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Impacts of *Fusarium* head blight (FHB)

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ABSTRACT: *Fusarium* head blight (FHB) is one of the most devastative diseases in wheat. Growing resistant cultivars is one of the most effective strategies to minimize the disease damage. The fungus produces a mycotoxin known as deoxynivalenol (DON) that poses a significant threat to the health of domestic animals and humans. Symptoms of FHB appear as "bleached heads" or heads with both green and bleached areas. Weather is an extremely important factor in the development of FHB, especially from flowering through kernel development. Moderate temperatures (20 to 30°C) coupled with prolonged periods of high humidity, and prolonged wet periods, are ideal conditions for FHB development. Use of FHB resistant cultivars has been found as one of the most effective solutions for reducing FHB damage.

***Keywords*:** FHB, *Fusarium*, resistant cultivars, reducing FHB damage.

INTRODUCTION

Fusarium head blight (FHB) resistance is an important objective of most wheat breeding programs. The disease is caused by *Fusarium graminearum*, *F. culmorum* and some other *Fusarium* species. Winter wheat (*Triticum aestivum* L.) is a main crop in Germany grown on 3.33 million ha in 2010 (DESTATIS 2010). FHB resistance is quantitatively inherited with a considerable genetic variation among breeding materials (Mesterhazy 1995; Miedaner 1997). Highly resistant varieties reduce the mycotoxin levels significantly (Miller 1985). To improve resistance levels and detect new sources of resistance tremendous efforts were made for identification, validation, and fine mapping of FHB resistance quantitative trait loci (QTL) in recent years. In a comprehensive meta-analysis Löffler (2009) compared 101 out of 176 FHB published resistance QTL and found that most of the chromosomes of hexaploid wheat were associated with FHB resistance. The most important and widely used QTL is *Fhb1* on chromosome 3BS, which explained 20 to 40 % of the phenotypic variance in the mapping populations (Anderson 2001; Bürstmayr 2003; Zhou 2002). A second important QTL is *Qfhs.ifa-5A*, which is located on chromosome 5A, and was detected in a cross between Remus and the Sumai3- derived CM-82036 (Bürstmayr 2003). This QTL explained 23 % of the phenotypic variation in the original mapping population. Further major resistance QTL with comparably smaller effects are *Fhb2* and *Fhb3* that were fine mapped on chromosomes 6BS and 7AL, respectively (Cuthbert 2007; Qi 2008). *Fhb1* is used widely in North America, for example in the US cultivar Alsen (Gamotin 2007; Mergoum 2007).

Fusarium head blight is of great concern in Brazil due to the high frequency of moderate to severe epidemics that led to significant yield losses since the early 1990s (Del Ponte , 2009). In such situations, yield loss is more likely when infections occur during the flowering stage (Goswami and Kistler, 2004), and economic losses related to rejection of grain contaminated with deoxinivalenol mycotoxin (DON) levels above maximum limits are due to a more complex interaction of biological and environmental factors during a larger window that extends from flowering up to grain filling (Cowger , 2009; Yoshida and Nakajima, 2010). Published mycotoxin data availabe on Brazilian wheat is scarce but recent reports showed the co-occurrence of DON and nivalenol (NIV) in commercial grain in levels that exceeded 2 ppm according to the year and region surveyed (Del Ponte , 2012).

The disease is best controlled with integrated practices such as resistant cultivars and fungicide applications (Hollingsworth , 2008; Paul , 2008; Wegulo , 2011). Although research efforts have effectively enhanced the resistance level to FHB in commercial varieties, host resistance cannot as yet be used solely to control FHB (Mesterhazy , 2011). Integrated use of resistant varieties with fungicides effectivelly reduced FHB levels and decreased mycotoxin levels when environmental conditions were favorable for the disease (Wegulo , 2011).

Triazoles, a class of fungicides in the demethylation-inhibiting (DMI) fungicide group that inhibits sterol biosynthesis, are the most efficient fungicides to supress FHB symptoms and reduce mycotoxin levels (Edwards , 2001; Paul , 2010). Tebuconazole, prothioconazole and metconazole, solely or in mixture of two triazoles, are the most commonly recommended fungicides for FHB control worldwide (Edwards , 2001; Pirgozliev , 2002; Paul , 2008). Strobilurin fungicides, although known to have relatively lower fungitoxicity to FHB pathogens, are commonly used in commercial mixtures together with triazoles to broaden the spectrum of protection against multiple leaf diseases of wheat and eventually lead to higher yield compared to triazole alone, especialy under high disease pressure (Blandino , 2006; Ransom and McMullen, 2008). Azoxystrobin are not usually recommended for FHB control because of the reports of DON increase, although exhibiting some effect in supressing FHB symptoms (Mesterházy , 2003). There is limited information in the literature for other strobilurins such as pyraclostrobin in FHB management (Bradley , 2011).

Fungicides targeting FHB are usually applied at the mid-flowering stage because extruded anthers are the primary infection sites, even though the window of vulnerability for infection can extend from flowering up to grain filling stages, depending on the variety (Del Ponte , 2007, Horevaj , 2011). In North Carolina, United States, fungicide residual period of 10 to 15 days from applications at early flowering were considered sufficient to protect the crop for up to two weeks (Cowger & Arrellano, 2010). In Brazil, anthesis can last from 10 to 20 days because of the asynchronous nature of heading and flowering depending on the environment (cloudy days extending flowering) and variety (Del Ponte , 2004). Such scenario combined with the well known difficulties in promoting good fungicide coverage of the infection sites, suggests that fungicide residual activity alone may be insufficient for effective FHB control when conditons remain favorable from flowering to grain filling stages, especially if the goal is to reduce mycotoxin levels.

Despite the importance and resurgence of FHB as one of the main disease of wheat in Brazil, published data on FHB control with fungicides is very limited in the country. Currently, fungicides are largely used in a preventive way to control foliar diseases and head blight of wheat, especially applying commercial mixtures of triazoles and strobilurins. The information is urgently critical given the inclusion of *Fusarium* mycotoxins in an updated list of regulated toxins in a broad range of cereal crops, including wheat and barley (Brasil, 2011).

Sumai 3 and Frontana, however, are inferior for grain yield, lodging tolerance, and other disease resistances and therefore, not yet exploited in European wheat varieties. Because of the very high yield level of 7 and 8 t ha-1 in Germany (DESTATIS 2010), European breeders are extremely cautious to use non-adapted germplasm and prefer resistance donors from their own programmes or European varieties. By rather intensive multi-step selection for FHB resistance accumulation of minor FHB resistance QTL in the European winter wheat pool has been achieved (Holzapfel 2008).

Fusarium head blight (FHB) is a destructive fungal disease of wheat and other cereals that has been reported worldwide (McMullen 1997; Parry 1995). Infection of wheat with FHB decreases yield and quality along with mycotoxin accumulation such as deoxynivalenol (DON) (Goswami and Kistler 2004; McMullen 1997; Osborne and Stein 2007; Parry 1995). These mycotoxins are hazardous to both human and animal health and Health Canada has imposed limits for non-staple and baby food in Canada (Canada 2012; Desjardins 2006; Pestka 2010).

Effects of Fusarium head curse

Fusarium head blight (FHB) is a devastative disease that can cause severe reduction in grain yield and quality in humid and semi-humid wheat growing regions worldwide (Bai and Shaner, 1994). When warm and wet weather coincides with anthesis and early kernel filling period, fungus can easily infect wheat plants and develop FHB. *Fusarium* infected florets often fail to produce kernels if infection occurs early or produce partially filled kernels that weight much less than normal ones. The infected kernels are light-weighted and very likely removed during threshing process, which significantly reduces harvested grain yield. FHB infection also lowers grain quality by reducing test weights and contaminating grain with mycotoxins such as Deoxynivalenol (DON) and zearalenone (De Wolf, 2003). Thus, FHB infection also causes severe impacts on the quality of cereals due to undeveloped kernels and mycotoxin accumulation. A significant positive relationship was observed between aggressiveness of the isolates and DON produced in the infected grain (Parry 1995). This suggests DON content might be a virulence component (Burlakoti , 2010; Bai , 2001; Desjardins , 1996). In addition, high DON content is also a food safety concern. Consumption of grain products contaminated with mycotoxins is detrimental to human and animal health. As low as 1 ppm of DON can cause significant reduction in feed intake and lower weight gain in animals, and 10 ppm DON can cause vomiting and feed refusal (De Wolf 2003). For human consumption, the acceptable DON levels in wheat have been set from 0.5 ppm to 2 ppm varied with countries. Thus, FHB not only reduces grain yield but also significantly lowers grain value in marketing, exporting, processing and feeding (Mcmullen, 1997).

FHB epidemics have been reported from many countries in Asia, Europe, North America and South America (Bai and shaner, 1994; Goswami and Kisler, 2004). In China, FHB has affected more than 7 million hectares of wheat and has caused more than 1 million tons of yield losses in 1990's (Bai and Shaner, 2004). In the U.S.A., direct value losses due to FHB from 1991 to 1997 in FHB-affected regions were estimated at $1.3 billion with the cumulative economy losses as high as $4.8 billion (Johnson, 1998). North and South Dakota and Minnesota suffered most from FHB outbreaks, accounted for about two-thirds of the total dollar losses due to all diseases (Nganje, 2004). In 1996, FHB has expanded to more than ten states in the central Great Plains areas of U.S.A. The disease continue to spread in Europe and South America, thus, FHB in wheat has become one of the most important crop diseases around the world.

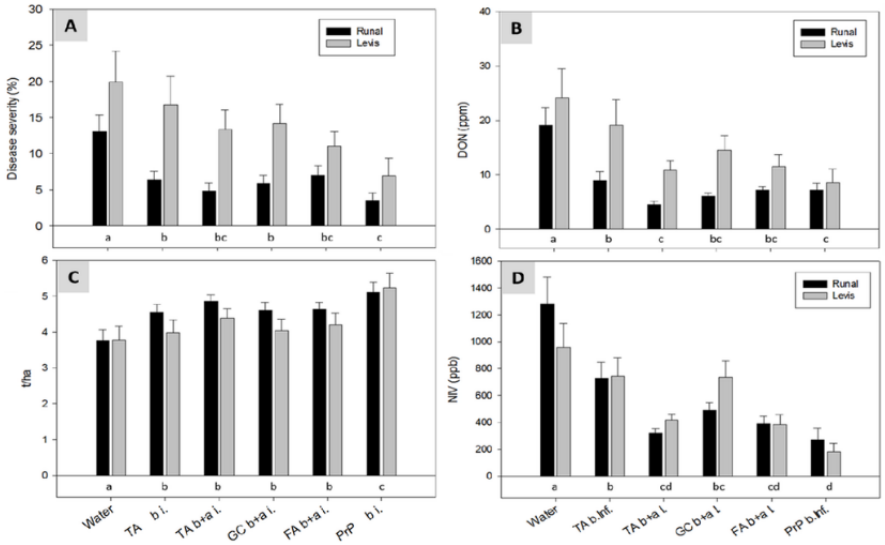


Figure 1. Field experiments with artificial inoculation: Effect of treatments with TA, GC, FA and PrP: (A) on the area of heads with FHB symptoms (disease severity); (B) on DON content; (C) on yield; and (D) on NIV content of the winter wheat cultivars "Runal" and "Levis" after artificial inoculation with a mixture of three FG and one FCr isolate/s. Bars with means of 16 values (four years and four replicates) and standard error of means. For treatments labeled by the same letter, mean values are statistically not different according to Tukey test ($p < 0.05$)

Causal life form, inoculums sources and scattering

About 19 *Fusarium* species can cause FHB (Liddell, 2003). Among major causal species, including *F. culmorum*, *F. graminearum, Microdochium nivale, M. majus*, *F. avenaceum,* and *F. poae* as (Xu and Nicholson, 2009), *F. graminearum* is the predominant FHB causal species in most areas of the world. Within *F. graminearum,* isolates may differ in virulence. For example, Chinese isolates may be more virulent than the isolates from U.S.A. (Bai , 2001; Lu , 2001). However, consistent specificity of cultivar resistance and pathogen virulence was not observed and proof evidence for race differentiation has not been found (Lu , 2001; Bai , 1996). Hence use of a mixture of different *F. graminearum* isolates as inoculums to screen FHB resistance is a common practice for inoculation (Bai , 1996, Zhou , 2002b).

Fusarium can survive in crop residues between host crop cycles. Ascospores, macroconidia, chlamydospores, and hyphal fragments can be all used as initial inoculums for infection (Bai and Shaner 2004, Dill-Macky 2003) with ascospores as the primary inocula during natural infection. However, *F. graminearum* conidia are often used as inoculums for experimental inoculation due to its easiness for production (Dill-Macky, 2003). In nature, *F. graminearum* forms perithecia to produce ascospores (*Gibberrella zeae* (Schw.) Petch). Very thick wall of perithecia can keep the fungus viable throughout the winter, which provides the pathogen a potential epidemiological advantage to overwinter (Xu and Nicholson, 2009). In late spring, matured perithecia forcibly discharge their ascospores into air when high moisture is available to initiate initial infection in wheat during wheat flowering (Webster and Weber, 2007). Thus, crop residuals from previous crop seasons are major sources of inoculum, and increased tillage may lower residue retention and the amount of overwintering inocula.

Wind blowing and rain splash are considered to be common mode of disease spread although birds and insects can also be the vectors of inoculum dispersion. Wind blows spores for long distance and rain splash can transfers them from crop debris on ground level to wheat heads (Frances , 2009). Upon reaching wheat head, ascospores will germinate and colonize in the wheat tissues of spikes to start infection.

FHB indications and disease pathway

Visible FHB infection symptom starts with tan or brown discoloration at the base of an inoculated floret (Wolf , 2003). A few days later, this light tan or bleached symptom will spread to entire inoculated spikelets. For resistant cultivars, the symptom could be limited to the inoculated spikelet without spread to adjacent uninoculated spikelets. However, for susceptible plants, the fungus invades rachis and spreads up and down to the entire spike if the weather is favorable for disease development. Infected florets on the spike can be infertile, or kernels become shriveled, bleached and chalky, also known as “tombstone”, if they are produced (Bai and Shaner, 1994).

During initial infection, conidia begin to germinate 6-12 h after the initial contact, and then germ tubes give rise to hyphae that will grow and extend on the interior surface to form dense mycelium networks (Xu and Nicholson, 2009). Hyphae grows through the interior surfaces of lemma, glume, and palea. After 24 to 36 h, hyphae may reach ovary. This infection process throughout floral parts is nonselective (Argyris , 2005). The fungus may enter the host tissue through stomata. Upon pathogen penetrating rachilla and rachis, disease will spread upward and downward on heads through vascular bundles and cortical parenchyma tissue (Goswami , 2004; Bushnell , 2003). Mycelium would clog the vascular tissue in the rachis that can cause head to premature and grains to be shriveled due to lacking of supply of water and nutrition (Xu and Nicholson, 2009). Other than that, stomata on glumes can be another entry point (Pritsch , 1999). Although anther can be the first part to be infected during FHB development. Then the disease spread horizontally from anthers to glumes, and vertically from anthers to rachis (Rinichich , 2000). However, the infection process normally occurred on the inner surfaces of lemma, glume, palea and rachis, not necessary through anthers (Xu and Nicholson, 2009). During colonization of wheat heads, the pathogen may secrete cell wall degradation enzymes that can decompose the host cells including cell wall, cytoplasm and cell organelles (Xu and Nicholson, 2009).

The pathogen hyphae may reach adjacent spikelets from initial infection site in two ways: through vascular bundles or stomata. When the weather is favorable, the fungal hyphae may penetrate rachilla and rachis, spread inter- or intra-cellularlly upward and downward on heads through vascular bundles and cortical parenchyma tissue to infect other neighboring spikelets (Goswami , 2004). Mycelium may also spread through outside glum from initially diseased spikelets to those uninfected spikelets (Ribichich , 2000). The hyphae can produce mycotoxins in 36 h after inoculation, which could be transferred upward and downward to the neighboring uninfected spikelets through xylem vessels and phloem sieve tubes (Kang and Buchenauer, 1999). Thus, given favorable environments and adequate time, toxins contamination between spikelets is unavoidable (Xu and Nicholson, 2009). Infection spread from spikes to spikes (secondary infection) is rare if any.

Disease symptoms are different between resistant and susceptible cultivars. In resistant plants, a dark-brown discoloration appears on an inoculated spikelet. In some cases, only a small dark brown spot could be observed on the lemma (Bai and Shanner, 1994). In FHB favorable conditions, the symptoms may spread to neighboring spikelets through vascular bundle, but it occurs very late, at least two weeks, and most spikelets in the spikes remains uninfected and still set normal seeds. However, in susceptible plants, entire inoculated spikes can be blighted with bleach discolorations on spikelets and dark brown rachis and culm. Infection spreads quickly to uninoculated spikes, usually in a week after inoculation and whole spike can be blighted in 7-10 days after inoculation. Thus resistant cultivars show much lower final disease severity than susceptible cultivars (Ribichich , 2000).

Components influencing FHB disease and improvement

Environmental factors have a significant impact on expression of FHB resistance as reflected by FHB incidence and severity (Parry 1995, Bai and Shanner, 1994). Warm temperature and high humidity coinciding with wheat anthesis favor FHB development. For greenhouse experiments, 20- 25 oC has been considered as a favorable temperature (Bai and Shaner, 1994, Brennan , 2005).

Table 1. FHB incidence (infected head/m^2) of the farm scale fungicide test across

Treatment	Petur		Miska		Kapos	
	Teejet XR	Turbo FloodJet	TeeJet XR	Turbo FloodJet	TeeJet XR	Turbo FloodJet
P125 + T125	0.37	0.07	2.13	1.00	1.37	0.93
T133	0.63	0.30	5.77	3.63	3.67	2.13
C300 + P120	4.37	0.70	6.23	4.47	4.70	2.70
EP125 + K125	1.30	0.97	7.33	5.37	5.00	3.60
EP84 + F250	2.43	1.00	8.67	6.17	6.97	3.10
Pro125 + CC40	1.70	1.53	8.60	6.87	6.23	4.80
AX200 + CC80	2.23	1.47	9.37	6.63	7.17	5.57
TET125	2.57	1.07	13.33	11.00	8.33	6.43
UTC *Fusarium* natural	5.87	5.87	15.73	15.73	10.40	10.40

Wet period during anthesis is also necessary for initial infection. The wet period that required for a high infection rate may vary with temperatures. It may take longer time for symptom development under low temperature (16 oC); and incubation period can be decreased with the increase of temperature (Rossi , 2001).

Table 2. Effect of fungicides against FHB in wheat. DON contamination in mg kg^{-1} on three cultiv

Fungicides	Zugoly (S)					Sámán (MS)					Bence (MR)			
a.i. g/ha	44Fg	12377Fg	12375Fc	12551Fc	Mean	44Fg	12377Fg	12375Fc	12551Fc	Mean	44Fg	12377Fg	12375Fc	12551Fc
P*125 + T125.	2.0	0.8	0.9	2.7	**1.6**	1.2	0.3	0.4	3.3	**1.3**	2.2	0.7	0.8	1.6
T250	4.4	2.2	1.4	2.7	**2.7**	2.6	1.4	0.7	6.6	**2.8**	5.5	1.1	2.4	3.8
T133	6.5	1.5	1.6	6.8	**4.1**	4.2	2.0	1.5	7.2	**3.7**	4.0	0.8	1.9	3.1
C300	8.9	5.6	6.5	13.3	**8.6**	9.1	1.5	3.3	9.5	**5.9**	5.9	1.8	3.8	7.3
UTC + *Fusarium*	21.6	12.5	32.7	27.1	**23.4**	26.0	25.4	13.2	24.7	**22.3**	13.6	5.1	7.1	13.9

Flower stage is the most susceptible stage for wheat to get infection by FHB pathogen although some cultivars may be susceptible at the beginning of caryopsis development (Bai and Shaner, 2004; Lu , 2001). Anthers contain a high level of chemicals such as choline and betaine that can facilitate the growth of *Fusarium* and function as initial infection points for fungus to enter spike tissues (Bai and Shanner, 1994). Given abundance of primary inoculums and optimum weather condition during anthesis stage, *F. graminearum* can cause severe FHB epidemics (Bai and Shaner, 1994). A positive relationship between width and duration of flower opening and incidence of FHB in wheat has been reported, because wider flower opening allows a larger area for *Fusarium* spores to enter a floret to initiate infection (Gilsinger , 2005)

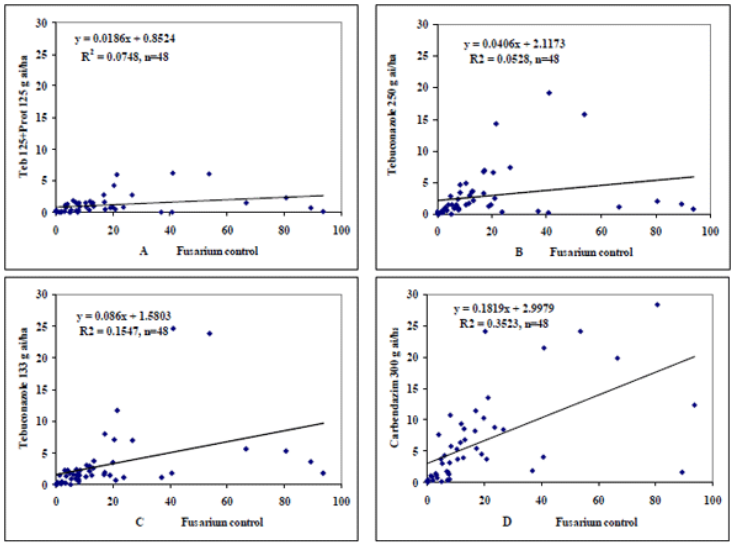


Figure 2. Stability of fungicides for controlling deoxynivalenol (DON) contamination, data: mg kg^{-1}. Data: four years *three cultivars* four isolates (=48 epidemic situations). Data of the Fusarium inoculated but not fungicide treated controls (X axis) were plotted against the data of the four fungicides tested (Y axis). (Commercial names: (A) Prosaro; (B) Folicur; (C) Falcon; (D) Kolfugo)

Agriculture practices such as crop rotation and crop management also have effects on FHB. Continuing to grow susceptible cultivars can increase initial inocula thus the FHB incidence (Dill-Macky and Jones, 2000). Wheat and non-host crop rotations may reduce the head blight incidence (Champeil , 2004). Limited soil tillage increases initial inoculum survival rate, and raises the FHB incidence, while ploughing (deep tillage) reduce inocula (McMullen 1997, Dill-Macky 2000, Teich 1989, Krebs 2000), to some extent, also modifies microclimate of the soil, and therefore reduces the development of *Fusarium.* Irrigation may also influence soil structure, and increases FHB frequency and severity. In addition to sowing date, wind speed, weeds, canopy (crop residue) density (Dill-Macky 2000) can all affect FHB pathogen development.

FHB resistance mechanism

Mechanisms of resistance to *Fusarium* in wheat are classified as morphological or physiological (Gilsinger , 2005). Morphological mechanisms refer to these crop traits that lead to unfavorable conditions for FHB to initiate infection, such as plant height, awnness, and degree of flower opening during flowering (Gilsinger , 2005). Plants with wide opening florets are more susceptible to FHB. Physiological mechanism involves biochemical pathways that produce chemicals to prohibit pathogens growth after initial infections. Resistance to FHB is considered to be non-race specific. Resistant wheat genotypes show similar reactions against different isolates of *F. graminearum* (Tóth , 2008).

FHB resistance can be phenotypically classified into five categories: type I, resistance to initial penetration of the pathogen; type II: resistance to disease spread within a spike; type III: resistance to kernel infection; type IV: tolerance and type V: resistance to accumulation of DON (Mesterhazy , 1999). Among them, type I, II and V are commonly accepted (Schroeder and Christensen, 1963, Miller, , 1985), but type V is usually referred as type III. Type I resistance is a major type of resistance in barley (Steffenson , 2003), type II is more stable resistance in wheat (Bai and Shaner, 2004; Kolb, 2001), while type III is evaluated for both barley and wheat. Type II resistance can be evaluated by point inoculation to a single floret of a spike and rating of symptom spread, within a spike. Percentage of symptom spread (PSS) within a spike usually used as the measurement for the level of type II resistance. Using this measurement, highly resistant cultivars may have as low as 5% PSS, while highly susceptible cultivars could reach 100% PSS. Moderate resistant and susceptible cultivars are in between (Bai , 1999). Significant correlations between the PSS and DON content were observed in single-point inoculation experiments (Bai , 2001; Yu , 2008b). All infected grains contain DON, even in a resistant cultivar. Contradicted results have been reported in different studies (Ma , 2006c; Mesterhazy , 1999). However, DON measurement is complicated, and inoculation time, harvesting and DON testing methods may significantly alter DON measurement. Early inoculation produces small highly-infected kernels and combine threshing may not be able to keep most of infected DON containing kernels for DON measurement, which all lead to underestimation of DON in susceptible cultivars (Ma , 2006c; Mesterhazy , 1999; Bai and Shaner, 2004).

Morphological variation is more likely to be associated with primary difference in initial infection, generally referred as Type I resistance, between cultivars, while difference in biochemical pathways is associated with symptom spread variation within a spike (Type II resistance). Although the processes of infection through anthers, floral bracts, rachilla and rachis are non-selective between resistant and susceptible wheat cultivars, biochemical responses to the infection are different between resistant and susceptible cultivars. Resistant wheat cultivars may produce substances that inhibit rapid growth of mycilium within a spike to prevent sudden desiccation on the spikelets above initial infected spikelets of a spike. Resistant wheat plants may form a physical barrier or accumulate chemical compounds such as phenols and triticens that are toxic to *F. graminearum* (Ribichich , 2000). These physical barriers include thickened cell wall and deposition of amorphous materials that can delay the disease progression. Trichothecenes may not be virulence factors for FHB initial infection of wheat floret (Bai , 2001; Jansen , 2005). When green fluorescence protein (GFP) labeled wild type and trichothecene knock-out mutant of *F. graminearum* strains were used to inoculate wheat, hyphae enters the cytosol of the epicarp cells in wheat, and leads to a cell death no matter inoculated with wild or mutant type of *F. graminearum* (Jansen , 2005). However, the action of DON may promote the spreading of *F.graminearum* in wheat from on infected spikelet to other ones by hyphae growth through rachis nodes (Bai , 2001; Jansen , 2005). However, DON may be detected in the infected kernels no matter the level of FHB resistance. Low DON content in resistant cultivars may be due to small amount of DON produced by the *F. graminearum*, DON degradation enzymes produced by wheat, and accumulation in spike tissues rather than kernels (Bai and Shaner, 2004). Some mapping studies detected that major resistance QTL for low FHB severity was associated to low DON content in wheat. (Jayatilake , 2011; Bai , 2000; Pena , 1999).

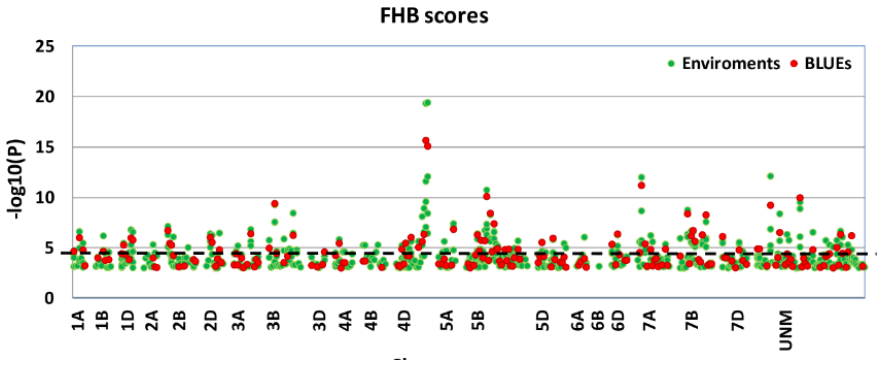
FHB pathogen development induces defense response genes during early infection of wheat spikes. The induced genes include pathogen resistant proteins PR-1, PR-2 (β-1,3-glucanase), PR-3 (chitinase), PR-4, and PR-5 (thaumatin-like protein) (Pritsch , 1999). Transcripts for the five defense-related genes are detected 6 to 12 h after inoculation, and peaked at 36 to 48 h after inoculation. More and earlier accumulation of PR-4 and PR-5 transcripts

was observed in resistant cultivars than susceptible cultivars (Bai and Shaner, 2004). A recent study reveals that a plant cytochrome P450 gene, CYP709CI, was associated with resistance in both spikes and seedlings (Li , 2010). Li and Yen (2008) indicated PR proteins might have nothing to do with FHB resistance, instead, Jasmonate (JA) and Ethylene (ET) mediated defense responses are important in wheat resistance to FHB based on observation that resistant wheat plants can have elevated JA or ET biosynthesis after inoculation (Li and Yen, 2008, Ding 2011). JA and ET are proposed to be important signaling pathways in plant defense response to FHB pathogen infection (Ding 2011). In JA pathway, lipoxygenase (LOX2) and chalcone synthase are up-regulated in resistant wheat plants but not in susceptible ones. ET can stimulate plant organs to senescence, which leads to cell wall dissolving and cell death (Li and Yen, 2008).

Biochemical composition in wheat may affect the resistance level of a cultivar (Brown and Brindle, 2007). Choline was considered to be the most influential metabolite. Other than that, betaine, the amino acids glutamine, glutamate and alanine, trans-aconitate and sucrose are correlated with FHB fungus (Brown and Brindle, 2007). Brown and Brindle (2007) reported a significant correlation between metabolic profiles and fungal hyphae growth. However, Engle (2004) did not find any significant correlation of fungal hyphae or spore growth associated with the metabolic levels. Thus, the biochemical mechanisms of FHB resistance are still debatable.

FHB resistant sources

Growing FHB resistant wheat cultivars is the most effective and economic method in FHB management (Bai and Shaner, 2004). Differences between cultivars in susceptibility to FHB were firstly reported in 19th century in US (Arthur , 1891). Since then, many breeders have attempted to find resistant sources. Although completely FHB immune cultivars have not been found (Fang , 1997), cultivars with various levels of resistance have been reported worldwide (Bai and Shaner, 2004). Most of highly resistant sources are from China and Japan (Bai , 2003b, Lu , 2001). In China, more than 30,000 *Triticum* accessions have been screened since 1980s (Fang , 1997), but only a small portion of them have good resistance, including Sumai3 and Wangshuibai (Bai and Shaner, 2004, Fang , 1997, Rudd 2001). Sumai3, as well as its derivatives such as 'Ning7840', are the mostly used FHB resistance source in breeding programs (Bai and Shaner, 1996, Kolb , 2001, Rudd , 2001), because of its high heritability, stable resistance across environments (Rudd , 2001). Chinese landrace Wangshuibai is another highly FHB resistant source that is unrelated to Sumai3 (Jia , 2005b; Lin , 2006; Yu , 2008c; Zhou , 2004). However, attempt to use this source as resistant parent in breeding was not successful due to its many undesired agronomic traits (Bai and Shanner, 2004). Some Japanese cultivars such as Shinchunaga, Nobeokabouzu and Nyu Bai also showed a high level of FHB resistance (Bai and Shanner, 2004; Ban, 2000), but they all have poor agronomic traits which are difficult to be separated from resistance using conventional breeding. Besides resistance from Asia, many germplasm from American also show a good level of resistance, e.g, Frontana and Encruzilhada from Brazil (Ban, 2001; Mesterhazy, 1995; Singh 1997), Ernie and Freedom from the U.S.A. (Rudd , 2001) were all reported to have a reasonable level of FHB resistance.



For FHB type II resistance in most of sources, additive effects play a major part in genetic effects, thus pyramiding of different genes in a wheat cultivar can upsurge FHB resistance in wheat (Bai and Shaner, 2000). In addition to highly resistant cultivars, moderately resistant cultivars are also good sources of breeding parents (Waldron , 1999). Moderate resistance may be easier to be achieved and can be easily to combine with desired agronomic traits. 'Alsen' was the first moderately resistant spring wheat cultivar released by NDSU (Mergoum , 2007). Alsen contains the *Fhb1* QTL for FHB resistance from Sumai3 with other QTL from native backgrounds as well as great agronomic performance, such as yield potential and end use quality (Mergoum , 2007). In US hard spring wheat region, 54% of

acreage was grown moderately resistant cultivars in 2011 (Connie, 2012). Some of the moderate resistant cultivars contain QTL from known FHB resistant parents, such as *Fhb1* from Sumai 3, while others may contain native resistant genes from local adapted parents. A cross between moderately resistant or moderately susceptible parents may generate highly resistant progenies. A good example is the well-known highly resistant cultivar Sumai3. It was developed by crossing two moderately susceptible parents, Funo and Taiwanmai (Bai , 2000). Thus, the resistance was derived by selection from transgressive segregation of resistance. One of advantages to use moderately resistant or moderately susceptible cultivars as a source of resistance is to allow a quick combination of genetically diverse resistant genes in more adapted genetic backgrounds. Some of the moderate resistant cultivars were mapped in breeding programs, such as Chokwang (Yang , 2005b), Frontana (Mardi , 2006) and Chinese spring (Grausgruber , 1999).

In addition, alien chromosome introgressions were used as an effective way to breed resistant cultivars. The same as homologous chromosome pairing during meiosis stage, the alien chromatin from monosomic alien addition line will recombine or translocate with the homologous to transfer resistant genes from alien sources to adapted common wheat (Cai , 2008). Such as *Fhb3* on chromosome 7A translocated from an alien species *leymus racemosus* (Qi , 2008). However, the biggest issues of this method are linkage drag and epistatic effects (Cai , 2005). Besides, genetic engineering provides novel approaches to develop transgenic wheat to enhance FHB resistance. Some examples are transgenic wheat expressing a barley class II chitinase (Shin , 2008), over-expressing the defense response genes α-1-purothionin, thaumatin-like protein1 and β-1.3-glucanase (Caroline , 2007), expression of an antibody fusion protein comprising a *Fusarium* –specific recombinant antibody derived from chicken and an antifungal peptide from *Aspergillus giganteus*, expression of Arabidopsis *NPR1* gene (Makandar , 2006).

Other FHB control measures

Besides cultivar resistance, weather is one of the key factors that determine epidemics of FHB. Wet and warm conditions facilitate all stages of development of the fungus (Champeil , 2004). Cultural practices that minimize initial inoculum can reduce the FHB epidemics. Crop rotation (Dill-Macky and Jones, 2000) and growing less susceptible crops (Champeil , 2004, McMullen , 1997, Dill-Macky , 2000) can be effective practices. Crop residues left on the soil surface are the major reservoir of inoculums (Bai and Shaner, 2004, Shaner, 2002). Traditional tillage practices left crop residues at the surface of soil after harvesting, which brings a high potential of FHB outbreak (Dill-Macky, 2008). Deep tillage can decrease the frequency of FHB outbreak (McMullen , 1997, Dill-Macky , 2000, Krebs , 2000) because ploughing buries inocula. Irrigation of wheat field may increase FHB infection, especially during anthesis, by providing moisture for spores development and release as well as establishing initial infection in wheat florets. Besides, early sowing or growing early maturity cultivars may escape heavy FHB infection in some locations (Champeil , 2004). Prior inoculation using other weak pathogen isolates (*F. culmorum, F. avenaceum, F. poae* etc.) has also been proposed to induce resistance by activating the host's defense response to provide cross protection to host plants (Diamond , 2003). Application of fungicides is still a major method for FHB control in commercial production. The effectiveness of fungicide application varies by active ingredient, method and date of application (Homdork , 2000). Using fungicides with specific active ingredients (triazoles containing tebuconazole) at a couple of days before a flowering season can be effective. However, difficulties in determination of an optimal time for fungicide application, high cost and lacking of fungicides with specific active ingredient are all problems involved with fungicide application (Bai and Shanner, 2004, Homdork , 2000). Suitable fungicide will reduce FHB severity and DON accumulation, especially in moderately resistant cultivars (Wegulo , 2011).

Inhibition of FHB through biological control agents is more environmental friendly compared with chemical control. Attempts to use biological control agents against FHB have been reported from several studies (Henkes , 2011, Petti , 2010, Khan and Doohan, 2009). But it is only a prosperous addition to current FHB manage-programs, and further studies are needed for a large-scale application in field. Therefore, no single solution is available for FHB control. A combination of cultural practices, chemical treatments, and use of resistant cultivars should be the best solution for FHB control.

Genetics of FHB Resistance and heritability

FHB resistance is a quantitative trait that is controlled by multiple QTL or genes, and the expression of the resistance is greatly affected by environments (Jia , 2005b; Bai , 2000; Parry , 1995). Some reports showed that many minor genes controlled the resistance (Chen , 1983; Liao and Yu, 1985), while others concluded that a few major genes plus some minor genes might control the resistance (Bai , 1990; Yao , 1997). The minimum number of genes for FHB resistance was estimated to be one to three (Bai , 2000). Additive effect is a major component of genetic variation for FHB resistance (Bai 2000) although epistasis might also play important role in some populations. Thus it is possible to pyramid several genes from different resistant sources to enhance wheat FHB resistance. It is

also possible to select FHB resistant lines from transgressive segregations that would be superior to the lines that they derived from (Yang , 2005a; Bai , 2000).

Molecular markers and genetic maps

Molecular marker is becoming the most popular tool in modern plant breeding. Conventional crop breeding mainly relies on direct selection of morphological variation in breeding populations. Earliest marker used in breeding was morphological markers (Stadler , 1929). The phenotypes associated with phenotypic variation such as pigment differences, vernalization habit and plant height etc. were used as indirect selection criteria. However, morphological markers were not extensively used due to its limitation on number of available markers (Worland 1987). Protein isozymes replaced morphological markers in 1970s (Market and Moller, 1959), but it has not been widely used in breeding (Tanksley, 1983). DNA markers are abundant, easy to be assayed and have become popular since 1980s.

Molecular markers can be classified into three categories: hybridization based, PCR-based and sequencing based. Hybridization- based markers include restriction fragment length polymorphism (RFLP; Botstein , 1980), fluorescent in situ hybridization (FISH), and microarray for marker detection. In 1990's, PCR-based markers are invented, which includes random amplified polymorphic DNA (RAPD; Williams , 1990), amplified fragment length polymorphism (AFLP; Vos , 1995), and simple sequence repeats (SSRs; Akkaya , 1992) etc. PCR-based markers quickly became popular because it needs small amount of DNA, without needing of radioisotopes, ability to amplify DNA from preserved tissues; high level of polymorphism that enables to generate molecular markers very fast; and ability to screen many genes simultaneously. The sequence-based markers include single nucleotide polymorphism (SNPs; Jordan and Humphries 1994), sequence tag sites (STSs) and expressed sequence tags (ESTs; Gupta , 1999). Microsatellite markers (SSRs) are 1-6bp tandem repeats, highly abundant, high polymorphic, and widely distributed throughout genomes. Many SSRs are locus specific and can be used as framework for linkage mapping (Gupta , 1999). SSR analysis requires small amount of DNA, fits high throughput analysis and has high reproducibility. Thus SSR is suitable for QTL mapping and validation (McCartney , 2004). Another type of DNA marker is STS (Olsen , 1989), a unique DNA fragment derived from known sequences (Farooq , 2002; Gupta , 1999). More recently, the newest type marker is SNP (Jordan and Humphries 1994), which can detect individual nucleotide variation, and is suitable for high-throughput marker detection, thus it should be unlimited and is the future of markers for genetics research and breeding.

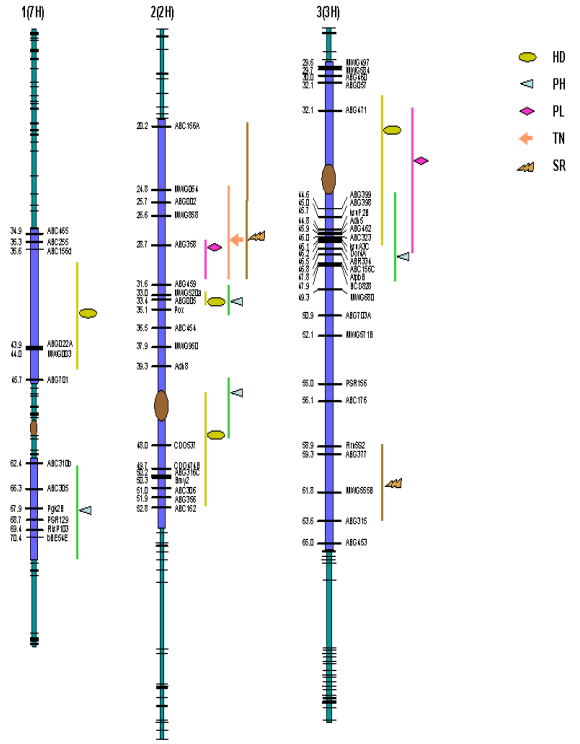


Figure 3. Frequency distribution plots for heading days (HD), plant height (PH), peduncle length (PL), tiller number (TN), and stripe rust (SR) in RILs

All types of molecular markers discussed above have been used for QTL mapping of FHB resistance (Anderson , 2001; Buerstmayr , 2002; Waldron , 1999; Mardi , 2005; Bai , 2003a; Bai , 1999; Zhang , 2004; Sun , 2003; Ban , 2000; Buerstmayr , 2011; Somers , 2003; Chen 2007; Liu , 2007; Steiner , 2004; Ma , 2006c; Cuthbert , 2006; Liu and Anderson, 2003; Bernardo , 2012; Yu , 2008a). These markers have been used to construct genetic linkage maps to locate QTL positions for FHB resistance. Relative positions of genetic markers are arranged in linkage maps according to recombination frequency (RF) among markers. According to marker trait relationship in the map, QTL are located to certain map locations.

Because each map is developed using different populations, marker positions in different maps from different populations may be different. A map combining all map information from several different populations (a consensus map) may be more useful reference for determining consensus chromosome locations of markers and QTL. In wheat, the first genetic linkage map with 279 SSR markers were constructed in 1990s' (Roder 1998). In 2004, Somers used 4 populations and developed a wheat consensus map with 1,235 SSR markers (Somers 2004). This consensus map provides framework for mapping QTL for traits of interest in different mapping populations and for map based cloning of different genes/QTL (Somers 2004).

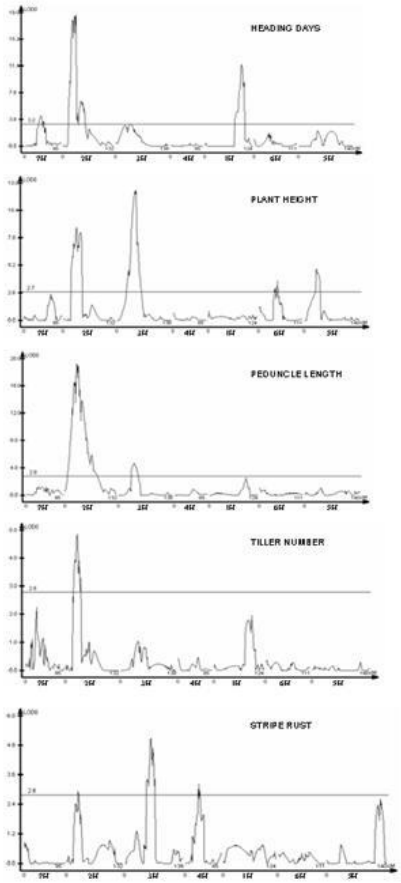


Figure 4. QTL likelihood maps for various traits obtained from composite interval mapping (CIM) analysis indicating LOD score along the ordinate while genetic map (all chromosomes together) along the abscissa. The respective threshold LOD estimated by 1000 permutations at 0.05 significance, are represented as horizontal line

QTL for FHB resistance

Quantitative trait locus (QTL) mapping using DNA marker is a highly effective approach for studying quantitative traits (Young, 1996; Tanksley, 1993). QTL mapping is used to dissect complicated traits, locate QTL underlining these traits in a genetic map, and determine their effects and interactions between QTL (Kearsey, 1998). Quantitative traits may be conditioned by several individual QTL that each may segregate in a Mendelian manner and affected

by environments. By fitting phenotypic data with predicted genetic models, it is possible to estimate gene number, genotype by environment effect and heritability.

QTL mapping was firstly proposed by Sax in 1923 (Sax, 1923), and elaborated by Thoday later (Thoday, 1961). The basic concept of QTL mapping is to test the association of genomic region with the quantitative traits of interest (Mohan , 1997; Young, 1996). If a marker tightly linked to a QTL, the QTL will co-segregate with the marker. If a recombinant inbred population is separated into two groups based on two alleles of the marker, significant difference ($P<0.05$) in the trait values between groups indicates that the DNA marker is more likely linked to the QTL (Collard , 2005; Young, 1996). QTL mapping has been widely used to develop markers for marker-assisted selection (MAS) and map-base cloning (Buerstmayr , 2009; Mohan , 1997; Collard , 2005). Several factors may affect accuracy of QTL mapping. First, high-density map may provide more power for QTL detection; second, minor QTL may not be detectable especially when heritability is low; third, low heritability also results in a large confidence interval of QTL (Hyne , 1995); fourth, it is difficult to discriminate two QTL that are not far apart on the same chromosome (Kearsey, 1998; Young, 1996).

QTL mapping starts with mapping population. For mapping FHB resistance QTL, the parents for mapping population should show significant contrast in FHB resistance (Liu, 1998; Collard , 2005). Population sizes used for preliminary genetic mapping construction have been reported from 70 to 250 lines (Mohan , 1997), however, larger populations are required for high-resolution QTL mapping (Collard , 2005). Several types of populations have been utilized in QTL mapping experiments. F2, backcross (BC) (Buerstmayr , 1999), and recombinant inbred lines (RILs) (Waldron , 1999; Yu , 2008c), double haploid (DH) (Chen , 2006; Jia , 2005b; Yang , 2005b) and chromosome recombinant inbred lines (CRILs; Garvin , 2009; Ma , 2006a) can all be used for QTL mapping. RIL has been more commonly used population type for FHB mapping because the same genotypes can be repeatedly evaluated for FHB in different years or locations (Collard , 2005), although its construction takes several years.

Several methods have been used in routine QTL mapping; single marker analysis (SMA), simple interval mapping (SIM), composite interval mapping (CIM), and multiple interval mapping (MIM) (Tanksley, 1993). SMA (single locus regression) calculates phenotypic difference between two allelic groups of each marker. A t-test at each marker can be used to identify significant trait difference between two allelic groups. If the difference is significant, the marker is assumed to link to the QTL. At the same time, genome wide type I error has to be taken into account (Lander and Botstein 1989). However, SMA cannot determine QTL locations in a map. SIM, firstly described by Lander and Botstein (1989) can determine map location (or marker interval) of a QTL. SIM, the earlier version of interval mapping, evaluates the association between the phenotypic values and a target QTL between multiple pairs of adjacent markers. The QTL genotype is estimated by flanking marker genotypes as well as marker-QTL distance (Manly and Olson, 1999). Compared with SMA, SIM improves the power of detecting QTL to some extent; and QTL locations can be better resolved in SIM. However, the disadvantages of SIM are that it cannot detect a QTL outside of the defined interval , it can not distinguish two linked QTL if they located in the same or close marker intervals (Manly and Olsen, 1999), and QTL and background effects, and only two markers are tested at each time (Zeng et al, 1993; 1994). Composite interval mapping (CIM) can solve some of the problems SIM has. It can detect hypothetic QTL by setting a certain number of markers as window size, while utilizing the background markers as cofactors to control background noises (Manly and Olsen, 1999; Jansen, 1993; Zeng , 1993, 1994). This refined mapping model enable us to distinguish one target QTL from the ones in adjacent intervals and thus is more efficient and precise. MIM is another method that utilizes multiple marker intervals simultaneously. It can also be used to estimate epistasis between QTL, genotypic values of individuals, and heritability of quantitative traits (Chen , 1999). However, the identified QTL still need to be further validated.

To detect significant QTL, a t-statistic (Zeng , 1994), the logarithmic of odds (LOD , Lander and Botstein, 1989) and the likelihood ratio statistics (LRS, Haley and Knott 1992) are commonly used. LOD score is a ratio between the 10-base-logarithm of likelihood of having linked QTL to the 10-base-logarithm of not having the linked QTL. LOD scores and LRS are convertible to each other with LRS=4.6×LOD (Liu, 1998) and both are commonly used in interval mapping to identify the most likely position of a QTL in a linkage map. If the peak or the highest point exceeds a LOD/LRS threshold the QTL is claimed to be a significant. Significant threshold of LOD or LRS is usually determined by 1000 random permutations (Churchill and Doerge, 1994). The permutation test breaks all the marker-trait associations, and shuffles all the phenotypic data while marker data remain constant. Permutation is performed to calculate the level of false positive QTL. Parameters such as LOD or LRS generated from the permutation on the random data form a distribution of LRS or LOD with H_0 = no QTL associated with the markers. This process is then repeated one thousand times to determine significant threshold at a given confidence level, usually 95% (Manly and Olsen, 1999). A conservative threshold at LOD of 3.0 is also used for claiming significant QTL (Collard , 2005). A QTL can be located in an interval between two markers called ‘flanking’ markers. This QTL can be ‘major’ or ‘minor’ QTL depends on the proportion of the phenotypic variation explained by the QTL (R2). A major QTL usually explains

a large portion of phenotypic variation (>10%) while a minor QTL accounts for a relatively small portion of phenotypic variation (<10%). Empirically, major QTL are more stable across environments and locations, especially for disease resistance QTL (Li , 2001; Collard , 2005).

In QTL mapping, several factors may affect the power of QTL detection. A high-density map is preferred, especially in the QTL region. A marker space less than 10 cM may have little effect on mapping result, however, marker space more than 20 cM may reduce the power of QTL detection to some extent (Collard , 2005). Accuracy and reproducibility of phenotypic data are important in mapping studies (Cuthbert , 2006; Kolb , 2001). For FHB resistance, environmental effect may have a huge influence on the trait scoring. Thus it is very difficult to get reproducible FHB data over different experiments, especially type I resistance because the variation can be mainly accounted by environments (Bai and Shanner, 1994). The same QTL may express various levels of resistance under different environments, and minor QTL is more sensitive to environments than a major QTL. Thus, it is necessary that QTL experiments should be done with replications across multiple locations and/or over times (George , 2003; Kolb , 2001, Collard , 2005; Haley and Anderson, 1997). Population size is another important factor influencing the power of detection. Larger population size can increase power in QTL detection (Darvasi , 1993). Quality of genotypic data may also important. Too many missing marker scores may alter the marker orders and distances in a linkage map (Hackett, 2003). QTL can be validated using different mapping populations (Lander and Kruglyak, 1995) and near isogenic lines (NIL) that have uniform genetic background but contrasting in the QTL of interest (Pumphrey , 2007).

QTL for FHB resistance have been mapped on about 50 wheat cultivars covering all 21 wheat chromosomes (Table 1.1; Liu , 2009). Among them *Fhb1* on 3BS shows the largest effect on type II and type III resistance (Bai and Shaner, 2004). This major QTL was validated later by other studies (Anderson , 2001; Chen , 2006). The QTL on chromosome 3A, 5AS, 7A, 1B, 3BS, 4B,5B, 6BS and 2D have been mapped in more than two populations in previous studies (Liu , 2009). Five of them were formally named as *Fhb1* on chromosome 3BS, *Fhb2* on chromosome 6B (Anderson , 2001), *Fhb3* on Chromosome 7AS from a Wheat-Leymus introgression line (Qi , 2008), *Fhb4* on Chromosome 4B (Xue , 2010), and *Fhb5* on Chromosome 5A (Xue , 2011). However, only the Sumai3-derived *Fhb1* is now extensively used in breeding programs due to its stable effect on type II and type III resistance across different genetic backgrounds (Bai , 1999; Anderson , 2001; Buerstmayr , 2003; Shen , 2003; Bourdoncle and Ohm, 2003; Yang , 2005; Chen , 2006; Jiang , 2007ab). The QTL on 3BS has been reported in more than 30 studies in which Sumai 3 was the major source of resistance. It was also reported in cultivars that are not related to Sumai 3 such as Chinese landrace Wangshuibai (Lin 2004; Zhou 2004) and Japanese landrace Nyu Bai (Somers 2003; Cuthbert 2006) etc. A wide range of R2 –values has even been reported for *Fhb1*, ranging from 6% to 60% for type II resistance (Bai, et al, 1999, Waldron , 1999, Anderson , 2001, Buerstmayr , 2002, Shen , 2003, Bourdoncle and Ohm 2003, Somers , 2003, Chen , 2006, Jiang , 2007ab, Lin 2004, Zhang , 2004, Ma , 2006c, Yu , 2008b, Yang , 2005b). Besides, QTL on 6B (*Fhb2*; Anderson , 2001; Yang , 2003, Shen , 2003; Cuthbert , 2007) flanked by *Xgwm133* and *Xgwm644* was another major QTL that explained a wide range of phenotypic variation for disease spread from 4.4% (Shen , 2003) to 23% (Somers , 2006). The QTL on 7AS was mapped close to *Xgwm276*, explained 3% in Wangshuibai (Zhou , 2004)., while according to Recently, a novel QTL on 7A was mapped close to *Xwmc17*, explained 41% of FHB type II resistance (Jayatilake , 2011). The QTL on 5A explained 4% (Li , 2011), 7% (Li , 2012; Zhang , 2012) and 11% (Buerstmayr , 2002) of phenotypic variation and was related to type I resistance. QTL for FHB resistance were mapped on almost all wheat chromosomes even on 7D (Li , 2011, Table 1.1) where QTL was not detected before (Buerstmayr , 2009). The QTL on 7D was peaked at *Xwmc121*, flanked by the SSR markers *Xcfd46* and *Xwmc702*, and explained up to 22.6% disease spread variations (Li , 2011).

The inconsistent numbers and locations of FHB resistant genes reported in different studies could be due to (Kolb et al, 2001): (I) some genes may segregate in some crosses but not the others; (II) different genetic backgrounds and parents used for population development; (III) some resistant alleles from susceptible parents; (IV) heterogeneous source of resistance; (V) different *Fusarium* species or different isolates of *F. graminearum* as inoculums; (VI) different types of resistance phenotyped at different environments. In addition, population size and degree of map saturation are also affect QTL detection.

Breeding strategies

To breed FHB resistant wheat, breeders desire to combine a high level of FHB resistance with favorable agronomic traits, by accumulating different genes to upsurge FHB resistance because additive effect is a major component of resistance (Bai and Shaner, 2000). Thus pyramiding FHB resistance genes from diverse gene pools and removal of susceptible genes is an effective method to upsurge the level of resistance (Ma , 2006b; Rudd , 2001). Since major QTL have a stable effect on type II resistance, transferring *Fhb1* into commercial susceptible or moderately susceptible cultivars may significantly improve the FHB resistance of cultivars in commercial production

(Kolb , 2001). This can be achieved by backcrossing and marker-assisted selection. However many highly FHB resistant sources usually have many unadapted agronomic traits. These sources are mainly landrace such as 'Wangshuibai' or un-adapted breeding lines such as Ning 7840 (Bai and Shanner, 2000). Thus, using backcross to move resistance QTL into adapted backgrounds to create middle parents may be critical for successful use of the QTL from these sources.

Owing to extensive breeding effort to improve wheat FHB resistance, many elite breeding lines and cultivars have moderate resistance or moderate susceptibility. They may either contain a few major QTL or some minor QTL for FHB resistance. Some of them are native resistance genes that may be different from the Asian resistance sources. These cultivars can be directly used in commercial production to reduce the losses caused by FHB epidemics. They also can serve breeding parents for transferring these major QTL from Asian sources for better resistance. For example, In the U.S. spring wheat cultivars with moderately resistant were grown in more than half of the total acreage in 2011 (Connie, 2012). Some of the cultivars contain QTL from Asian sources, such as *Fhb1*, others may contain native resistance QTL.

Although Sumai3 is the major source of FHB resistance in wheat breeding programs worldwide, some other resistance sources should also be explored. Combining QTL that originated from different geographical regions can broaden the genetic diversity (Bai , 2003). For example, the US winter wheat cultivars Heyne, Ernie and Freedom may have different QTL from Asian sources (Bai and Shanner, 2000). An example of pyramided FHB resistance line has been reported. In that case, WSY was developed by pyramiding QTL from a 'three way crosses' among Sumai3, Wangshuibai and Nobeokabouzu (Shi , 2008). In the U.S.A., Some commercial soft wheat cultivars harbor *FHB1* have been released, including Pioneer Brands 25R18, 25R42, and 25R51, most of them are developed by marker-assisted backcross (Brown-Guedira , 2008). In addition to incorporating *Fhb*1 as well as other minor QTL from Asia into new wheat cultivars, breeders in US also use native resistance in breeding, such as Heyne, Ernie and Freedom. These winter wheat cultivars show good FHB resistance, but do not have *Fhb1* (Bai and Shaner, 2004). Truman and Bess developed at the University of Missouri, have better FHB resistance and overall performance on yield, test weight under disease pressure (Mckendry, 2008). Besides, many US hard red spring wheat cultivars also have FHB resistance, such as Bacup developed in Minnesota, and Alsen, Steele ND2710 and Glenn developed by North Dakota State University (Mergoum , 2007). Among them, Alsen was derived from a three way cross from "ND674/ND2710/ND688", where ND2710 was from a cross involving Sumai 3 (Mergoum , 2007).

Transgressive segregation has been successfully used in creating resistant cultivars. Examples of this are some wheat cultivars from Southern Chinese: Sumai 3, Zhen 7495, Xiangmai 2, Jingzhou 1 and Jingzhou 47 (Bechtel , 1985; Bai , 2000). Thus, selecting elite resistant lines from transgressive segregation of resistance may be able to improve the level of FHB resistance. Besides, many other breeding strategies have been applied in wheat FHB resistance (Bai and Shanner, 2004; Lu , 2001), such as introduction of alien resistance genes or chromosomes to develop new synthetic hexaploid wheat cultivars (Gilchrist , 1997, Rudd , 2001). For example, wheat addition lines with chromosomes from *Roegneria ciliaris*, *R. kamoji*, or *Elymus giganteus* showed FHB resistance as Sumai 3 in China (Chen , 1993).

REFERENCES

Akkaya MS, Bhagwat AA and Cregan PB. 1992. Length polymorphisms of simple sequence repeat DNA in soybean. Genetics, 132: 1131-1139.

Anderson JA, Stack RW, Liu S, Waldron BL and Fjeld AD. 2001. DNA markers for Fusarium head blight resistance QTL in two wheat populations. Theor. Appl. Genet, 102(8): 1164–1168.

Argyris J, TeKrony D, Hershman D, VanSanford D, Hall M, Kennedy B, Rucker M and Edge C. 2005. Fusarium Head Blight Infection following Point Inoculation in the Greenhouse Compared with Movement of Fusarium graminearum in Seed and Floral Components. Crop Science, 45: 626-634.

Arthur JC. 1891. Wheat scab. Indiana Agric. Exp. Stn. Bull, 36: 129–38.

Bai GH, Zhou CF, Qian CM and Ge YF. 1990. Genetic analysis on resistance genes to scab spread in wheat cultivars. In L-H. Zhu (ed) Advances in Researches on Inheritance to Diseases in Major Crops: 171-177. Jiangsu Sci-Tech Publ House, Nanjing. China.

Bai GH and Shaner G. 1994. Scab of wheat: Prospects and Control. Plant Dis, 78: 760-766.

Bai GH and Shaner G. 1996. Variation in Fusarium graminearum and cultivar resistance to wheat scab. Plant Dis, 80: 975-979.

Bai GH, Kolb F, Shaner G and Domier L. 1999. AFLP markers linked to one major QTL controlling scab resistance in wheat. Phytopathology, 89: 343-348.

Bai GH, Shaner G and Ohm H. 2000. Inheritance of resistance to Fusarium graminearumin wheat. Theor. Appl. Genet, 100: 1-8.

Bai GH, Plattner RD, Desjardins A and Kolb FL. 2001. Resistance to Fusarium head blight and Deoxynivalenol accumulation in wheat. Plant Breeding, 120: 1-6.

Bai GH, Guo PG and Kolb FL. 2003a. Genetic relationships among scab-resistant cultivars of wheat based on molecular markers. Crop Science, 43: 498-507.
Bai GH, Chen LF and Shaner GE. 2003b. Breeding for resistance to Fusarium head blight of wheat in China. St Paul, MN. APS Press: 296–317.
Bai GH and Shaner G. 2004. Management and Resistance in Wheat and Barley to Fusarium Head Blight. Annual Review of Phytopathology, 42: 135-161.
Ban T. 2000. Analysis of quantitative trait loci associated with resistance to Fusarium head blight caused by Fusarium graminearum Schwabe and of resistance mechanisms in wheat (Triticum aestivum L.). Breed. Sci. 50(2): 131–137. 63