BS* and 60.38% for the *Qfhb.hwwgr.7AS* (Fig. 2.11). *Qfhb.hwwgr.4BS* showed the largest effect on resistance types II. The mean PSS of the genotypic group carrying only *Qfhb.hwwgr.4BS* was always lower than the genotypic group carrying *Qfhb.hwwgr.5AL* only, but the difference was not significant.

Discussion

Repeatability of FHB and related traits and correlations among these traits

Fusarium head blight is a quantitative trait controlled by multiple genes usually with minor effects (Bai et al., 2018). Precise mapping of these minor QTLs is a great challenge due to large environmental effects on the expressions of these minor QTLs. A large mapping population, an increased number of replications at multiple locations can minimize the environmental effects, increase the repeatability of experiments and improve the accuracy of QTL data (Bai et al. 1999). In the current study, a relatively large mapping population of 177 RILs was repeatedly evaluated for FHB resistance and related traits in both greenhouse and field

experiments compared to other studies that were evaluated in either greenhouse or field experiment alone (Anderson et al., 2001; Buerstmayr et al., 2003; Cai & Bai, 2014; Clinesmith et al., 2019; Lemes da Silva et al., 2019). These repeatable QTLs identified from different experiments should be stable QTLs and useful for breeding to improve wheat FHB resistance.

Accurate assessment of FHB depends upon effective screening protocols. Single floret inoculation is a standard method used for the evaluation of type II resistance. Type II resistance is measured by either counting symptomatic spikelets per spike (Bai & Shaner, 2004) or determined by rating a spike on a visual scale of 1 to 10 (Stack & McMullen, 1995). In this study, single floret inoculation was performed in the greenhouse and grain-spawn inoculation was used in the field misted nursery to ensure sufficient initial infection. Significant correlations ($P < 0.05$) among greenhouse and field experiments indicated the repeatability of these phenotypic data. A weak correlation (r = 0.10 – 0.34, $P < 0.05$) was observed between field and greenhouse PSS, consistent with several previous studies (Chen et al., 2006; Liu et al., 2013; Zwart et al., 2008). The poor correlation might be due to different inoculation methods and the conditions for FHB development used between greenhouse and field experiments. Significant correlations were observed among PSS, FDK and DON (r = 0.31-0.70, $P < 0.05$) in the field experiments, agreeing with previous findings (Bai et al., 2001; Cai et al., 2019; Malihipour et al., 2017; Mesterházy et al., 1999). The significant positive correlations among the three traits suggested the dependency of type III and type IV resistance on type II resistance (Bai et al., 2001; Lu et al., 2013), thus the PSS can be used indirectly to predict the DON level in the scabby kernels. A strong, negative correlation between PSS and PH was significant (r = - 0.14 – - 0.51, $P < 0.05$) in the field experiments but not in the greenhouse experiments except in the spring 2017 greenhouse experiment. Association of FHB resistance with tall plants was also

reported in numerous studies (Draeger et al., 2007; Lemes da Silva et al., 2019; Hashimi, 2019; Lu et al., 2013; Mesterházy, 1995; Steiner et al., 2004). This could be due to that spikes in the taller plants are further from the inoculum source on the soil surface than these in shorter plants in fields (Mesterházy, 1995; Steiner et al., 2004), which reduces initial infection rate and creates unfavorable microclimate conditions for FHB initiation in taller plants (Buerstmayr & Buerstmayr, 2016; Hatfield & Dold, 2018; Hilton et al., 1999; Jones et al., 2018).

In the greenhouse experiments, however, both tall and short plants were equally inoculated by point inoculation and they had the same level of initial infection (Buerstmayr et al., 2011). In the current study, an FHB resistance QTL was co-localized with a gene (*Rht-1B*) conditioning plant height, and reduced height allele was associated with increased FHB susceptibility in the fields, which is consistent with several other studies that co-localized FHB resistance QTLs with *Rht* genes (Draeger et al., 2007; Häberle et al., 2009; Klahr et al., 2007; Liu et al., 2013; Steiner et al., 2004b). However, Yan et al. (2011) studied the effect of plant height on type I and type II resistance using ten pairs of NILs containing nine different *Rht* genes at the different genomic regions and found that four shorter NILs showed more type II resistance, whereas their taller sister lines showed more type I resistance. It is still unknown if the genes for plant height and FHB resistance are tightly linked or one gene has a pleiotropic effect on both traits (Draeger et al., 2007).

Significant QTLs for FHB resistance in HWW

The result from this study indicated that multiple QTLs might involve in governing the FHB resistance in the RIL population. Five QTLs on chromosomes 4B, 5A, 7A, 1B and 6B were repeatedly detected in four greenhouse experiments and two field trials. Some of them were co-

located with FHB resistance QTLs reported in previous studies (Buerstmayr et al., 2012; Buerstmayr., 2011; Chu et al., 2011; He et al., 2016; Lu et al., 2011; Skinnes et al., 2010)

Qfhb.hwwgr.4B for type II resistance on 4BS from Overland was significant in fall 2017, fall 2018 and mean greenhouse PSS and explained the largest portion of the phenotypic variation (12.33 ~ 16.22%) among all QTL detected in this study. In the fields, however, Everest contributed the resistance allele for low PSS, FDK and DON from field experiments on the same location which explained 17.23%, 21.70% and 8.65% of the phenotypic variation, respectively. The 4BS QTL is the most frequently detected in hexaploid wheat (Buerstmayr & Buerstmayr, 2016; Buerstmayr et al., 2012; Chu et al., 2011; He et al., 2016; Holder, 2018; Liu et al., 2013; Lv et al., 2014; Srinivasachary et al., 2009). Recently, a QTL on the distal end of 4BL has been discovered contributed by Everest in a DH population derived from Everest and WB-Cedar, overlapped with *Fhb4* (Lemes da Silva et al., 2019). The marker *xgwm149* tightly linked to *Fhb4* does not show polymorphism in the Everest/WB-Cedar population. In the current study, we did not found the 4BL QTL, but Everest contributed the resistance allele at 4BS QTL under the field conditions. The reason for this discrepancy may be that Everest is contributing to the resistance QTLs on both arms of chromosome 4B in fields. The differences in parent combination for mapping populations, inoculation methods, disease measuring methods and environmental conditions for FHB evaluation may all contribute to the discrepancy between the two studies. In the current study, the *Qfhb.hwwgr.4BS* was mapped on the same genomic region as the *Rht-B1*, and a strong negative correlation was observed between plant height and FHB severity in the field experiments. The overlapping QTLs were also reported in *Triticum dicoccoides* and durum wheat (Buerstmayr et al., 2012; Prat et al., 2017), and *Triticum aestivum* (Buerstmayr et al., 2012; Chu et al., 2011; He et al., 2016; Liu et al., 2013), supporting the hypothesis of the strong

linkage between the two QTLs. Fine mapping in this QTL region will further resolve if they are the same or linked QTLs for the two traits.

The *Qfhb.hwwgr.5AL* from Overland showed significant type II, type III and type IV resistance in the spring and fall 2017 experiments and mean greenhouse data. Overlapping QTLs have been reported on chromosome arm 5AL and explained up to 35% of the phenotypic variation for type I or type II resistance across different studies (Buerstmayr et al., 2011; Chu et al., 2011; Clinesmith, 2016; He et al., 2016; Malihipour et al., 2017; Sari et al., 2018; Szabó-Hevér et al., 2014). In some studies, this QTL was also overlapped with QTLs for low DON and low FDK (Buerstmayr et al., 2009; Malihipour et al., 2017). Recenlty, Hashimi (2019) mapped the QTL on 5AL with a large effect on FHB resistance from winter wheat CI13227 and Lakin. In that study, the 5AL QTL was mapped in a similar interval as in the present study, suggesting that they are likely the same QTL. *Qfhb.hwwgr.5AL* is on 5AL, whereas *Fhb5* is on the 5AS (Jia et al., 2018), thus they are different QTLs. *Qfhb.hwwgr.5AL* also showed resistance to FDK and DON accumulation, consistent with Malihipour et al (2017). In spring 2018 greenhouse experiment the peak of *Qfhb.hwwgr.5A* was overlapped with a plant height QTL, which agrees with the results of Chu et al. (2011) and Gervais et al. (2002).

Qfhb.hwwgr.7AS from Everest influenced both type II and type III resistance in spring 2017 and mean greenhouse data and explained up to 19.40% of the phenotypic variance for PSS and 12.50% of the phenotypic variance for FDK. QTLs on 7A chromosome have been previously reported on several cultivars including Glenn (ElFatih et al., 2020), Frontana (Mardi et al., 2006), NK93604 (Semagn et al., 2007), and CS-SM3-7ADSL (Jayatilake et al., 2011). Lemes da Silva et al. (2019) reported a QTL on the long arm of chromosome 7A for field FHB resistance in a DH population derived from Everest × WB-Cedar. The difference in the QTL

positions from the same parent suggests that Everest may carry resistance alleles for field and greenhouse PSS on the different arms of the same 7A chromosome. The QTL identified in the current study also shows a significant impact on FDK.

The QTL *Qfhb.hwwgr.4AS* from Overland was significant only in the spring 2017 experiment. Previously, a QTL for type I resistance was found on the short arm of chromosome 4A (Burt et al., 2015; Steed et al., 2005). In a meta-analysis study of five RIL populations, Cai et al. (2019) reported a QTL on 4A for a lower disease rate from Huangfangzhu (HFZ) × Wheaton. Since Overland has no Chinese landrace in its pedigree, this may be a novel QTL from Overland for FHB resistance.

The QTL *Qfhb.hwwgr.6BL* was associated with lower PSS in the fall 2017 greenhouse and 2017-18 field experiments, and explained 13.47 and 8.73% variation for PSS, respectively. Several studies have reported the presence of 6B QTL for type II resistance in greenhouse and field experiments in tetraploid (Somers et al., 2006; Buerstmayr et al., 2012) and hexaploid wheat and this QTL explained 7.5 to 25.0% of the phenotypic variation (Cai, 2016; Cuthbert et al., 2007; ElDoliefy et al., 2020; Lemes da Silva et al., 2019; Yang et al., 2003). *Qfhb.hwwgr.6BL* is on the long arm of chromosome 6B while *Fhb2* was finely mapped on the short arm of chromosome 6B near *Xgwm133*, therefore *Fhb2* is absent in our population. Buerstmayr and Buerstmayr, (2015) also identified favorable allele in the same genomic region in European winter wheat Capo/Arina, corroborating with our findings.

The QTL *Qfhb.hwwgr.1BL* was significant in the fall 2017 greenhouse and 2018-19 field experiments and explained 8.25% and 12.56% of the phenotypic variation. Out of 100 studies that reported QTLs for FHB resistance, 14 studies reported QTL on chromosome 1B (Buerstmayr et al., 2019) including 1BL-1RS translocation from rye chromosome. Previously,

type II resistance QTL has been reported on 1BL in European cultivar Lynx (Schmolke et al., 2005), CIMMYT line CM82036 (Buerstmayr et al., 2002), Chinese landrace Wangshuibai (Zhang et al., 2004), CIMMYT line Seri82 (Mardi et al., 2006) within the 1BL.1RS region. *Qfhb.hwwgr.1BL* was mapped on wheat 1B chromosome and the resistance allele is coming from Everest which is in agreement with previously reported QTLs on 1BL (Klahr et al., 2007; Haberle et al., 2009; Semagn et al., 2007; Holzapfel et al., 2008; Peterson et al., 2016).

The effects of each repeatable QTL and their combinations were assessed by grouping the RILs based on their allele combinations of the three significant QTLs on 4BS, 5AL and 7AS. The lines carrying the only QTL on 4B chromosome showed reduce most PSS compared to the lines carrying resistance alleles at 5A and 7A. Overall, the lines carrying resistance alleles showed a significant reduction in PSS compared to the lines carrying all susceptible alleles at the three QTLs.

Marker-assisted breeding using KASP markers

GBS is one of the robust technique that allowed the quick SNP discovery and genotyping in a diversity panel or a mapping population to identify genomic regions associated with a trait of interest (Li et al., 2015; Lin et al., 2015; Poland et al., 2012). However, a large number of missing data is one of the biggest disadvantages of GBS due to low sequencing depth (Poland et al., 2012). In the current study, GBS markers in QTL regions were converted into KASP markers and the population was re-genotyped with the KASP markers, which not only filled the missing data points but also corrected possible sequencing errors of GBS-SNPs in the QTL regions. Among 22 KASP markers we designed, 12 were polymorphic and segregated in the population. These KASP can help the breeders to pyramid these repeatable resistant QTLs in a new cultivar using marker-assisted selection.

Conclusion

High-density GBS-SNPs allow the identification of resistant QTLs associated with FHB resistance in HWW. To identify native QTLs from Everest and Overland developed from the U.S. Great Plains, we evaluated a RIL population from the cross of Everest x Overland for different types of FHB resistance and genotyped the population using GBS-SNPs. Six QTLs were identified in the RIL population confers type II resistance with the resistance QTLs on 1BL, 4BS, 6BL and 7AS were contributed by Everest and QTLs on 4A, 4BS and 5AL were contributed by Overland. A single QTL from Everest was identified on chromosome 4BS and one QTL from Overland on chromosome 5AL being repeatedly associated with type III resistance. Three repeated QTLs on chromosome 4BS, 5AL and 7AS were identified for type IV resistance with resistance allele on 4BS and 7AL were governed by Everest and resistance allele on 5AL were contributed by Overland. A large effect QTL on 4BS from Everest explained up to 21% of the FDK variation in the two years field experiments. All mapped QTLs have additive effect indicating the pyramiding of favorable alleles would enhance native resistance. Tightly linked markers on 4BS, 5AL and 7AS were converted into KASP assays which can be used to transfer resistance QTL in the breeding program.

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Figures and Tables

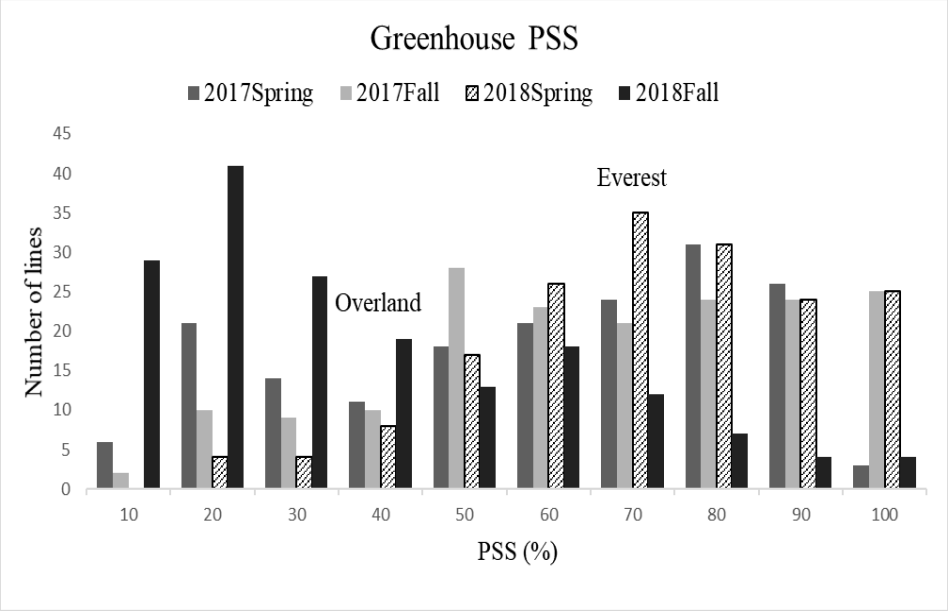
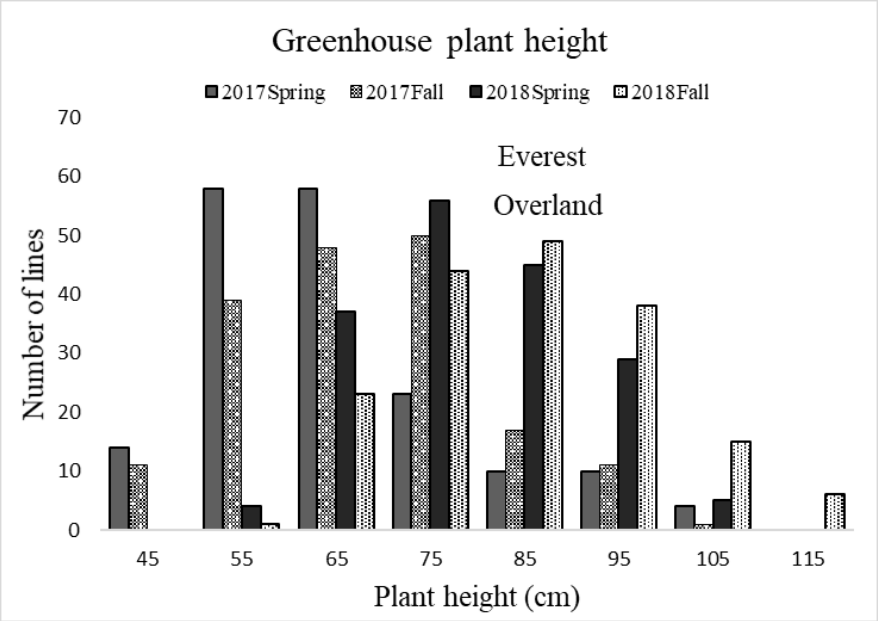


Figure 2.1. Frequency distribution of the mean percentage of symptomatic spikelets in a spike (PSS) for the recombinant inbred line (RIL) population derived from Overland × Everest evaluated in greenhouse experiments 2017 spring, 2017 fall, 2018 spring, 2018 fall.

a)



b)

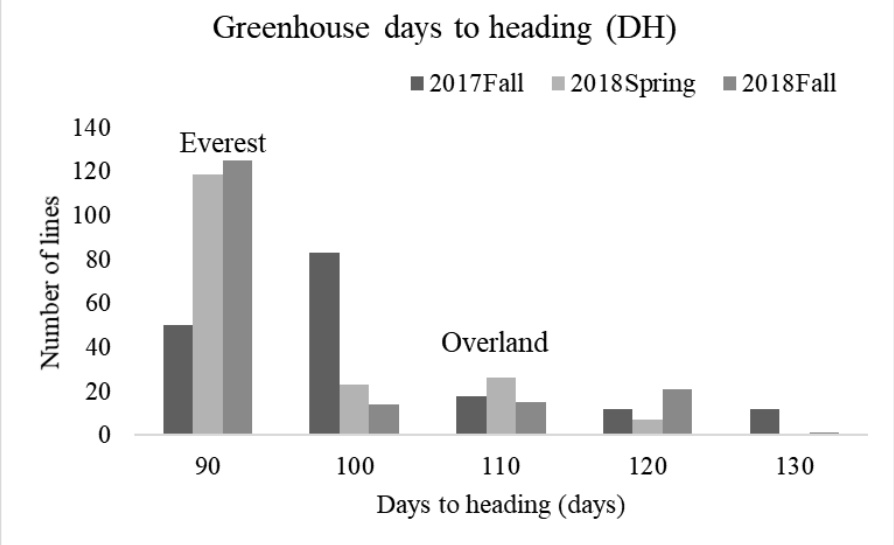


Figure 2.2 (a) Frequency distribution of the mean plant height (PH) for the recombinant inbred line (RIL) population (b) Frequency distribution of mean days to heading for the RIL population derived from Overland × Everest evaluated in greenhouse experiments; 2017 spring, 2017 fall, 2018 spring, 2018 fall.

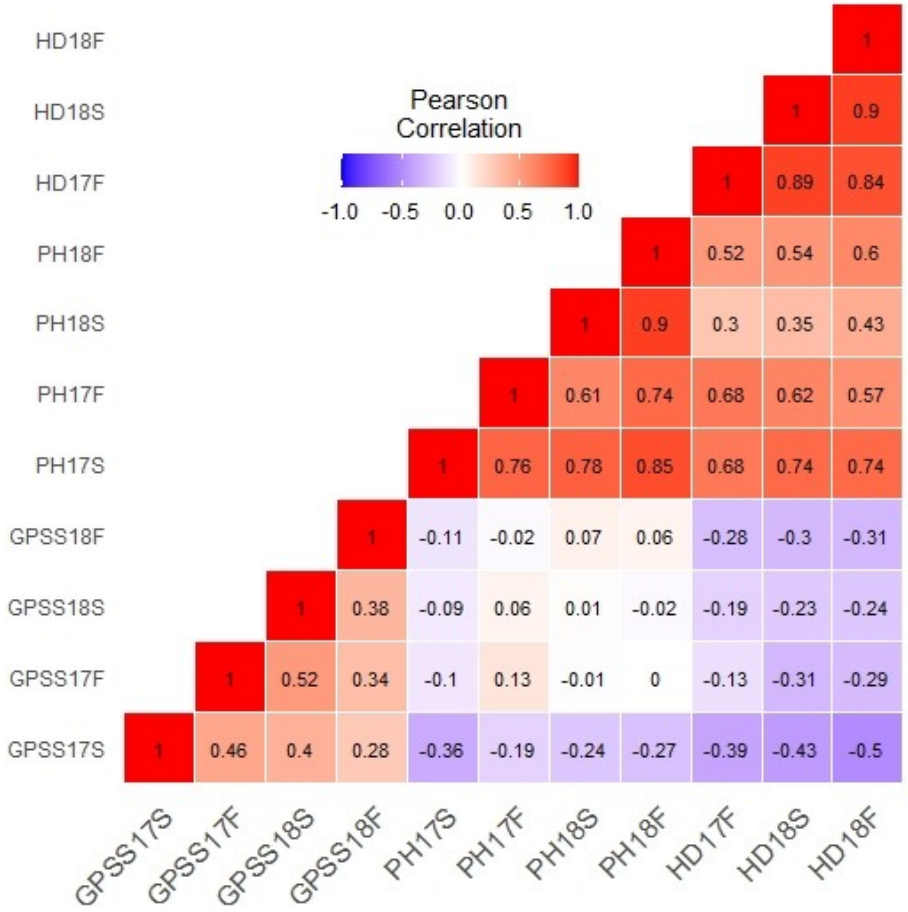


Figure 2.3. Pearson correlations among all the percentage of symptomatic spikelets in a spike (PSS), Fusarium damaged kernels (FDK) and deoxynivalenol (DON) content, plant height (PH) and heading date (HD) evaluated in spring 2017 (GPSS17S), fall 2017 (GPSS17F), spring 2018 (GPSS18S) and fall 2018 (GPSS18F) experiments.

*All the correlations were significant from zero at $P < 0.05$ except for the correlation between DON2017 (DON17) and DON2018 (DON18) with all greenhouses PSS and heading date of fall 2017 (HD17D), spring 2018 (HD18S) and fall 2018 (HD18F). Except for the correlation between spring 2017 PSS (GPSS17S) and plant height, all the correlations between greenhouse PSS and plant height were not significant.

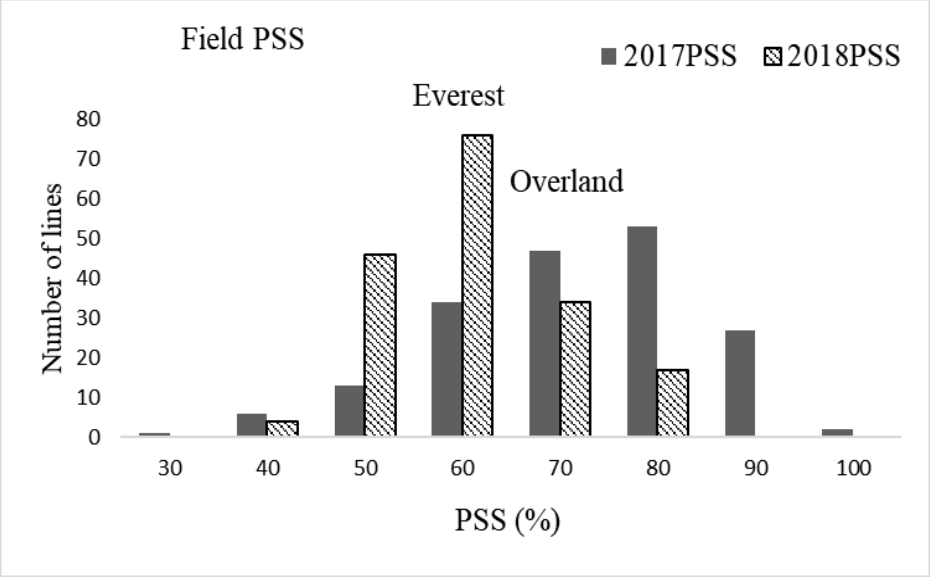


Figure 2.4. Frequency distribution of the mean percentage of symptomatic spikelets in a spike (PSS) for the recombinant inbred line (RIL) population derived from Overland × Everest evaluated in two field experiments conducted in the 2017-18 growing season (2018) and 2018-19 growing season (2019).

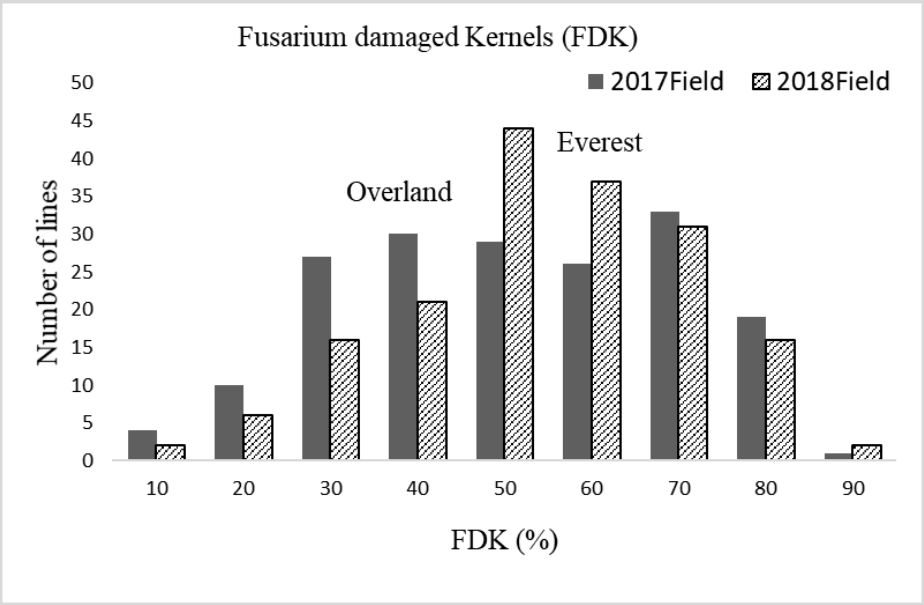


Figure 2.5 Frequency distribution of the mean Fusarium damaged Kernels (FDK) for the recombinant inbred line (RIL) population derived from Overland × Everest evaluated in two field experiments conducted in the 2017-18 growing season (2018) and 2018-19 growing season (2019).

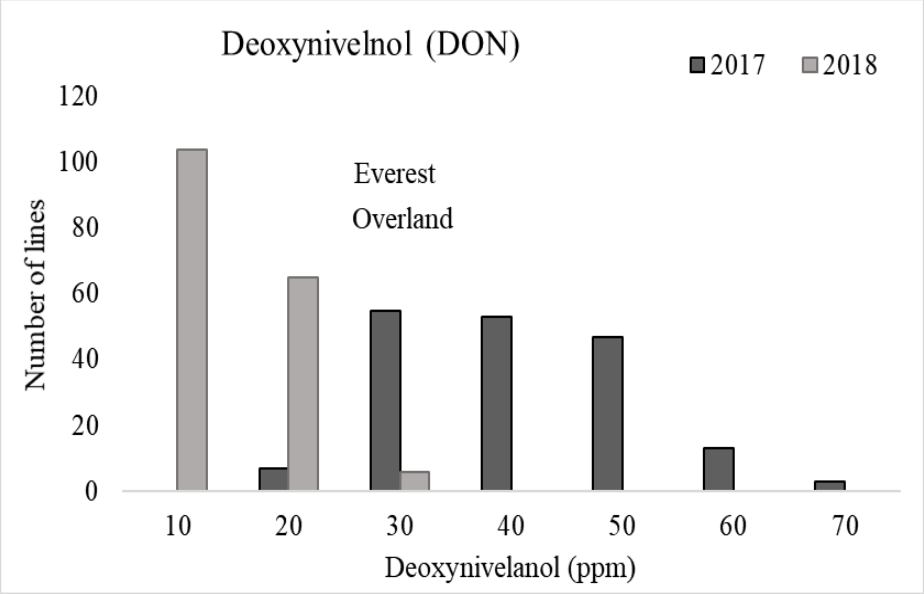
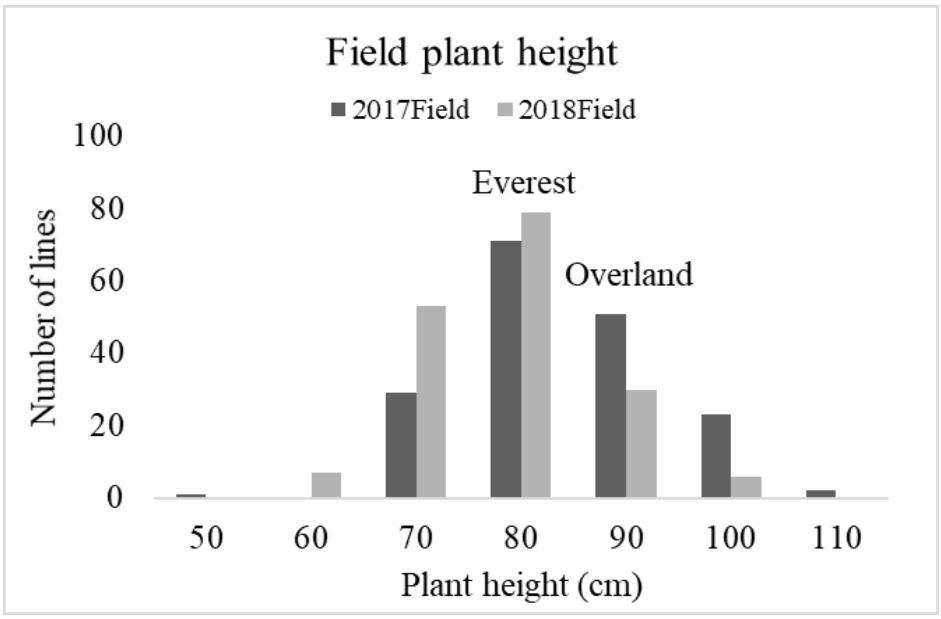


Figure 2.6 Frequency distribution of the mean deoxynivalenol (DON) for the recombinant inbred line (RIL) population derived from Overland × Everest evaluated in two field experiments conducted in the 2017-18 growing season (2018) and 2018-19 growing season (2019).

a)



b)

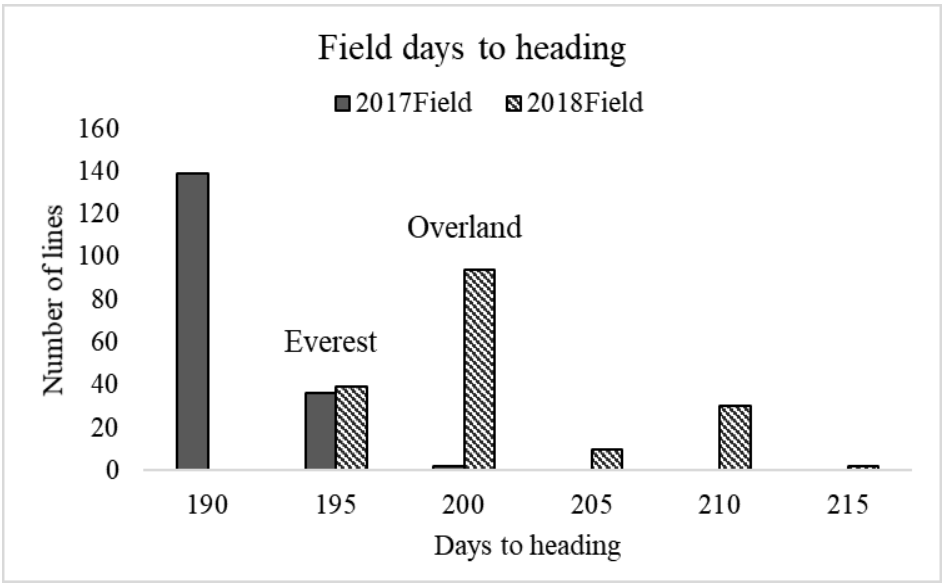


Figure 2.7 Frequency distribution of plant height (PH) and days to heading (HD) in field experiments. (a) Frequency distribution of PH (b) Frequency distribution of HD for the recombinant inbred line (RIL) population derived from Overland × Everest evaluated in two field experiments conducted in the 2017-18 growing season (2018) and 2018-19 growing season (2019).

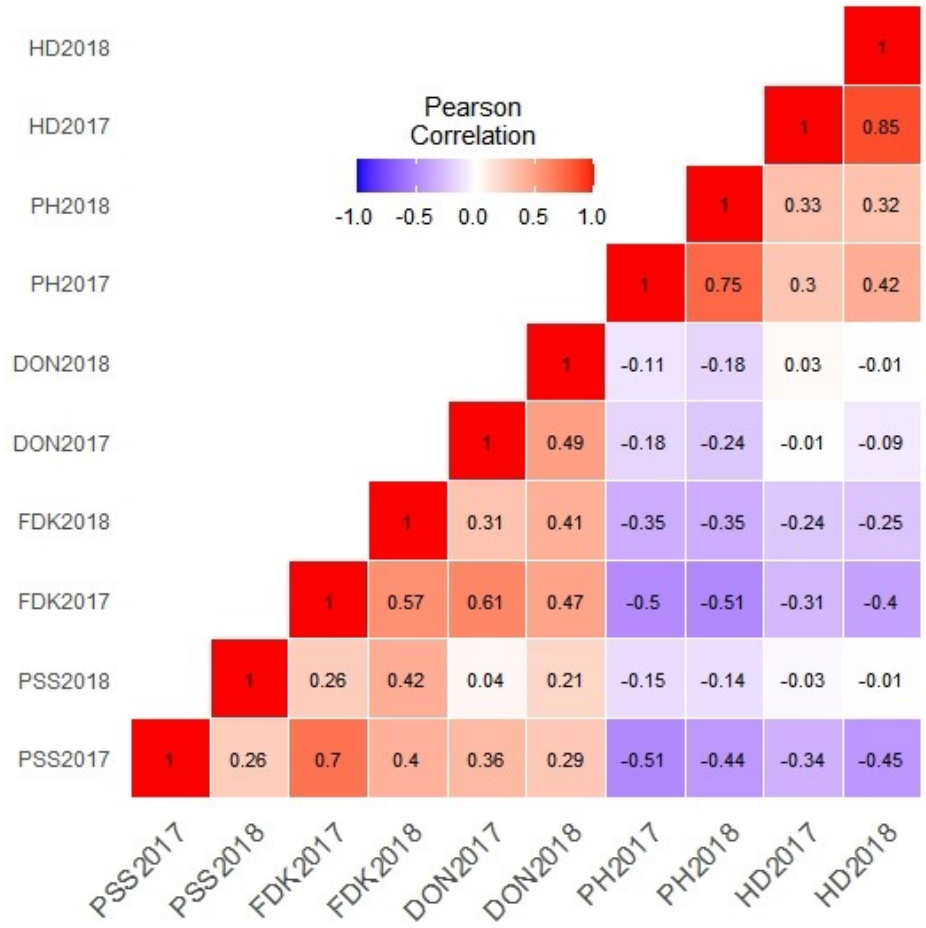


Figure 2.8. Pearson correlations among all the percentage of symptomatic spikelets in a spike (PSS), Fusarium damaged kernels (FDK) and deoxynivalenol (DON) content, plant height (PH) and heading date (HD) evaluated in spring 2017 (GPSS17S), fall 2017 (GPSS17F), spring 2018 (GPSS18S) and fall 2018 (GPSS18F) experiments.

*All the correlations were significant from zero at $P < 0.05$ except for the correlations between DON2017 and field PSS2018, HD2017 and field PSS2018, and HD2018 and PSS2018. The correlations of DON2017 and DON2018 with heading date HD2017 and HD2018 were also not significant.

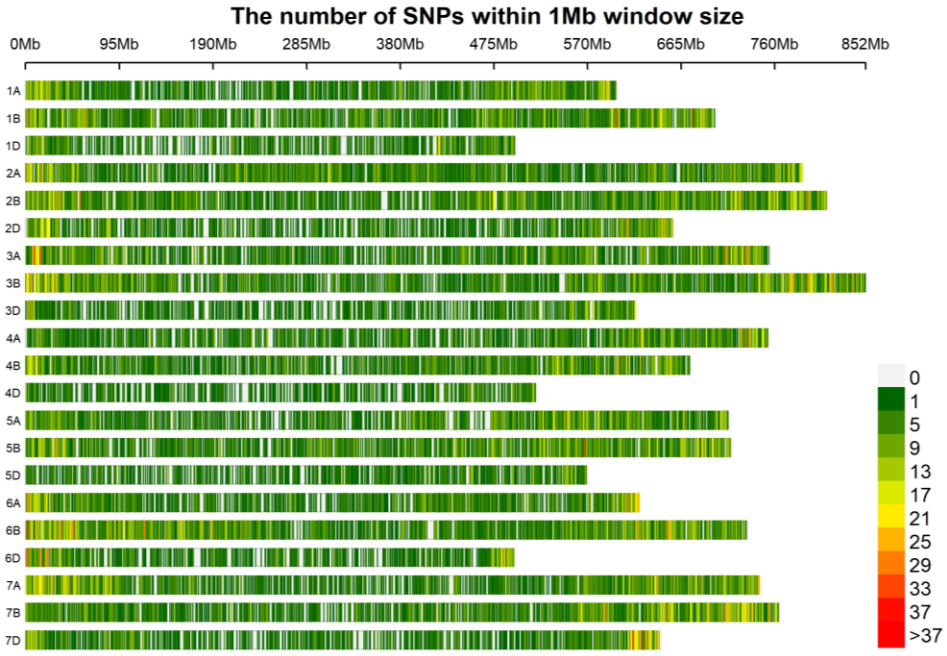


Figure 2.9. Distribution of single nucleotide polymorphisms (SNPs) generated by genotyping by sequencing (GBS) in the wheat genome. The color key with marker densities indicates the number of makers within a window size of 1Mb. The 54,349 SNP markers denote the filtered set with < 20% missing or low-quality data, minor allele frequency (MAF) > 0.2.

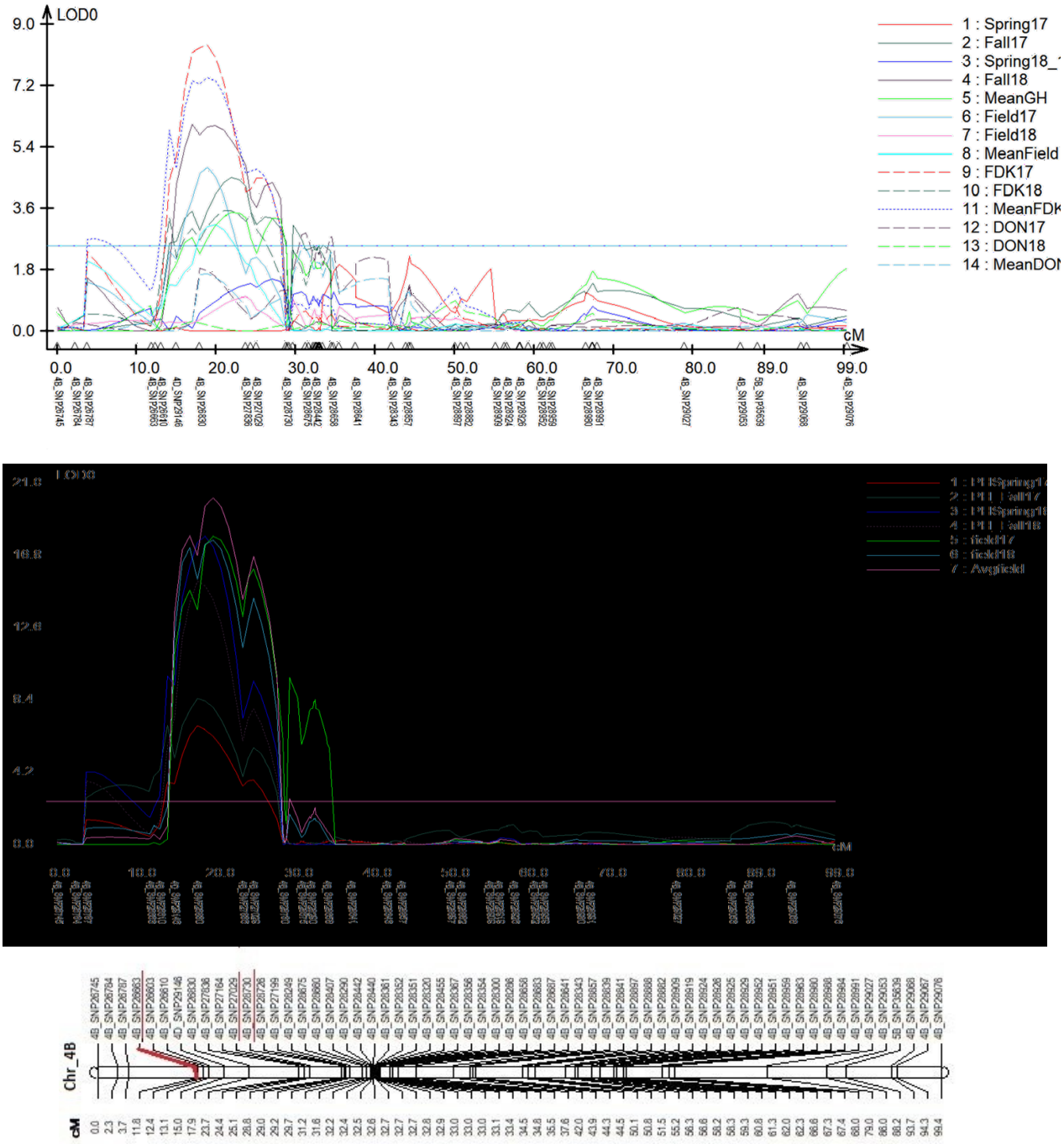


Figure 2.10. Maps of QTLs on 4B for FHB type II resistance (top) and plant height (bottom) constructed using the RIL population derived from the cross Overland × Everest.

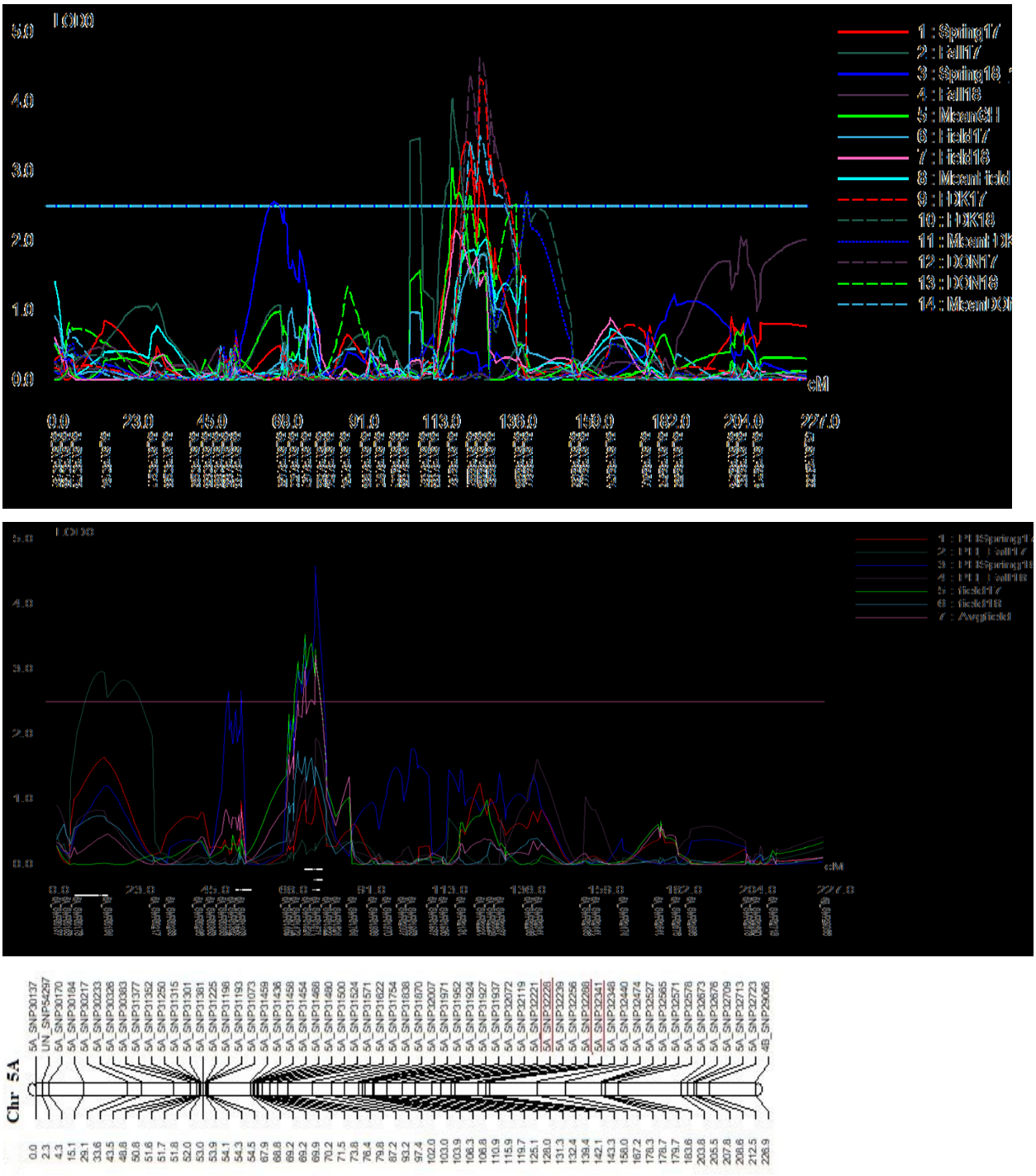


Figure 2.11. Maps of QTLs on 5A for FHB type II resistance constructed from the RIL population derived from the cross Overland × Everest based on four greenhouse and two field experiments.

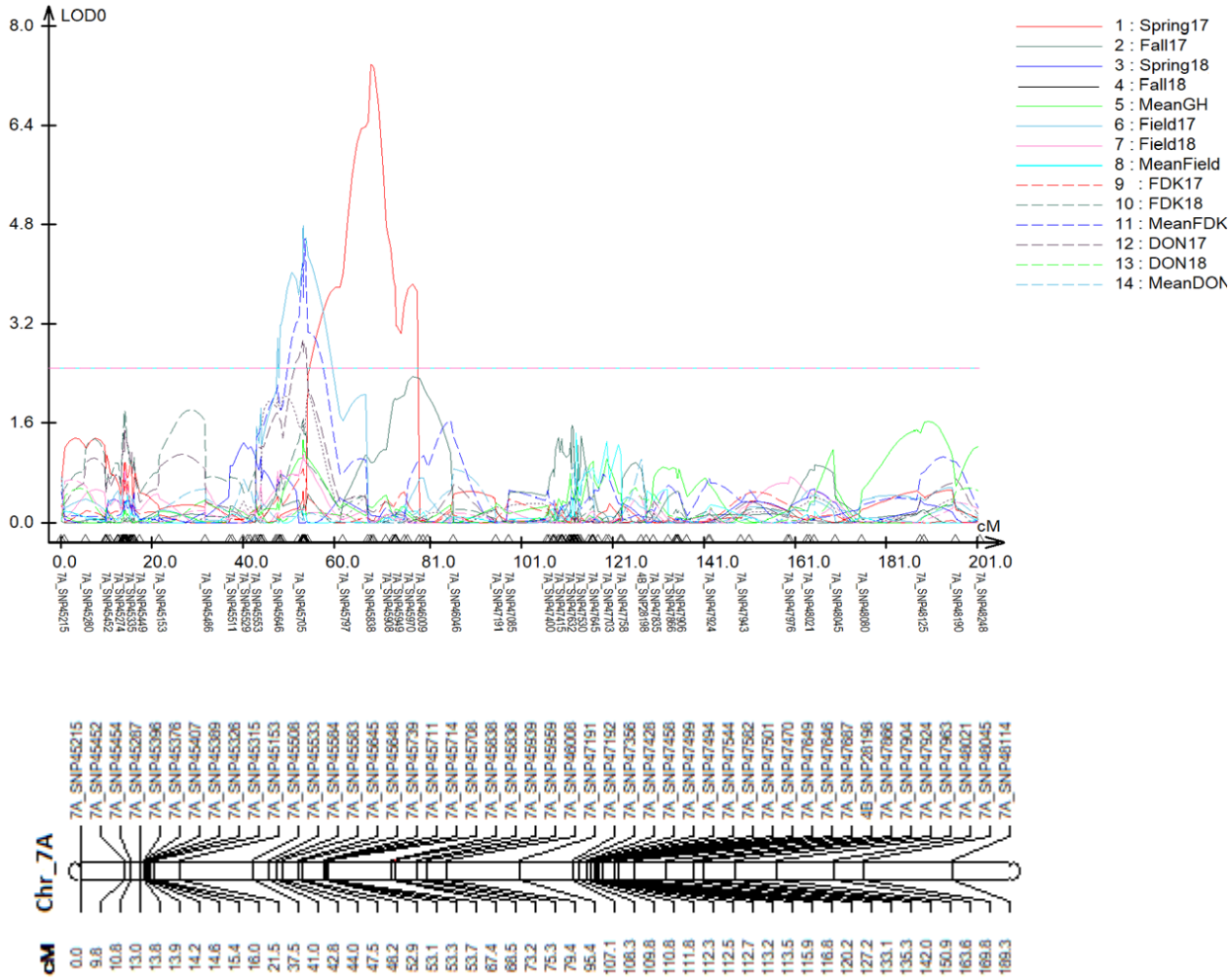


Figure 2.12. Maps of QTLs on 7AS for FHB type II resistance constructed from the RIL population derived from the cross Overland × Everest.

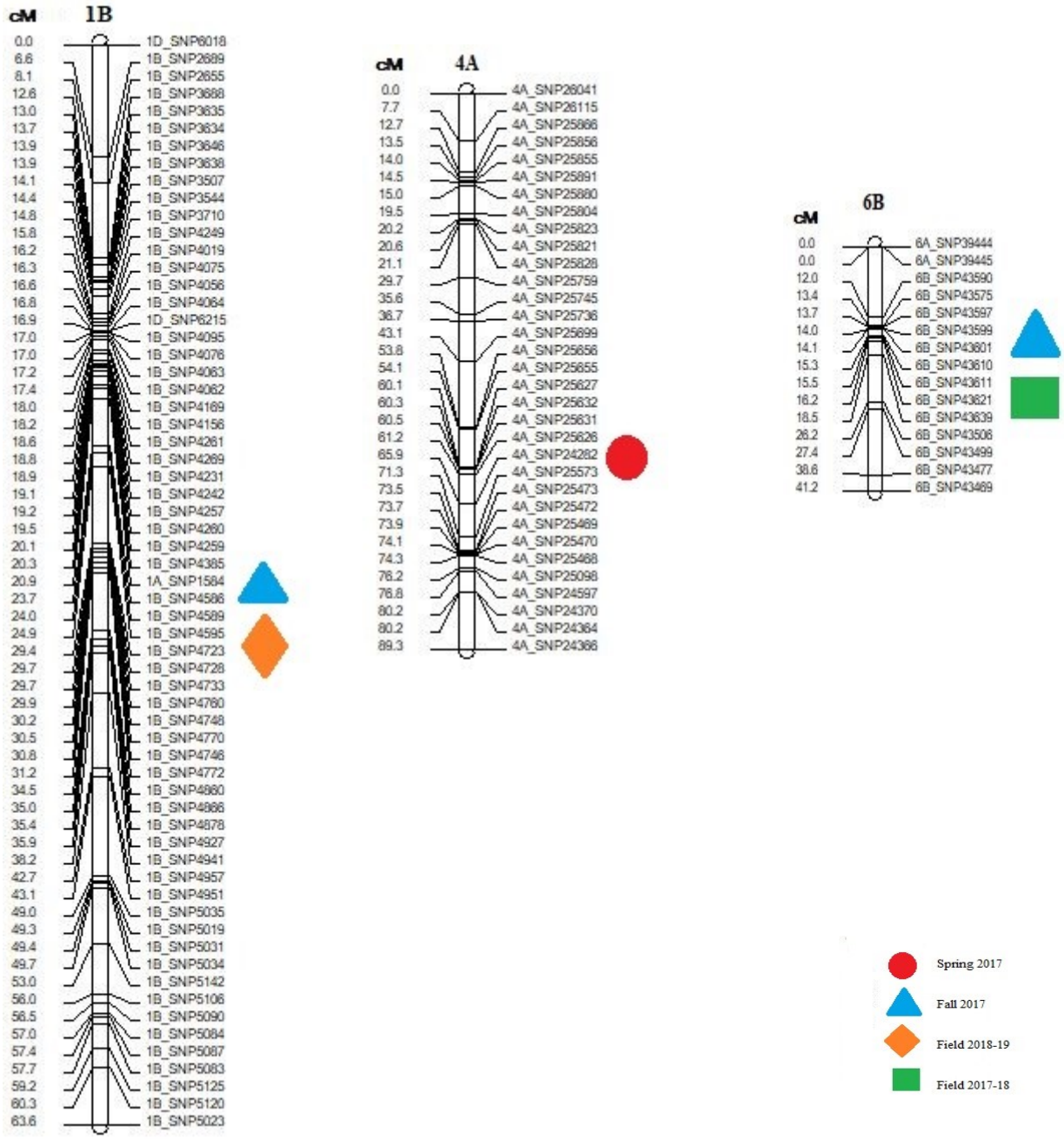
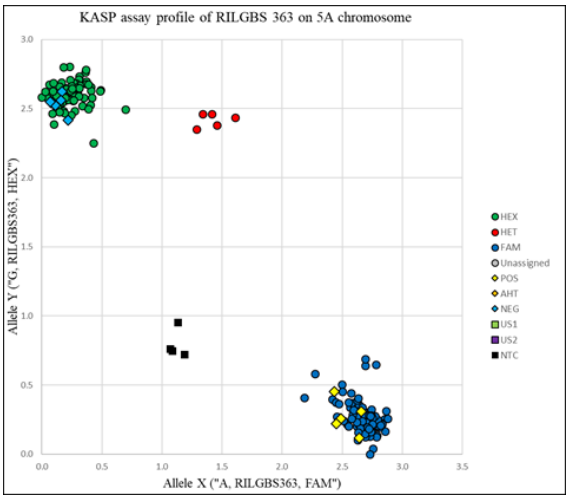
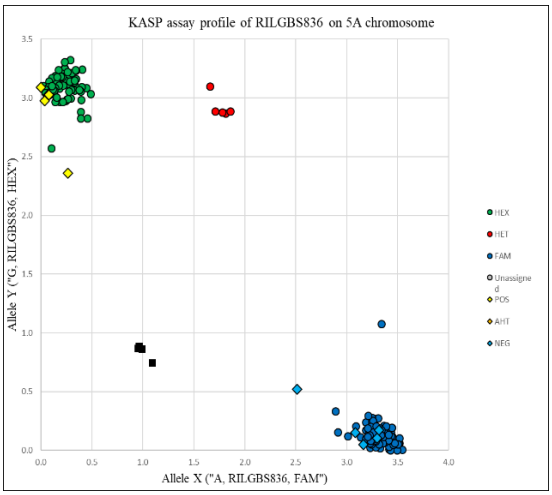


Figure 2.13. Maps of QTLs on 4A, 1B, 6B for FHB type II resistance constructed from the RIL population derived from the cross Overland × Everest.

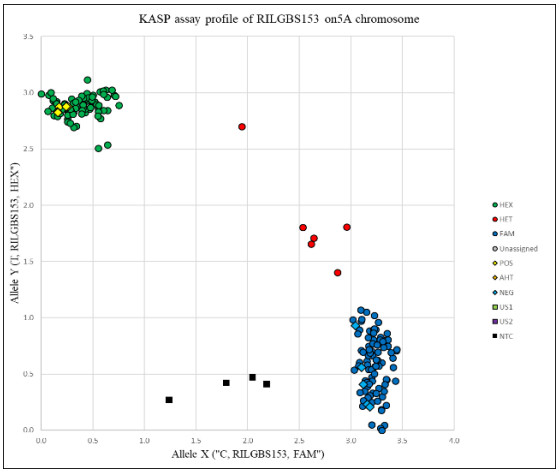
(1) KASP *RILGBS363* on 5A



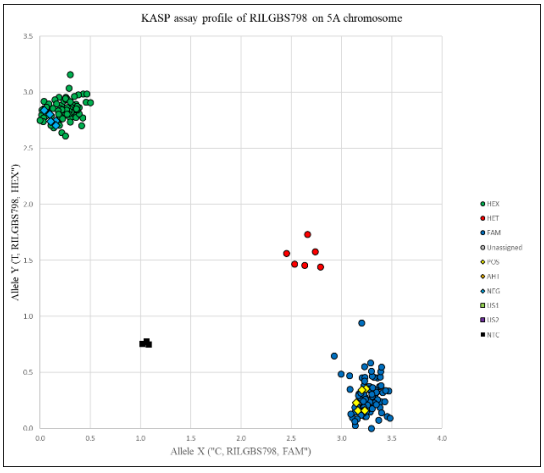
(2) KASP *RILGBS836* on 5A



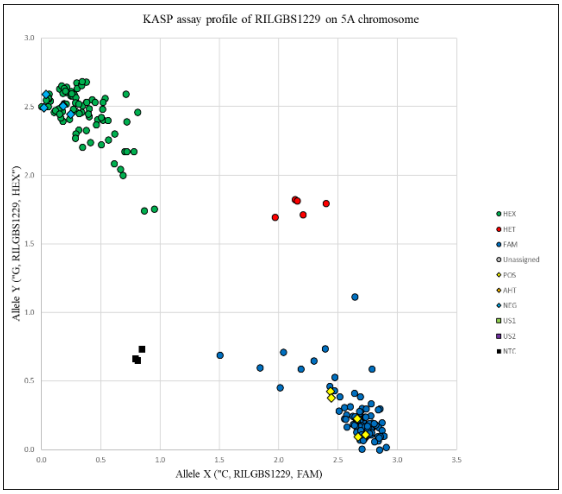
(3) KASP *RILGBS153* on 5A



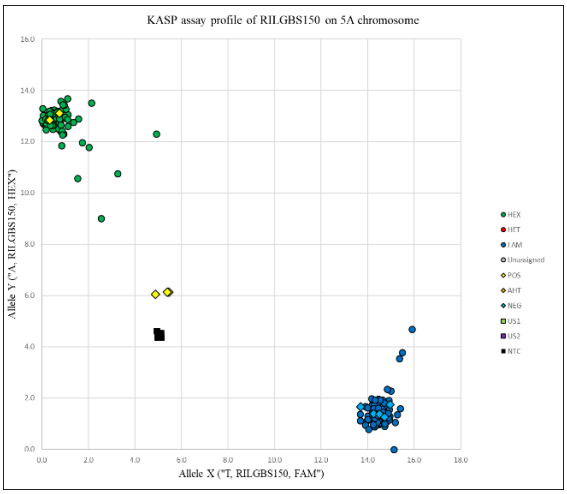
(4) KASP *RILGBS798* on 5A



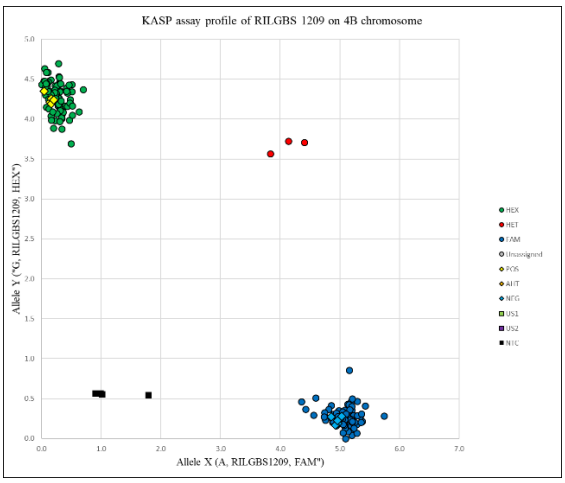
(5) KASP *RILGBS1229* on 5A



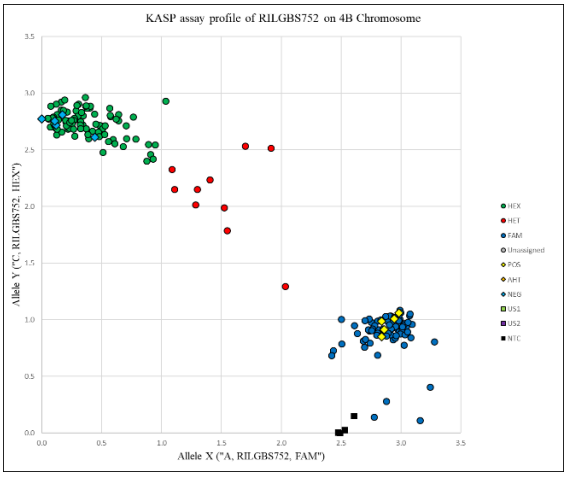
(6) KASP *RILGBS150* on 5A



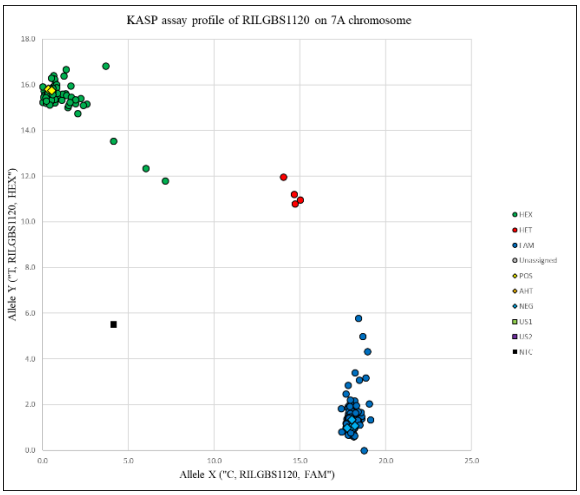
(7) KASP *RILGBS1209* on 4B



(8) KASP *RILGBS752* on 4B



(9) KASP *RILGBS1120* on 7A



(10) KASP *RILGBS292* on 7A

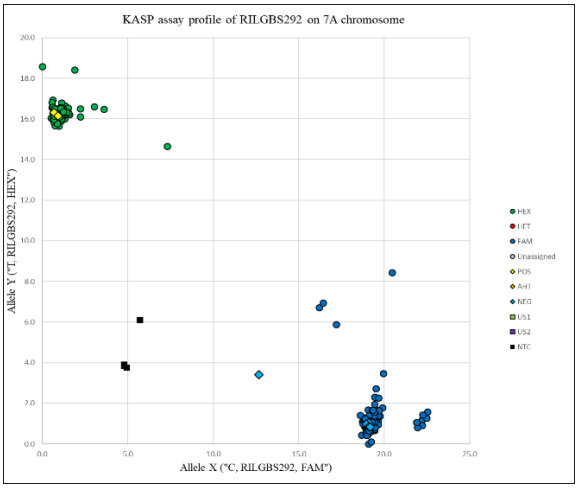


Figure 2.14. KASP markers segregation in the recombinant inbred population of Evrest × Overland. (1) KASP RILGBS 363 on 5A, (2) KASP RILGBS836 on 5A, (3) KASP RILGBS153 on 5A, (4) KASP GBS798 on 5A, (5) KASP RILGBS1229 on 5A, (6) KASP RILGBS150 on 6B, (7) KASP RILGBS1209 on 4B, (8) KASP RILGBS752 on 4B, (9) KASP RILGBS1120 on 7A, (10) KASP RILGBS292 on 7A. Blue dots represent Everest alleles, Yellow dots represents Overland alleles, red dots refer to heterozygotes, and the black crosses or dots are ddH_2O.

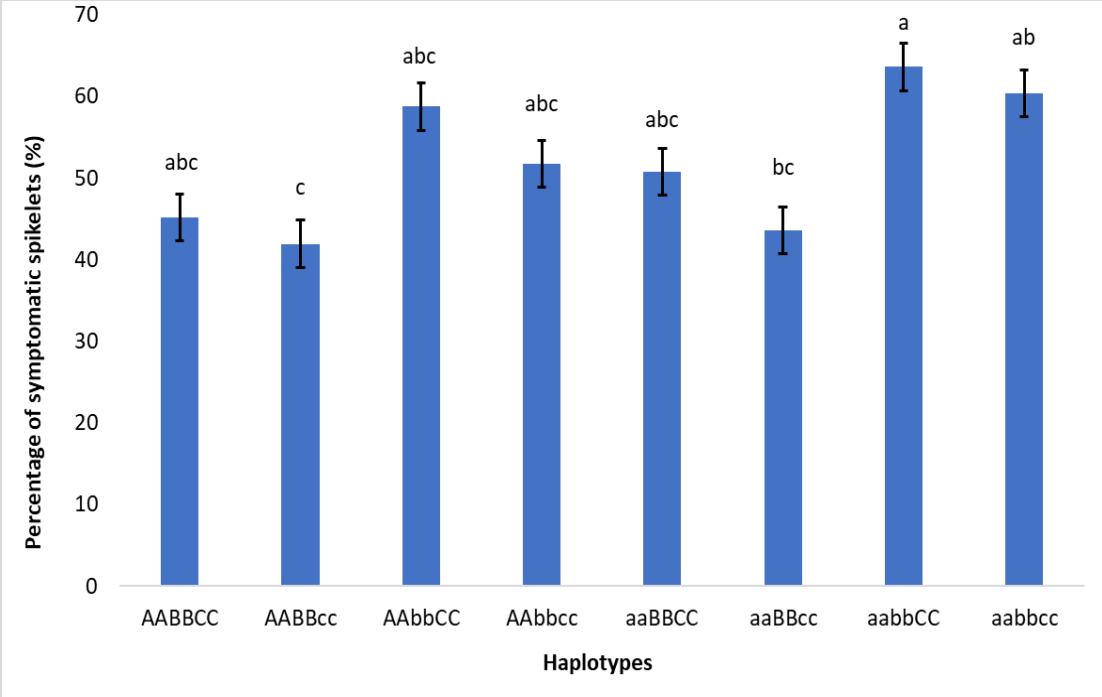


Figure 2.15. Effect of different combinations of three QTLs on 5A, 7A and 4B for the percentage of symptomatic spikelets in a spike (PSS) from greenhouse analyzed in the RIL population. 'Overland' alleles were assigned as AA (5A), BB (4B) and CC (7A) and 'Everest' allele as aa (5A), bb (4B) and cc (7A). The solid bars stand for mean PSS of each group, and the length of each line refers to the standard error of each haplotype.

Table 2.1. Analysis of variance (ANOVA) of the mean percentage of symptomatic spikelets in a spike (PSS) for the RIL population evaluated in four greenhouse experiments.

Source of variation	**DF**	**Sum of Square**	**Mean Square**	**F Value**	**Pr>F**	**H^2**
Experiment	3	1409.65	469.66	2.9216	0.0033	0.78
Genotype	176	52257.21	293.57	1.83	<0.0001	
Genotype x Experiment	519	38675.63	227.503	1.42	0.0039	
Replication(Experiment)	6	1190.04	396.68	2.47	0.0620	
PH (covariate)	1	1.87096	1.8709	0.01	0.0914	
Error	458	53372.09	160.7593			
Total	1163					

Table 2.2. Mean squares of the individual year and across years from the analyses of variance of RIL population evaluated in two field experiments conducting in 2017-18 and 2018-19 growing seasons.

Year	Source	d.f.	PSS	FDK	DON
2017-18	Genotype	176	347.332**	429.959**	197.031*
	Replication	1	616.943ns	109.525 ns	122.791 ns
	Covariate PH	1	103.832ns	3.05786 ns	61.008 ns
	Error	174	118.365	95.0026	108.17182
	Mean	-	68.44	48.33	35.93
	CV (%)	-	15.89	20.16	28.94
2018-19	Genotype	173	240.254*	463.928**	35.971*
	Replication	1	902.295ns	1193.144 ns	330.550*
	Covariate PH	1	111.981ns	676.599 *	1.823 ns
	Error	156	207.24542	102.260	19.32201
	Mean	-	57.50	50.617	9.797708
	CV (%)	-	23.914	19.97794	44.86
Across years	Environment	1	9974.838**	18.8402 ns	59912.191***
	Genotype	176	290.070***	667.5841***	159.518**
	Environment*Genotype	173	206.210*	240.2831***	79.261*
	Replication(Environment)	2	657.268 ns	588.6877 ns	219.838 ns
	Covariate PH	1	10.976ns	544.6028*	26.020 ns
	Error	332	151.955	98.7818	63.1468
	Mean	-	63.1851	49.48127	22.82658
	H^2	-	0.6727	0.8217	0.7421
	CV (%)	-	19.52372	20.08620	34.81247

d.f. degrees of freedom, C.V. coefficient of variation, PSS: final evaluation of the percentage of symptomatic spikelets, FDK : mean Fusarium-damaged kernels, DON : mean deoxynivalenol content, PH plant height. ** and *** represent respectively significance at $p < 0.05$ and $p < 0.01$ of the probability of error, while *ns* indicates the absence of statistical significance.

Table 2.3. Quantitative trait loci (QTLs) for Fusarium head blight resistance detected by composite interval mapping using mean PSS of the RIL population evaluated in four greenhouses and two field experiments.

Trait	Experiments	QTLs	Position (cM)	Position (MB)	LOD	R^2	Flanking markers		Contribute by
PSS	Fall 2017	*Qfhb.hwwgr.4BS*	26.11	19,161,848 - 31,176,508	4.51	12.33	*SNP26787*	*SNP26830*	Overland
PSS	Fall 2018	*Qfhb.hwwgr.4BS*	18.91	19,161,848 - 31,176,508	6.07	16.22	*SNP26787*	*SNP26830*	Overland
PSS	Spring 2018	*Qfhb.hwwgr.4BS*	27.11	19,161,848 - 31,176,508	4.57	12.48	*SNP26787*	*SNP26830*	Overland
PSS	Across greenhouses	*Qfhb.hwwgr.4BS*	16.01	19,161,848 - 31,176,508	3.48	13.19	*SNP26787*	*SNP26830*	Overland
F_PSS	Field 2017-18	*Qfhb.hwwgr.4BS*	27.11	19,161,848 - 31,176,508	4.80	17.23	*SNP26787*	*SNP26830*	Everest
F_PSS	Across field	*Qfhb.hwwgr.4BS*	19.91	19,161,848 - 31,176,508	2.53	7.12	*SNP26787*	*SNP26830*	Everest
FDK	Field 2017-18	*Qfhb.hwwgr.4BS*	25.11	19,161,848 - 31,176,508	8.39	21.70	*SNP26787*	*SNP26830*	Everest
FDK	Field 2018-19	*Qfhb.hwwgr.4BS*	32.31	19,161,848 - 31,176,508	3.34	9.29	*SNP26787*	*SNP26830*	Everest
FDK	Across field	*Qfhb.hwwgr.4BS*	18.91	19,161,848 - 31,176,508	7.45	20.46	*SNP26787*	*SNP26830*	Everest
DON	Field 2017-18	*Qfhb.hwwgr.4BS*	31.21	19,161,848 - 31,176,508	2.86	8.65	*SNP26787*	*SNP26830*	Everest
PSS	Spring 2017	*Qfhb.hwwgr.5AL*	124.61	556,092,890 - 618,167,762	3.42	9.49	*SNP31937*	*SNP32341*	Overland
PSS	Fall 2017	*Qfhb.hwwgr.5AL*	119.81	556,092,890 - 618,167,762	4.03	11.10	*SNP31937*	*SNP32341*	Overland
PSS	Spring 2018	*Qfhb.hwwgr.5AL*	66.01	556,092,890 -	2.56	7.19	*SNP31937*	*SNP32341*	Overland

				618,167,762					
PSS	Across greenhouses	*Qfhb.hwwgr.5AL*	119.81	556,092,890 - 618,167,762	3.04	8.48	*SNP31937*	*SNP32341*	Overland
FDK	Field 2017-18	*Qfhb.hwwgr.5AL*	128.01	556,092,890 - 618,167,762	4.33	11.86	*SNP31937*	*SNP32341*	Overland
FDK	Field 2018-19	*Qfhb.hwwgr.5AL*	142.11	556,092,890 - 618,167,762	2.69	7.54	*SNP31937*	*SNP32341*	Overland
FDK	Across field	*Qfhb.hwwgr.5AL*	142.11	556,092,890 - 618,167,762	2.71	7.61	*SNP31937*	*SNP32341*	Overland
DON	Field 2017-18	*Qfhb.hwwgr.5AL*	128.01	556,092,890 - 618,167,762	4.63	12.63	*SNP31937*	*SNP32341*	Overland
DON	Field 2018-19	*Qfhb.hwwgr.5AL*	125.11	556,092,890 - 618,167,762	2.67	7.49	*SNP31937*	*SNP32341*	Overland
DON	Across field	*Qfhb.hwwgr.5AL*	125.11	556,092,890 - 618,167,762	3.42	9.50	*SNP31937*	*SNP32341*	Overland
PSS	Spring 2017	*Qfhb.hwwgr.7AS*	68.01	48,550,888 - 86,783,839	7.40	19.40	*SNP45639*	*SNP45970*	Everest
PSS	Across greenhouses	*Qfhb.hwwgr.7AS*	53.11	48,550,888 - 86,783,839	4.74	12.92	*SNP45639*	*SNP45970*	Everest
FDK	Field 2018-19	*Qfhb.hwwgr.7AS*	53.31	48,550,888 - 86,783,839	4.58	12.50	*SNP45639*	*SNP45970*	Everest
PSS	Spring 2017	*Qfhb.hwwgr.4A*	75.31	29,824,097 - 60,334,589	4.43	12.11	*SNP24282*	*SNP24370*	Overland
PSS	Fall 2017	*Qfhb.hwwgr.1BL*	23.71	460,806,875 - 572,303,559	2.95	8.25	*SNP4056*	*SNP4595*	Everest
F_PSS	Field 2018-19	*Qfhb.hwwgr.1BL*	16.31	460,806,875 - 572,303,559	4.60	12.56	*SNP4056*	*SNP4595*	Everest
PSS	Fall 2017	*Qfhb.hwwgr.6BS*	15.11	718,194,422 - 724,799,167	4.96	13.47	*SNP43590*	*SNP43639*	Everest
F_PSS	Field 2017-18	*Qfhb.hwwgr.6BS*	13.41	718,194,422 - 724,799,167	3.13	8.73	*SNP43590*	*SNP43639*	Everest

Table 2.4. List of KASP assays developed from GBS SNPs markers

Primer name	Position	Primer Sequence (5'-3')
5A_RILGBS363FAM	5A	gaaggtgaccaagttcatgctGCTAGCTTCTGGTGTAAGT
5A_RILGBS363HEX	5A	gaaggtcggagtcaacggattGCTAGCTTCTGGTGTAAGC
5A_RILGBS363R	5A	ACGATGACAATGGCATACCTG
5A_RILGBS836FAM	5A	gaaggtgaccaagttcatgctATCAGGTTCTTGGCGACA
5A_RILGBS836HEX	5A	gaaggtcggagtcaacggattATCAGGTTCTTGGCGACG
5A_RILGBS836R	5A	CACTAGGAAGTTCTAGTCGAAGTT
5A_RILGBS153FAM	5A	gaaggtgaccaagttcatgctATGATCAGCATTTGTTCCAC
5A_RILGBS153HEX	5A	gaaggtcggagtcaacggattATGATCAGCATTTGTTCCAT
5A_RILGBS153R	5A	TCTGCCGCCGACTTGTATAG
5A_RILGBS798FAM	5A	gaaggtgaccaagttcatgctCTGCATTGGACAGAACAAAAC
5A_RILGBS798HEX	5A	gaaggtcggagtcaacggattCTGCATTGGACAGAACAAAAT
5A_RILGBS798R	5A	CGTTTCAGTGGATTGGTTCAG
5A_RILGBS1229FAM	5A	gaaggtgaccaagttcatgctATAACCAGCGAGGAGAAGAG
5A_RILGBS1229HEX	5A	gaaggtcggagtcaacggattATAACCAGCGAGGAGAAGAC
5A_RILGBS1229R	5A	TGAAACGATGCTGCAGTTACTTA
5A_RILGBS213FAM	5A	gaaggtgaccaagttcatgctCCTCCCTGACCATTGTCA
5A_RILGBS213HEX	5A	gaaggtcggagtcaacggattCCTCCCTGACCATTGTCG
5A_RILGBS213R	5A	GGCCCTGCAAGTTCATTGT
4B_RILGBS752FAM	4B	gaaggtgaccaagttcatgctTCCAACTGCCACACCTA
4B_RILGBS752HEX	4B	gaaggtcggagtcaacggattTCCAACTGCCACACCTC
4B_RILGBS752R	4B	CATGAAATGGTTGCCTTCAGTC
4B_RILGBS1209FAM	4B	gaaggtgaccaagttcatgctTGTAGACTGCAGTGGTAGTA
4B_RILGBS1209HEX	4B	gaaggtcggagtcaacggattTGTAGACTGCAGTGGTAGTG
4B_RILGBS1209R	4B	ACGTGAACCATCTTGCGA
7A_RILGBS1120FAM	7A	gaaggtgaccaagttcatgctGTCGGGCTGTCTGCG
7A_RILGBS1120HEX	7A	gaaggtcggagtcaacggattGTCGGGCTGTCTGCA

7A_RILGBS1120R	7A	GGCCGGGATGACATGATC
7A_RILGBS292FAM	7A	gaaggtgaccaagttcatgctGCCTTGGCCCGCC
7A_RILGBS292HEX	7A	gaaggtcggagtcaacggattGCCTTGGCCCGC
7A_RILGBS292R	7A	ACCCCAAACTAGGGCCAATA
7A_RILGBS415FAM	7A	gaaggtgaccaagttcatgctGAAGAGAGATTCAGCACTGA
7A_RILGBS415HEX	7A	gaaggtcggagtcaacggattGAAGAGAGATTCAGCACTGG
7A_RILGBS415R	7A	CTCCATTCTGGGGTGTGTTG
7A_RILGBS969FAM	7A	gaaggtgaccaagttcatgctGCAGGGACCTTCGTCT
7A_RILGBS969HEX	7A	gaaggtcggagtcaacggattGCAGGGACCTTCGTCG
7A_RILGBS969R	7A	ACAAAATTTACCTGCAGGGC
7A_RILGBS89FAM	7A	gaaggtgaccaagttcatgctCAAAGAGGGTTCGTCACAG
7A_RILGBS89HEX	7A	gaaggtcggagtcaacggattCAAAGAGGGTTCGTCACAA
7A_RILGBS89R	7A	CCACAGTCTCAAGCGCCTTA
7A_RILGBS369FAM	7A	gaaggtgaccaagttcatgctCTATCGAGGCAGAGAATAAGTG
7A_RILGBS369HEX	7A	gaaggtcggagtcaacggattCTATCGAGGCAGAGAATAAGTT
7A_RILGBS369R	7A	GCAAGTCCAACATGTCCAACA
7A_RILGBS439FAM	7A	gaaggtgaccaagttcatgctCAGCCACAGCGACGTA
7A_RILGBS439HEX	7A	gaaggtcggagtcaacggattCAGCCACAGCGACGTC
7A_RILGBS439R	7A	GAAGAGCGTGACGTCCAGG
7A_RILGBS498FAM	7A	gaaggtgaccaagttcatgctTTCCAAGCATGTTCTAGTTTCTC
7A_RILGBS498HEX	7A	gaaggtcggagtcaacggattTTCCAAGCATGTTCTAGTTTCTT
7A_RILGBS498R	7A	TACCACCCATGCGGAAAA

Chapter 3 - Mapping quantitative trait loci for type I FHB resistance in Everest and Overland using genotyping-by-sequencing

Introduction

Among the five types of resistance, type I (resistance to fungal initial infection) and type II (resistance to fungal spread within the spike) have been considered the major types of resistance (Bai & Shaner, 2004), and the other three types of resistance may depend on the two types of resistance. Type II resistance is easier to phenotypically evaluate, is considered a more stable type of resistance, and has been extensively studied. Despite the importance of type I resistance, it has not been well characterized due to 1) phenotype is significantly affected by environments; 2) resistant cultivars with stable type I cultivars have not been reported and; 3) robust methods of phenotyping are lacking (Lin et al., 2006b). Currently, type I resistance can be evaluated by spraying a spore suspension, also known as spray inoculation (SI) over wheat spikes and scoring the wheat spikes before symptoms spread within a spike by counting the number of initially infected spikelets in the spike. Spray inoculation can be used to assess both type I and type II resistance by taking disease notes at different times, thus type I resistance can be confounded by type II resistance if disease notes for type I resistance are not taken at a right time (Tóth et al., 2008).

Among more than 500 QTLs that have been identified for FHB resistance (Buerstmayr et al., 2019), only a few are type I resistance. The QTLs associated with type I resistance have been frequently reported on chromosomes 5A (Buerstmayr et al., 2009; Lin et al., 2006a; Steiner et al., 2004b), 4B (Lin et al., 2006a), and 4A (Burt et al., 2015; Steed et al., 2005). Type I resistance

is important because it confers resistance to colonization by toxin-producing *Fusarium* species. In current breeding programs, Sumai 3 and its derived lines are widely used as resistant sources (Buerstmayr et al., 2009). The high resistance of Sumai 3 is because it harbors type I (Buerstmayr et al., 2003), type II (Anderson et al., 2001a), and type III resistances (Lemmens et al., 2005). In addition, the Brazilian cultivar Frontana has also been reported as a source of type I resistance due to its morphological features such as hard glume, narrow floral opening, and spike compactness (Buerstmayr et al., 2009). Other resistant sources include hexaploid wheat *Triticum macha* (Burt et al., 2015), the Swiss wheat cultivar Arina (Paillard et al., 2004), Chinese landrace Wangshuibai (Jia et al., 2005; Jia et al., 2018) have moderate type I resistance, but these accessions also had undesirable agronomic traits, therefore not being widely used as parents in breeding (Chu et al., 2011). It is highly desirable to find additional sources of resistance to breed for durable resistance through gene pyramiding.

Breeding with native FHB resistant sources has an advantage in avoiding introgression of alien segments from wheat relatives and quick incorporation of resistance QTLs into breeding programs. Everest is widely adapted in Kansas due to its effect in lowering the FHB infection rate (Bockus et al., 2012; Fritz et al., 2011). FHB resistance QTLs were identified in Everest on chromosomes 1BS, 3DS, 4BL, and 5AS for type II resistance (Lemes da Silva et al., 2019). Everest was shown to confer a high level of type I resistance observed from SI, but not evident using single floret inoculation (Chapter 2). Therefore, we took the advantage of already developed RIL population of Everest × Overland to map type I FHB resistance QTLs. The objectives of this study were to 1) identify QTLs associated with type I FHB resistance in the recombinant inbred line (RIL) population of Overland × Everest using spraying inoculation; 2) identify tightly linked GBS-SNPs to the QTLs and convert them to Kompetitive allele-specific

PCR (KASP) assays for marker-assisted selection (MAS) and; 3) evaluate the association of morphological and developmental traits on type I FHB resistance in the RIL population in the greenhouse experiments.

Materials and Methods

Plant materials

A recombinant inbred line (RIL) population consisting of 178 individuals was derived from the cross between hard red winter wheat cultivars Everest (HBK1064-3/Jaggerw//X960103) and Overland (Millennium sib//Seward/Archer). Everest has moderate type I resistance and Overland has moderate type I susceptibility (Clinesmith et al., 2019; Eckard et al., 2015; Lemes da Silva et al., 2019).

Phenotyping the RIL population for type I resistance

The RIL population and both parents were assessed for type I FHB resistance in four greenhouse experiments (fall 2018, spring and fall 2019, spring 2020) using randomized complete block design (RCBD) with two replications in each experiment. A detailed description on greenhouse plant management, inoculum preparation and FHB scoring were described in Chapter 2. At the anthesis stage, each spike was sprayed with a 2 mL conidial spore suspension (~20,000 spores/spike) with five spikes per pot. The inoculated plants were moved into a moist chamber with 100% relative humidity at 21 ± 5 °C for 60 h. When the disease symptoms appeared on multiple spikelets in each inoculated spike, the plants were moved to greenhouse benches for further disease symptom development. The infected spikelets in each spike were counted seven days post-inoculation to calculate the percentage of symptomatic spikelets (PSS) using equation 2.1 described in Chapter 2.

Heading dates of RIL population and parents were recorded at the emergence of 50% spikes from the flag leaves. Plant height was measured prior to harvesting from the soil surface to the tip of the spike excluding awns.

The statistical analysis of greenhouse data was performed using PROC GLIMMIX using SAS statistical software version 9.4 (SAS Institute Inc. 2011). The model $Y_{ijk} = u + g_i + t_j + gt_{ij} + r(t)_{ik} + e_{ijk}$ where Y_{ijk} is the trait, g_i states the fixed effect of the i^{th} genotype, t_j states the random effect of the j^{th} experiment, gt_{ij} states the random interaction of the i^{th} genotype with the j^{th} experiment, $r(t)_{ik}$ defines the random effect of the k^{th} replicate nested within the j^{th} experiment, and e_{ijk} is the random error term. Adjusted mean comparisons between parents and RIL lines were performed using a Tukey-Kramer test (Tukey, 1949). Pearson correlations for all traits were estimated using a ggplot2 package in RStudio v 1.2.5001 (RStudio, Boston, MA). The broad-sense heritability (*H*) was calculated using the method described in Chapter 2.

Genotyping and QTL analysis

Methods for DNA extraction and normalization, GBS library construction and sequencing, SNP calling and filtration, linkage map construction, QTL mapping, and KASP assays were described in Chapter 2.

Results

Phenotypic analysis of the parents and RIL population

Analysis of variance (ANOVA) showed significant genotypic effects and genotype-by-experiment effects for the PSS data (Table 3.1). The RIL population showed normal distribution for mean PSS in each individual experiment and mean greenhouse data (Figure 3.1). Everest showed significantly lower PSS for type I in all greenhouse experiments with an overall mean PSS of 32.79%, ranging from 23.41 to 40.08%; while Overland had a higher PSS of 56.8%,

ranging from 39.84 to 70.26% (Table 3.2, Fig. 3.1). In fall 2019, significant difference was not observed for the mean PSS between Overland and Everest. Mean PSS of RILs ranged from 43.29% (fall 2018) to 53.83% (spring 2019), indicating the highest disease pressure in the spring 2019 experiment and the lowest in the fall 2018 experiment. Transgressive segregations were observed in all greenhouse experiments suggesting that both parents contributed the QTLs for type I resistance in the population. Significant differences were observed between two parents for heading date (HD) in all the four greenhouse experiments. The pairwise Pearson correlations were positive among PSS of the four greenhouse experiments (r = 0.26 to 0.54, $P < 0.05$). Significantly positive correlations were also observed between PSS and plant height (PH) in three greenhouse experiments (r = 0.10 to 0.36, $P < 0.05$) except for the spring 2020 experiment (Fig. 3.2). PSS showed a significantly negative correlation with HD in all greenhouse experiments (r = -0.37 to -0.93, $P < 0.05$). The broad-sense heritability of PSS across greenhouse experiments was 0.77.

QTL analysis

Linkage maps were constructed using GBS derived SNPs from 158 RILs as described in Chapter 2. Briefly, 2,593 polymorphic markers after removal of duplicate markers were used for linkage mapping. A total of 2,018 SNPs were mapped in 32 linkage groups representing all 21 wheat chromosomes.

Composite interval mapping (CIM) detected seven QTLs for type I FHB resistance on chromosome arms 1AL, 3BL, 4BS, 4BL, 6AL, 6BL, 7AS and 7BL using the PSS data from the four greenhouse experiments (Table 3.1, Fig. 3.4). The QTL on 4BL, designated as *Qfhb.hwwgr.4BL*, showed a significantly major effect on type I resistance, situated pericentromeric region and explained 14.89% of the phenotypic variation in fall 2019 experiment

(Table 3.3). The QTL was mapped between the markers *SNP28407* and *SNP28687* at 545.94 to 604.79 Mb, respectively. Another QTL, designated as *Qfhb.hwwgr.4BS,* was mapped for type I resistance using mean greenhouse data located ~17 cM away from *Qfhb.hwwgr.4BL* (Table 3.3). The resistance allele of the QTL was from Overland. This QTL overlapped with a PH gene (*Rht-1B*) in the same interval and explained 11.50% of the phenotypic variation with the peak marker *SNP26830* positioned at 31.17 Mb. *Rht-B1* was significant in all greenhouse experiments and explained up to 46% of the phenotypic variation for PH.

The QTL *Qfhb.hwwgr.7BL* for type I resistance from Everest was mapped at 41.71 cM and explained 10.28% of the phenotypic variation using the mean greenhouse data. The peak markers *SNP51134* was positioned at 710.10 Mb on the long arm of chromosome 7B. Only four markers were mapped to this QTL region with a 9 cM interval.

The QTL on 1AL, designated as *Qfhb.hwwgr.1AL* flanked by *SNP1624* and *SNP1892* was significant for fall 2019 and mean greenhouse PSS data. This QTL explained 8.82 to 9.26% of the phenotypic variation (Table 3.3, Fig. 3.3). The QTL interval had a large interval of 50 Mb with a peak near *SNP1624* at 496.01 Mb. The resistance allele was contributed by Everest. Another QTL, *Qfhb.hwwgr.3BL* flanked by *SNP22576* and *SNP22678,* was significant only in the spring 2020 experiment and explained 9.38% of the phenotypic variation. The resistance allele for this QTL was also contributed by Everest (Table 3.2, Fig. 3.3).

Two QTLs for FHB resistance, designated as *Qfhb.hwwgr.7AS* and *Qfhb.hwwgr.7AL,* were mapped on the short and long arm of chromosome 7A, respectively. Everest contributed to the resistance alleles at both QTLs. The *Qfhb.hwwgr.7AL* was significant in fall 2019 only, flanked by *SNP47865* (684.57Mb) and *SNP47976* (705.13Mb) and explained 7.03% phenotypic variation (Table 3.3, Fig. 3.3); whereas *Qfhb.hwwgr.7AS* flanked by *SNP45297* (15.88Mb) and

SNP45486 (35.33Mb) was mapped on 7AS and explained 9.47% of the phenotypic variation. The peak of *Qfhb.hwwgr.7AS* was different from the type II QTL mapped on the same chromosome arm in the population.

One QTL on the long arm of chromosome 6A, designated as *Qfhb.hwwgr.6AL,* was mapped between *SNP39362* and *SNP39459*. *Qfhb.hwwgr.6AL* was contributed by Everest and explained 9.31% of the phenotypic variation in the fall 2018 experiment with the peak SNP positioned at 614.42 Mb (Table 3.3, Fig. 3.4). Another QTL on 6BL, *Qfhb.hwwgr.6BL* flanked by *SNP43575* and *SNP43621* was significant in the spring 2019 experiment and explained 13.04% of the phenotypic variation. The resistance allele was contributed by Overland (Table 3.3, Fig. 3.3)

Four GBS-SNPs were mapped in the 7B QTL region, eight in the 7A QTL region, seven in the 1A QTL region, three in the 3B QTL region, five in the 4B QTL region, five in the 6A and 6B QTL region. To verify the accuracy of these SNPs and to fill the missing data in the QTL region, 27 KASP assays were designed from the corresponding GBS SNP carrying sequences. 11 KASP assays showed polymorphisms between parents and segregated in the population (Fig 3.4).

Discussion

FHB resistance consists of several types and each type is controlled by multiple minor QTLs and the expression of these QTLs is affected by environments (He et al., 2016). Repeatability of phenotypic data from multiple experiments is critical for accurate identification of the minor QTLs. Among five types of resistance, type I and type II are considered as the two major types of resistance and the other three types may be associated with the two types of resistance. However, relative to type II resistance, type I resistance has been poorly studied. Type

I FHB resistance is usually evaluated using spray inoculation, it is a big challenge to maintain appropriate and consistent disease pressures among different experiments because of significant variation in inoculum concentrations, spore viability, temperatures and moistures for FHB initiation among experiments. Type II resistance may also confound the expression of the Type I resistance (Liu et al., 2007). To minimize the environmental variation in the current study, we evaluated the population in the temperature-controlled greenhouse conditions and inoculated each plant by spraying the same amount of spore suspension at the same concentration in four repeated experiments. In each experiment, we inoculated five plants per line with two replications. Thus in the current study, four repeated experiments and replications in each experiment improved phenotyping accuracy (Bai & Shaner, 2004). Highly significant correlations of PSS ($P < 0.05$) were observed among the four experiments suggest reasonable repeatability of PSS among the four greenhouse experiments and should be useful for QTL analysis. To eliminate the confounding effects of type II resistance to type I, we counted the number of symptomatic spikelets per spike before the symptoms spread to other spikelets. The correlation coefficients were (0.21-0.42) slightly lower than these (0.28-0.52) for type II resistance (Chapter 2), indicating type I resistance is not as stable as type II resistance, in agreement with other studies (Petersen et al., 2016; Prat et al., 2017; Ren et al., 2019).

The significant, positive correlations between PH and PSS in three experiments (except for the spring 2020 experiment) suggest that taller plants had higher PSS than the short plants, agreeing with the findings of Clinesmith et al. (2019). The *Rht1* gene interferes with cell division and cell elongation and leads to more stiff plant tissues affecting spike compactness, flower morphology and anther extrusion (Buerstmayr & Buerstmayr, 2016, Miralles et al., 1998). Due to the mode of infection of the disease, FHB is highly modulated by a floral structure as narrow

flower opening and spike morphology related traits (Gilsinger et al., 2005). Yi et al. (2018) conducted a bivariate analysis to study the genetic relationship between FHB and PH and concluded that spike compactness has a strong influence on FHB after single floret infection. PH contributes to FHB severity for spray inoculation.

To date, only a limited number of germplasm has been reported to carry type I resistance. Previously, Fontana and Wangshuibai were frequently reported to carry QTLs for type I resistance, but these germplasms are unadapted to U.S. and have linkage drag with poor agronomic traits, therefore, the QTLs from these sources have not been deployed in modern wheat breeding programs. Identification of locally adapted sources of type I resistance will facilitate the utilization of type I resistance in breeding. In this study, we found that although Everest had much lower type II resistance than Overland (Chapter 2), it showed fewer initially infected spikelets in a spike than that in Overland after spray inoculation, suggesting Everest carries more QTLs for type I FHB resistance than Overland does. QTL analysis confirmed the result. Based on both greenhouse and field performances, Everest mainly contributed type I resistance and Overland contributed type II resistance.

QTL analysis identified seven QTLs for type I resistance on chromosomes 1A, 3B, 4B, 6A, 6B, 7A and 7B. Among them, five QTL are from Everest and only two from Overland. In this study, one QTL for type I was identified on each arm of chromosome 4. *Qfhb.hwwgr.4BL* links to *SNP28407* and *SNP28687* on 4BL and explained 14.89% of phenotypic variation for type I resistance, corroborating with the findings of Lin et al. (2006a) where 4B QTL for type I resistance explained 13.1% phenotypic variation. Several previous studies reported QTLs on 4BL for type I resistance (Giancaspro et al., 2016; Somers et al., 2003b; Xue et al., 2010) and for type II FHB resistance (Clinesmith et al., 2019; Lemes da Silva et al., 2019; Liu et al., 2007). Jia

et al. (2018b) reported *Fhb4* for type I resistance on 4BL from Wangshuibai and later Clinesmith et al. (2019) found the QTL on 4BL from Art considered as Fhb4 because the tightly linked marker *Xgwm149* showed polymorphism between parents and population. However, *Xgwm149* was monomorphic between the two parents used in the current study, and *Qfhb.hwwgr.4BL* (545.94 Mb) is at least 100 Mb away from the 4BL QTL in Art (445.05 Mb), it remains to be determined if *Qfhb.hwwgr.4BL is Fhb4*. Another QTL on 4BS is *Qfhb.hwwgr.4BS,* which is overlapped with reduced height genes *Rht-B1.* Liu et al. (2013) reported a similar QTL in winter wheat cultivars Becker and Mo 94-317 in field experiments. Later, a QTL for type II resistance was also reported near *Rht-B1* in durum wheat using an association mapping panel (Miedaner et al., 2017) and in *T. dicoccum* (Buerstmayr et al., 2012). A large effect QTL for type II FHB resistance was also reported in the current study on the same genomic region which is also associated with *Rht-B1* (Chapter 2). However, the genetic relationship between *Rht-B1* and *Qfhb.hwwgr.4BS* remains to be investigated.

Qfhb.hwwgr.7BL was flanked by *SNP51077* and *SNP51134* and explained 10.28% of the phenotypic variation of the mean greenhouse PSS data. Several QTLs for FHB severity have been reported on chromosome 7B in hexaploidy wheat (Szabó-Hevér et al., 2012) and tetraploid wheat, *T. durum* (Pirseyedi et al., 2019), and *T. dicoccum* (Buerstmayr et al., 2012; Ruan et al., 2012). The QTL *Qfhi.nau-7B* mapped for type I FHB resistance in the RIL population derived from Nanda × Wangshuibai and explained 10% phenotypic variation (Lin et al., 2006a), agreeing with our results. While Gupta et al. (2000) also reported the same genomic region associated with FHB resistance. Likewise, Schmolke et al. (2005) also reported the same genomic region associated with FHB resistance. Yang et al. (2005) reported QTL on 7BL for type II resistance which explained 8% phenotypic variation and maybe the same QTL mapped in the current study

since type II resistance has a confounding effect on type I resistance and it is hard to separate them in the field experiments.

Two minor QTLs for type I resistance were mapped on the short and long arm of chromosome 7A, respectively. *Qfhb.hwwgr.7AS* and *Qfhb.hwwgr.7AL* explained 9.47% and 7.03% of the phenotypic variations in the fall 2019 experiment and the mean PSS, respectively. In a similar study, Lin et al. (2006b) identified a QTL on 7B in the RIL population derived from Nanda × Wangshuibai overlapping the same genomic region mapped in the current study and explained 12.5% phenotypic variation. A number of studies reported QTLs for FHB severity on 7A chromosome of hexaploid wheat (Jayatilake et al., 2011a; Skinnes et al., 2010), and two QTLs for FHB incidence in tetraploid wheat *T. durum* linked with *barc165* and *wPt-3425* markers (Ruan et al., 2012) and they have a similar effect as the *Qfhb.hwwgr.7AS* in the current study. *Qfhb.hwwgr.7AS* was significant for type II resistance (Chapter 2) with the peak marker positioned at 72.20 Mb. *Qfhb.hwwgr.7AS* for type I resistance was mapped 15.88 to 35.33 Mb interval, suggesting that these two QTLs could be same but their peaks mapped on different regions due to large environmental variation.

Qfhb.hwwgr.1AL for type I resistance near *SNP1455* explained 9.26% of the phenotypic variation and significant for the fall 2019 experiment and the mean greenhouse data. Several cultivars have been reported to carry QTLs for FHB resistance on 1A chromosome, including Chinese line CJ9306 (Jiang et al., 2007b), Norwegian line NK93604 (Semagn et al., 2007), and Wangshuibai (Yu et al., 2008). Only one QTL for type I resistance from NC-Neuse has been mapped on 1AL with *IWA886* as the closest markers (Petersen et al., 2016, 2017). However, marker *IWA886* was far away (about 300 Mb) from *SNP1455* according to the Chinese Spring

reference genome sequence (https://wheat.pw.usda.gov/GG3/) (IWGSC, 2018), therefore, the 1AL QTL mapped in this study is most likely a novel QTL for type I resistance.

Qfhb.hwwgr.3BL is flanked by *SNP22576* and *SNP22678* on 3BL and was significant in the spring 2020 experiment and explained 9.38% of the phenotypic variation. To date, a number of studies reported QTLs for type II resistance on 3BS chromosome (Cuthbert et al., 2006; Jayatilake et al., 2011; Ruan et al., 2012b; Yang et al., 2005; Zhang et al., 2004), and 3BL (Bourdoncle & Ohm, 2003; Cai et al., 2016). *Qfhb.hwwgr.3BL* for FHB incidence has a similar effect as the QTL reported in tetraploid wheat (Pirseyedi et al., 2019) but has not been reported earlier in U.S winter wheat cultivar.

Qfhb.hwwgr.6AL on 6AL was significant in the fall 2018 experiment and explained 9.31% of the phenotypic variation for type I resistance. Other studies have also identified QTLs on 6A associated with multiple components of FHB resistance. QTLs for type II resistance on 6AL have been reported from European (Holzapfel et al., 2008; Schmolke et al., 2005; Srinivasachary et al., 2008) and U.S. wheat cultivars (Eckard et al., 2015). Recently, Petersen et al. (2016) mapped a QTL *Qfhb.nc.6A* on 6A chromosome which influences type I and type II resistance in the NC-Neuse and AGS 2000 population but this QTL spans the centromeric region of chromosome 6A in an interval of 81.8 to 138.5 cM while *Qfhb.hwwgr.6AL* mapped to the proximal end of the chromosome at 611.34 Mbs in the current study. Eckard et al. (2015) also reported a QTL on 6A from Overland, which is 10 cM away from *Qfhb.nc.6A* at 123.3 cM. Therefore, based on the marker information of already reported QTL, this *Qfhb.hwwgr.6AL* interval overlaps the already reported genomic locations. This study provides strong evidence that the *Qfhb.hwwgr.6AL* could be a common QTL across multiple wheat cultivars resistant to FHB.

Another QTL for type I resistance on 6BL was from Overland and explained 13.04% phenotypic variation in the spring 2019 experiment. Previously, several QTLs on 6BL have been reported for type II and type III resistance and explained 6.3% to 10.2% the phenotypic variation (Anderson et al., 2001a; Buerstmayr & Buerstmayr, 2015; Draeger et al., 2007). Basnet et al. (2012) reported a QTL on 6BL for disease index and FDK. The genetic relationship among these QTLs remains to be determined due to the lack of common markers among these QTLs.

In this study, we identified several QTLs for type I resistance from both parents using high throughput GBS-SNPs. However, a large amount of missing data for some genotypes is the major disadvantage associated with GBS, and also GBS is not suitable for marker-assisted selection of individual QTL in breeding. To fill up the missing points for important SNPs and develop breeder-friendly markers for breeding, we converted a set of GBS-SNPs in the major QTL regions into KASP assays. KASP markers not only filled the missing gaps created by GBS technique but also correct the GBS errors, and improved the map for QTL analysis. For 27 converted KASP assays from the GBS-SNPs in the QTL regions, 11 showed the same segregation pattern as corresponding GBS-SNPs. The SNP markers closest to the QTL can be used in marker-assisted breeding to pyramid multiple FHB resistance QTLs and increase the selection efficiency.

Conclusion

Genotyping-by-sequencing (GBS) based linkage analysis and QTL mapping of 178 RIL population derived from winter wheat cultivars Everest and Overland as a native source of resistance enabled mapping of seven type I FHB resistance QTLs. Several of these loci were governed by Everest and corresponded to known loci contributing to FHB resistance including 4BS, 4BL, 6A and 7B validating the reliability of GBS based linkage analysis to detect multiple

QTLs. The QTLs detected for type I resistance explained 8.82 to 14.89% phenotypic variance. The QTL on 4B chromosome was found to be associated with *Rht-B1* gene. The QTLs associated with type I FHB resistance showed less repeatability due to the inoculation method and large environmental effect. Our results indicated that Everest appeared to provide resistance against the initial infection of pathogen and can be exploited in North American winter wheat breeding programs. QTL mapping leads towards the identification of markers that can be directly applicable to marker-assisted selection. However future work is needed to understand the morphological trait association with FHB resistance and validation of the QTL positions.

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Figures and Tables

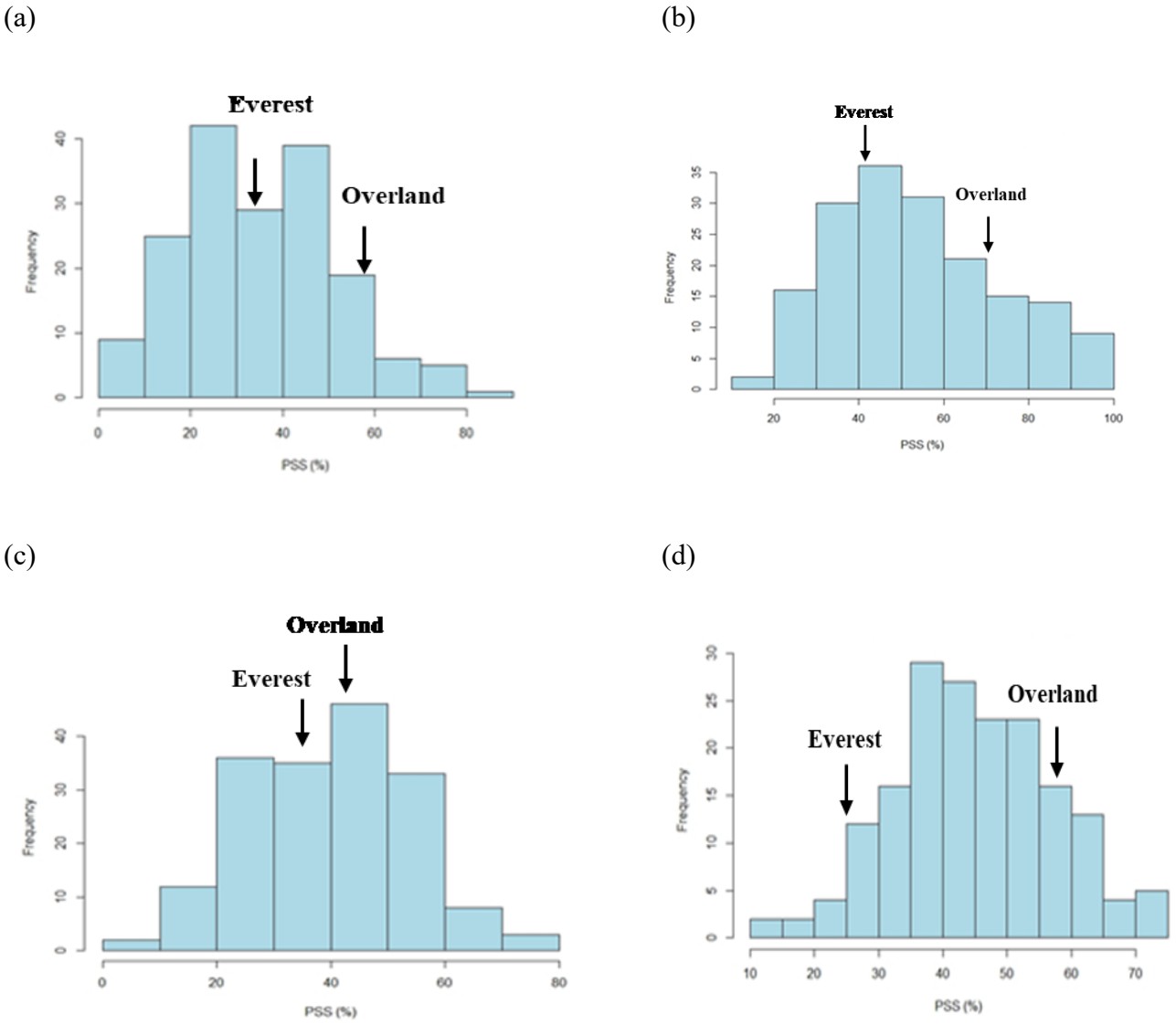


Figure 3.1 Frequency distribution of the mean percentage of symptomatic spikelets in a spike (PSS) for the recombinant inbred line (RIL) population derived from Overland × Everest evaluated in four greenhouse experiments (a) PSS frequency distribution of fall 2018; (b) PSS frequency distribution of spring 2019; (c) PSS frequency distribution of fall 2019; (d) PSS frequency distribution of spring 2020.

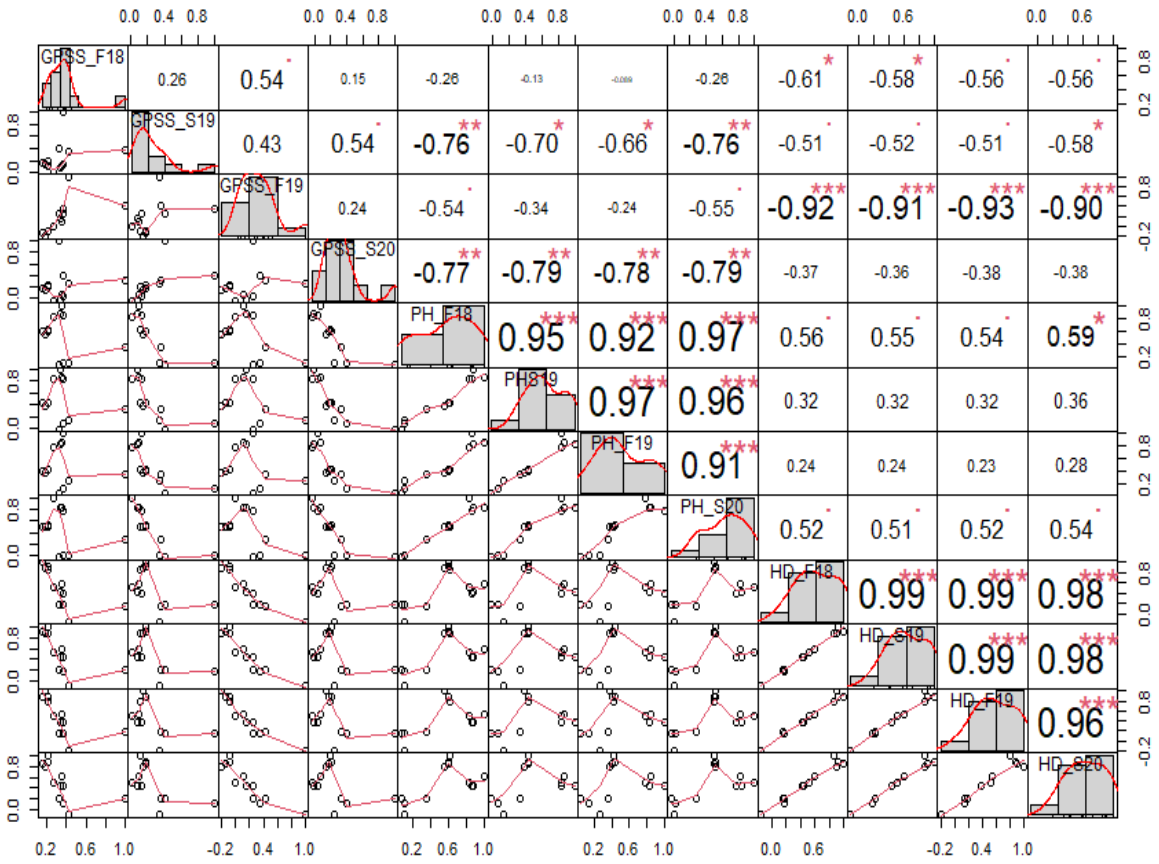
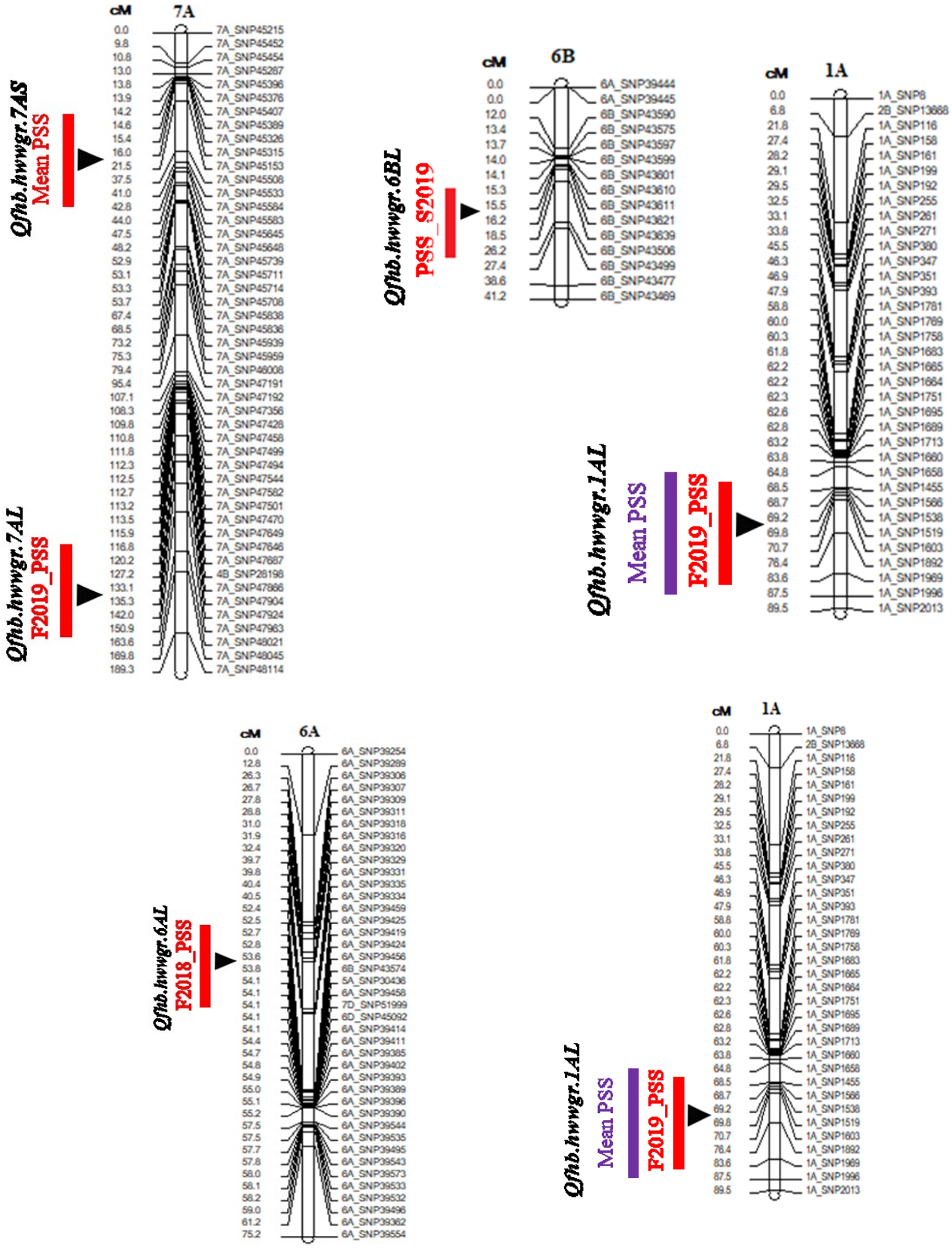


Figure 3.2 Pearson correlation coefficients among the percentage of symptomatic spikelets in a spike (PSS), plant height (PH) and heading date (HD) evaluated in the experiments conducted in fall 2018 (GPSS_F18), spring 2019 (GPSS_S19), fall 2019 (GPSS_F19) and spring 2020 (GPSS_S20). The distribution of each variable is shown on the diagonal. On the top right of the diagonal are values of correlations with the significance levels labeled as "***", "**", "*" and "." for *p*-values < 0.0001, 0.01, 0.05, and 0.1. The bottom left of the diagonal are the bivariate scatter plots with fitted lines.


Qfhb.hwwgr.7AS
Mean PSS
Qfhb.hwwgr.7AL
F2019_PSS
cM
7A
0.0 7A_SNP45215
9.8 7A_SNP45452
10.8 7A_SNP45454
13.0 7A_SNP45287
13.8 7A_SNP45396
13.9 7A_SNP45376
14.2 7A_SNP45407
14.6 7A_SNP45389
15.4 7A_SNP45326
16.0 7A_SNP45315
21.5 7A_SNP45153
37.5 7A_SNP45506
41.0 7A_SNP45533
42.8 7A_SNP45584
44.0 7A_SNP45583
47.5 7A_SNP45645
48.2 7A_SNP45648
52.9 7A_SNP45739
53.1 7A_SNP45711
53.3 7A_SNP45714
53.7 7A_SNP45708
67.4 7A_SNP45838
68.5 7A_SNP45836
73.2 7A_SNP45939
75.3 7A_SNP45959
79.4 7A_SNP46008
95.4 7A_SNP47191
107.1 7A_SNP47192
108.3 7A_SNP47356
109.8 7A_SNP47428
110.8 7A_SNP47458
111.8 7A_SNP47499
112.3 7A_SNP47494
112.5 7A_SNP47544
112.7 7A_SNP47582
113.2 7A_SNP47501
113.5 7A_SNP47470
115.9 7A_SNP47649
116.8 7A_SNP47646
120.2 7A_SNP47687
127.2 4B_SNP28198
133.1 7A_SNP47866
135.3 7A_SNP47904
142.0 7A_SNP47924
150.9 7A_SNP47963
163.6 7A_SNP48021
169.8 7A_SNP48045
189.3 7A_SNP48114
Qfhb.hwwgr.6BL
PSS_S2019
cM
6B
0.0 6A_SNP39444
0.0 6A_SNP39445
12.0 6B_SNP43590
13.4 6B_SNP43575
13.7 6B_SNP43597
14.0 6B_SNP43599
14.1 6B_SNP43601
15.3 6B_SNP43610
15.5 6B_SNP43611
16.2 6B_SNP43621
18.5 6B_SNP43639
26.2 6B_SNP43506
27.4 6B_SNP43499
38.6 6B_SNP43477
41.2 6B_SNP43469
Qfhb.hwwgr.1AL
Mean PSS
F2019_PSS
cM
1A
0.0 1A_SNP6
6.8 2B_SNP13668
21.8 1A_SNP116
27.4 1A_SNP158
28.2 1A_SNP161
29.1 1A_SNP199
29.5 1A_SNP192
32.5 1A_SNP255
33.1 1A_SNP261
33.8 1A_SNP271
45.5 1A_SNP380
46.3 1A_SNP347
46.9 1A_SNP351
47.9 1A_SNP393
58.8 1A_SNP1781
60.0 1A_SNP1769
60.3 1A_SNP1758
61.8 1A_SNP1683
62.2 1A_SNP1665
62.2 1A_SNP1664
62.3 1A_SNP1751
62.6 1A_SNP1695
62.8 1A_SNP1689
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64.8 1A_SNP1658
66.5 1A_SNP1455
66.7 1A_SNP1566
69.2 1A_SNP1538
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76.4 1A_SNP1892
83.6 1A_SNP1969
87.5 1A_SNP1996
89.5 1A_SNP2013
Qfhb.hwwgr.6AL
F2018_PSS
cM
6A
0.0 6A_SNP39254
12.8 6A_SNP39289
26.3 6A_SNP39306
26.7 6A_SNP39307
27.8 6A_SNP39309
28.8 6A_SNP39311
31.0 6A_SNP39318
31.9 6A_SNP39316
32.4 6A_SNP39320
39.7 6A_SNP39329
39.8 6A_SNP39331
40.4 6A_SNP39335
40.5 6A_SNP39334
52.4 6A_SNP39459
52.5 6A_SNP39425
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54.7 6A_SNP39385
54.8 6A_SNP39402
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55.2 6A_SNP39390
57.5 6A_SNP39544
57.5 6A_SNP39535
57.7 6A_SNP39495
57.8 6A_SNP39543
58.0 6A_SNP39573
58.1 6A_SNP39533
58.2 6A_SNP39532
59.0 6A_SNP39496
61.2 6A_SNP39382
75.2 6A_SNP39554
Qfhb.hwwgr.1AL
Mean PSS
F2019_PSS
cM
1A
0.0 1A_SNP6
6.8 2B_SNP13668
21.8 1A_SNP116
27.4 1A_SNP158
28.2 1A_SNP161
29.1 1A_SNP199
29.5 1A_SNP192
32.5 1A_SNP255
33.1 1A_SNP261
33.8 1A_SNP271
45.5 1A_SNP380
46.3 1A_SNP347
46.9 1A_SNP351
47.9 1A_SNP393
58.8 1A_SNP1781
60.0 1A_SNP1769
60.3 1A_SNP1758
61.8 1A_SNP1683
62.2 1A_SNP1665
62.2 1A_SNP1664
62.3 1A_SNP1751
62.6 1A_SNP1695
62.8 1A_SNP1689
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64.8 1A_SNP1658
66.5 1A_SNP1455
66.7 1A_SNP1566
69.2 1A_SNP1538
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89.5 1A_SNP2013

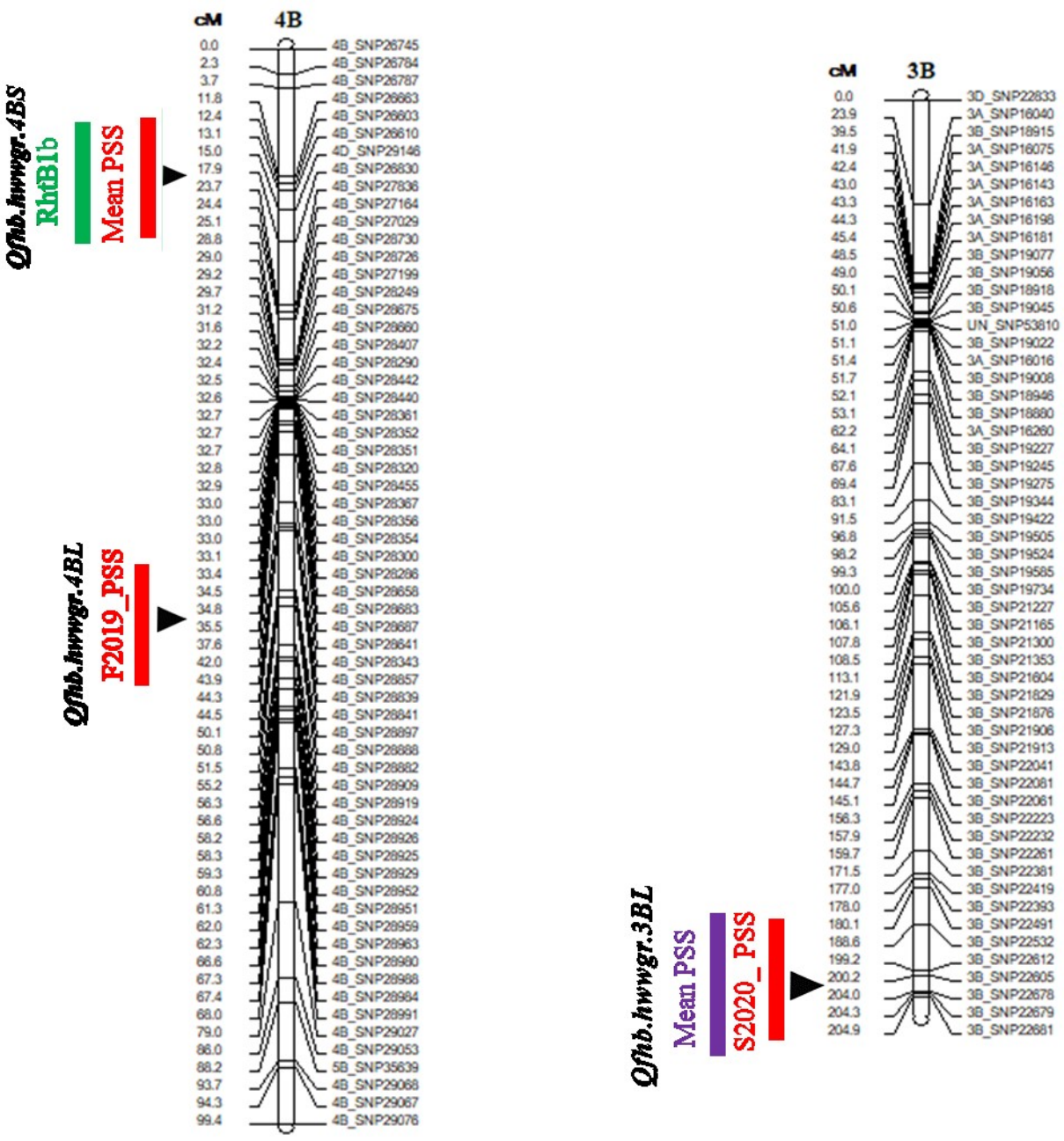
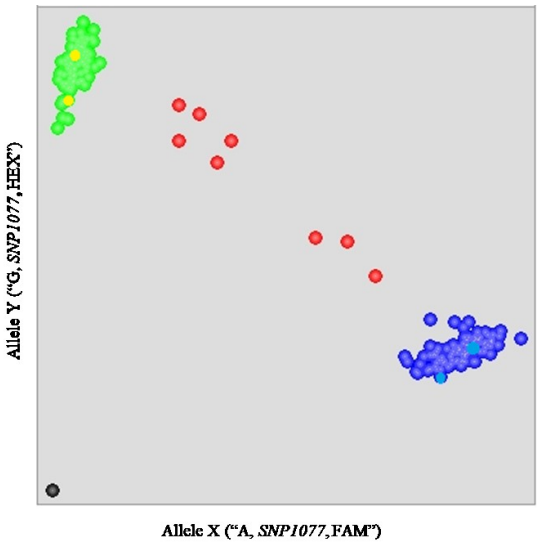
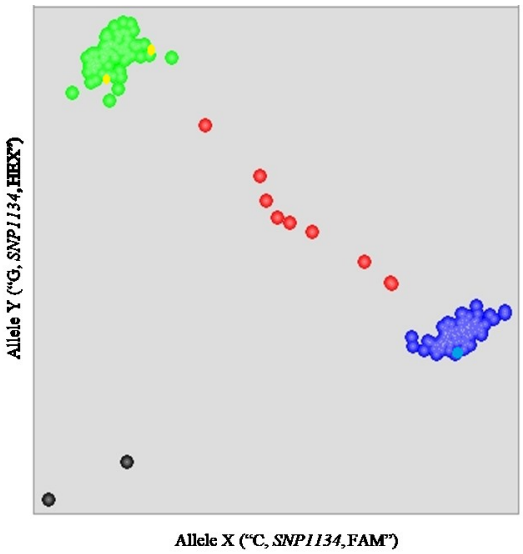


Figure 3.3 Chromosome maps of 1A, 3B, 4B, 6A, 6B, 7A and 7B carrying significant QTLs for FHB type I resistance in the RIL population of Everest × Overland.

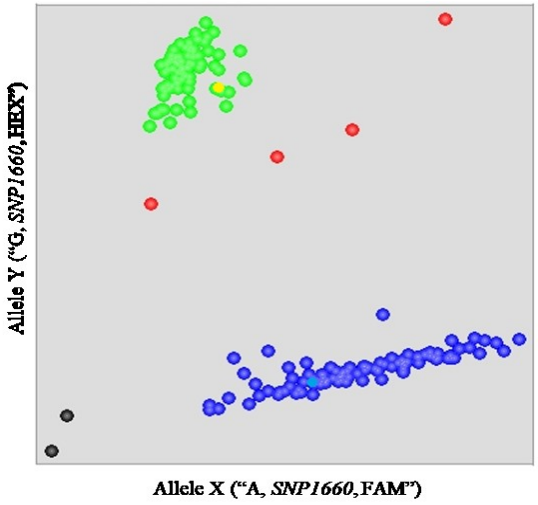
(1) KASP *SNP1077* on 7B



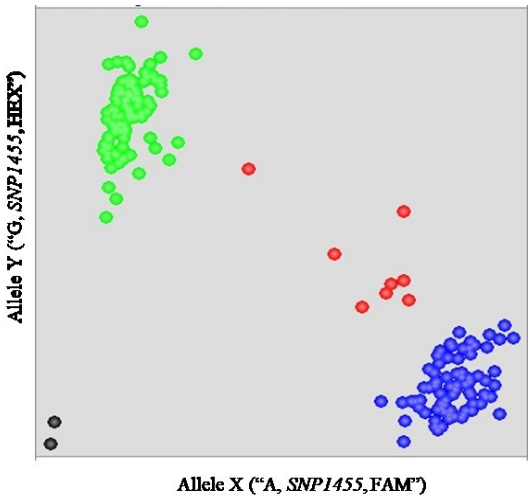
(2) KASP *SNP1134* on 7B



(3) KASP *SNP1660* on 1A

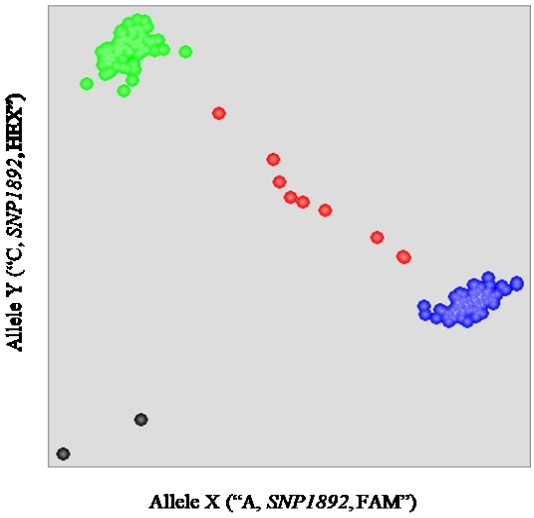


(4) KASP *SNP1660* on 1A

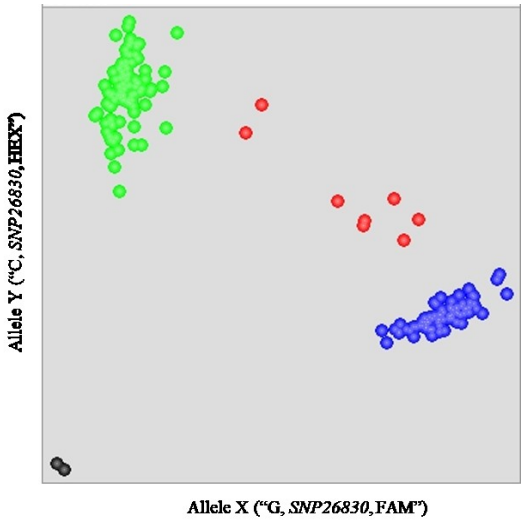


(5) KASP *SNP1892* on 1A

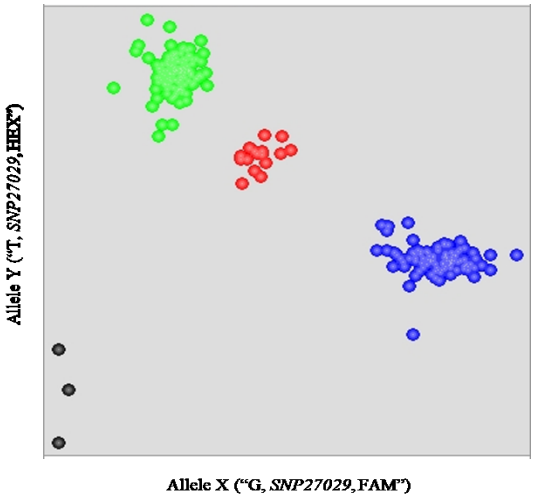
(6) KASP *SNP26830* on 4B



(7) KASP *SNP27029* on 4B



(8) KASP *SNP28343* on 4B



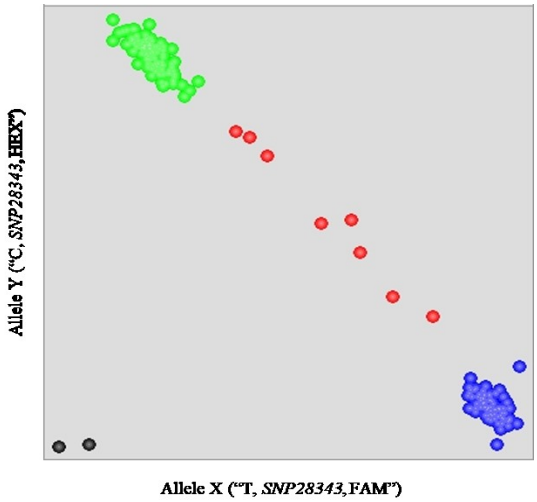


Figure 3.4 KASP marker segregation in the recombinant inbred population of Everest × Overland (1) KASP SNP1077 on 7B, (2) KASP SNP1134 on 7B, (3) KASP SNP1660 on 1A, (4) KASP SNP1455 on 1A, (5) KASP SNP1892 on 5A, (6) KASP SNP26830 on 4B, (7) KASP SNP27029 on 4B, and (8) KASP SNP28343 on 4B. Blue dots represent Everest alleles, green dots represent Overland alleles, red dots refer to heterozygotes and the black dots are ddH_2O.

Table 3.1 Analysis of variance (ANOVA) of the mean PSS data of recombinant inbred lines (RILs) evaluated in the four greenhouse experiments.

Source of variation	**DF**	**Type III SS**	**Mean Square**	**F-value**	***P*-value**	H^2
Experiment	3	6821.5584	2273.853	7.88	0.0373	0.77
Genotype	175	90391.42	516.5224	1.79	< 0.0001	
Genotype × Experiment	520	228077.82	438.6112	1.52	< 0.0001	
Replication (Experiment)	6	78.8874	13.1479	0.04	0.082	
PH (covariate)	1	851.252	851.252	2.95	0.0861	
Error	637	183812.72	288.56			
Total	1342					

DF: Degree of freedom; SS: Sum of square; H^2: Broad-sense heritability

Table 3.2 Adjusted means for percent symptomatic spikelets (PSS) per spike, plant height (PH) and heading date (HD) for Everest, Overland and the RIL population

Trait	Everest	Overland	RIL means	RIL range
			Fall2018	
PSS	29.57	58.38	43.29	4.42-83.02
PH	87.79	93.13	90.14	63-120.83
HD	103	125.5	104.68	92-134
			Spring2019	
PSS	40.08	70.26	53.83	16.44-97.87
PH	76.13	84.75	86.44	66.75-109
HD	99.1	111.1	103.6	95-124.5
			Fall2019	
PSS	38.10	39.84	39.54	8.01-74.44
PH	86.9	78.6	88.9	61.3-120.0
HD	102.3	122.9	108.1	94.7-139.3
			Spring2020	
PSS	23.41	57.34	45.0	13.68-73.21
PH	82.9	82.1	84.17	57.33-111.50
HD	95.1	116	99.9	88-122

Table 3.3 Quantitative trait loci (QTLs), their flanking markers, logarithm of odds (LOD) values and coefficients of determination (R^2) detected by composite interval maping (CIM) using the phenotypic data of RIL population derived from Everest × Overland collected in fall 2018, spring 2019, fall 2019 and spring 2020 greenhouse experiments.

Trait	**Experiments**	**QTLs**	**Position (cM)**	**Position (Mb)**	**LOD**	**Variance explained**	**Flanking markers**		**Resistance allele from**
							Left	Right	
PSS	Fall 2019	*Qfhb.hwwgr.1AL*	66.21	534.14 – 496.01	3.33	9.26	*SNP1624*	*SNP1892*	Everest
PSS	Mean greenhouses	*Qfhb.hwwgr.1AL*	66.21	534.14 – 496.01	3.17	8.82	*SNP1624*	*SNP1892*	Everest
PSS	Spring 2020	*Qfhb.hwwgr.3BL*	200.11	829.73 – 848.03	3.38	9.38	*SNP22576*	*SNP22678*	Everest
PSS	Fall 2019	*Qfhb.hwwgr.4BL*	34.81	545.94 – 604.79	5.53	14.89	*SNP28407*	*SNP28687*	Overland
PSS	Mean greenhouses	*Qfhb.hwwgr.4BS*	17.91	5.73 – 31.17	4.19	11.50	*SNP26663*	*SNP26830*	Overland
PSS	Fall 2018	*Qfhb.hwwgr.6AL*	54.11	605.72 – 614.82	3.35	9.31	*SNP39362*	*SNP39459*	Everest
PSS	Spring 2019	*Qfhb.hwwgr.6BL*	142.11	718.19-724.79	4.79	13.04	*SNP43575*	*SNP43621*	Overland
PSS	Fall 2019	*Qfhb.hwwgr.7AL*	78.61	684.57 – 705.13	2.50	7.03	*SNP47865*	*SNP47976*	Everest
PSS	Mean greenhouse	*Qfhb.hwwgr.7AS*	23.61	15.88 – 35.33	3.42	9.47	*SNP45297*	*SNP45486*	Everest
PSS	Mean greenhouse	*Qfhb.hwwgr.7BL*	41.71	704.06 – 710.70	3.72	10.28	*SNP51077*	*SNP51134*	Everest

Table 3.4 List of KASP primers developed from GBS SNPs markers

Primer name	Position	Primer Sequence (5'-3')
SNP1077FAM	7B	gaaggtgaccaagttcatgctCCAACTTTTTCCAGAGTCCATTCAA
SNP1077HEX	7B	gaaggtcggagtcaacggattCCAACTTTTTCCAGAGTCCATTCAG
SNP1077R	7B	GATGCGGTGCCATCATAGCT
SNP1134FAM	7B	gaaggtgaccaagttcatgctGTCCCATGAGACCTTCTCCAC
SNP1134HEX	7B	gaaggtcggagtcaacggattGTCCCATGAGACCTTCTCCAG
SNP1134R	7B	ATCTTCCGCCTGTACCAACG
SNP1660FAM	1A	gaaggtgaccaagttcatgctGTTGTCCACGAACAAACATATCAA
SNP1660HEX	1A	gaaggtcggagtcaacggattGTTGTCCACGAACAAACATATCAG
SNP1660R	1A	TGCACATATACTGTTTGTTTGACT
SNP1658FAM	1A	gaaggtgaccaagttcatgctGTCGCGCTGGAACTAGAAT
SNP1658HEX	1A	gaaggtcggagtcaacggattGTCGCGCTGGAACTAGAAG
SNP1658R	1A	GGGTTGGTAGTGTTGTCCAAT
SNP1624FAM	1A	gaaggtgaccaagttcatgctTGACTCCGTGTTATTTTTCCATCT
SNP1624HEX	1A	gaaggtcggagtcaacggattTGACTCCGTGTTATTTTTCCATCC
SNP1624R	1A	TGTTATGAATCTGCAGCCACC
SNP1455FAM	1A	gaaggtgaccaagttcatgctGTGGGACTGGGAGGAGGA
SNP1455HEX	1A	gaaggtcggagtcaacggattGTGGGACTGGGAGGAGGG
SNP1455R	1A	CAGAATACCTCTGCAGCAGA
SNP1892FAM	1A	gaaggtgaccaagttcatgctACAACTACCGCAAAACTTCTCTA
SNP1892HEX	1A	gaaggtcggagtcaacggattACAACTACCGCAAAACTTCTCTC
SNP1892R	1A	CCGAATCCGAATATGAGATCAGTTT
SNP26830FAM	4B	gaaggtgaccaagttcatgctCGACGCAACTTGCCACTG
SNP26830HEX	4B	gaaggtcggagtcaacggattCGACGCAACTTGCCACTC
SNP26830R	4B	TTCTTGCCGCCATGCTCT
SNP27029FAM	4B	gaaggtgaccaagttcatgctCGTAGCCTCTTCCGCCCG
SNP27029HEX	4B	gaaggtcggagtcaacggattCGTAGCCTCTTCCGCCCT

SNP27029R	4B	CTTCGACAGCCTGGAGGAAG
SNP28675FAM	4B	gaaggtgaccaagttcatgctCGTCGCTGGTGCCAGGAA
SNP28675HEX	4B	gaaggtcggagtcaacggattCGTCGCTGGTGCCAGGAG
SNP28675R	4B	GAGCGAAGATTCCGAACCCA
SNP28407FAM	4B	gaaggtgaccaagttcatgctCCTTTCAGCTTCCGTCGTTT
SNP28407HEX	4B	gaaggtcggagtcaacggattCCTTTCAGCTTCCGTCGTTC
SNP28407R	4B	GGGCACATCCAGAGCATACA
SNP28343FAM	4B	gaaggtgaccaagttcatgctAGCATGATGTAAACGCAAACAT
SNP28343HEX	4B	gaaggtcggagtcaacggattAGCATGATGTAAACGCAAACAC
SNP28343R	4B	CGCAGGTACGTCCAGTAGAT
SNP39362FAM	6A	gaaggtgaccaagttcatgctCCTTGCACGCAACATGATGT
SNP39362HEX	6A	gaaggtcggagtcaacggattCCTTGCACGCAACATGATGC
SNP39362R	6A	GCACACACCCATGTTTTGCT
SNP39496FAM	6A	gaaggtgaccaagttcatgctTCACCGTCGTAGCTTCCCT
SNP39496HEX	6A	gaaggtcggagtcaacggattTCACCGTCGTAGCTTCCCC
SNP39494R	6A	CAAGCGACAATGGCGAAGAG
SNP39544FAM	6A	gaaggtgaccaagttcatgctTGGGACAGTGACGGTTGTATATG
SNP39544HEX	6A	gaaggtcggagtcaacggattTGGGACAGTGACGGTTGTATATA
SNP39544R	6A	GGGCCTGCTCCTTTACTGTT
SNP39411FAM	6A	gaaggtgaccaagttcatgctATGTTCATTATTCCTTCCATTCCAA
SNP39411HEX	6A	gaaggtcggagtcaacggattATGTTCATTATTCCTTCCATTCCAT
SNP39411R	6A	GGCGTTATGGTTGTCTGAGATA
SNP39459FAM	6A	gaaggtgaccaagttcatgctCGGAAGAAGCTGACACCGTG
SNP39459HEX	6A	gaaggtcggagtcaacggattCGGAAGAAGCTGACACCGTT
SNP39459R	6A	CGATGAGTCTCCAAGCAGCA
SNP43590FAM	6B	gaaggtgaccaagttcatgctCTGCAGGGGCTATCGGGA
SNP43590HEX	6B	gaaggtcggagtcaacggattCTGCAGGGGCTATCGGGG
SNP43590R	6B	GCAACACGGTCAGCTTCA
SNP43575FAM	6B	gaaggtgaccaagttcatgctCTAGGCCCATGCTGCTTCAG
SNP53575HEX	6B	gaaggtcggagtcaacggattCTAGGCCCATGCTGCTTCAA

SNP43575R	6B	CCTCCTCGTGCTAACTGGTT
SNP43599FAM	6B	gaaggtgaccaagttcatgctTGTTATGATATGACCAGGAGAACAT
SNP43599HEX	6B	gaaggtcggagtcaacggattTGTTATGATATGACCAGGAGAACAC
SNP43599R	6B	AAAGCATCATCCTCCTGGGC
SNP43610FAM	6B	gaaggtgaccaagttcatgctCCCGTCATCAAGTACAGCCC
SNP43610HEX	6B	gaaggtcggagtcaacggattCCCGTCATCAAGTACAGCCT
SNP43610R	6B	GCTAGCTAGCTGATTTCTGCAG
SNP43621FAM	6B	gaaggtgaccaagttcatgctTGACGTGATGGAGTTGCCTC
SNP43621HEX	6B	gaaggtcggagtcaacggattTGACGTGATGGAGTTGCCTT
SNP43621R	6B	GGTTGATTTGTGGCATCGGC