IDENTIFICATION OF NEW SOURCES AND MAPPING OF QTL FOR FHB RESISTANCE IN ASIAN WHEAT GERMPLASM

by

JIANBIN YU

B.S., Nanjing Agricultural University, China, 1986
M.S. Oklahoma State University, Oklahoma, 1999

AN ABSTRACT OF A DISSERTATION

submitted in partial fulfillment of the requirements for the degree

DOCTOR OF PHILOSOPHY

Department of Agronomy
College of Agriculture

KANSAS STATE UNIVERSITY
Manhattan, Kansas

2007

Abstract

Growing resistant cultivars is an economically effective method to control wheat disease *Fusarium* head blight (FHB) caused by *Fusarium graminearum*. Ninety-five wheat lines mainly from China and Japan were evaluated for resistance to initial infection (type I), spread of symptoms within a spike (type II), and deoxynivalenol (DON) accumulation in infected grains (type III). Most of lines were resistant or moderately resistant, 15 lines had DON content lower than 2 ppm and six lines showed a high level of resistance for all the three types. Deoxynivalenol content was significantly correlated with type II, but not type I resistance.

Fifty-nine of the ninety-five lines were evaluated for genetic diversity on the basis of amplified fragment length polymorphism (AFLP) and simple sequence repeats (SSRs). Genetic relationships among these lines were consistent with pedigrees and their geographic distribution. Chinese lines had broader genetic diversity than Japanese lines. Sumai 3 is a widely used Chinese variety for FHB-resistant breeding in the US and elsewhere. Haplotype patterns of the SSR markers linked to FHB resistance quantitative trait loci (QTL) on chromosomes 3BS, 5AS and 6BS of Sumai 3 indicated that only a few Sumai 3 derivatives carry all of these Sumai 3 QTL. SSR data also suggested that these QTL in Sumai 3 were derived from Chinese landrace Taiwan Xiaomai. Some highly resistant lines may carry novel QTL for FHB-resistance QTL, and need further investigation.

A mapping population of 139 recombinant inbred lines derived from the cross of Wangshuibai (resistant Chinese landrace)/Wheaton (susceptible cultivar) was genotyped with more than 1300 SSR and AFLP markers. Five QTL for type I resistance were detected on chromosome arms 3BS, 4BS, 5DL, 3AS, and 5AS; seven QTL for type II resistance on 3BS, 1AL, 5AS, 5DL, 7AL, and 3DL; and seven QTL for type III resistance on 3BS, 5AS, 1AS, 5DL, 1BL, and 7AL. These QTL together explained 31.7%, 64%, and 52.8% of the phenotypic variation for FHB type I, II, and III resistance, respectively. FHB resistance QTL identified in Wangshuibai can be used in developing wheat cultivars with enhanced FHB resistance by pyramiding FHB resistance QTL from other sources.

IDENTIFICATION OF NEW SOURCES AND MAPPING OF QTL FOR FHB RESISTANCE IN ASIAN WHEAT GERMPLASM

by

JIANBIN YU

B.S., Nanjing Agricultural University, China, 1986
M.S. Oklahoma State University, Oklahoma, 1999

A DISSERTATION

submitted in partial fulfillment of the requirements for the degree

DOCTOR OF PHILOSOPHY

Department of Agronomy
College of Agriculture

KANSAS STATE UNIVERSITY
Manhattan, Kansas

2007

Approved by:

Major Professor
Guihua Bai

Abstract

Growing resistant cultivars is an economically effective method to control wheat disease *Fusarium* head blight (FHB) caused by *Fusarium graminearum*. Ninety-five wheat lines mainly from China and Japan were evaluated for resistance to initial infection (type I), spread of symptoms within a spike (type II), and deoxynivalenol (DON) accumulation in infected grains (type III). Most of lines were resistant or moderately resistant, 15 lines had DON content lower than 2 ppm and six lines showed a high level of resistance for all the three types. Deoxynivalenol content was significantly correlated with type II, but not type I resistance.

Fifty-nine of the ninety-five lines were evaluated for genetic diversity on the basis of amplified fragment length polymorphism (AFLP) and simple sequence repeats (SSRs). Genetic relationships among these lines were consistent with pedigrees and their geographic distribution. Chinese lines had broader genetic diversity than Japanese lines. Sumai 3 is a widely used Chinese variety for FHB-resistant breeding in the US and elsewhere. Haplotype patterns of the SSR markers linked to FHB resistance quantitative trait loci (QTL) on chromosomes 3BS, 5AS and 6BS of Sumai 3 indicated that only a few Sumai 3 derivatives carry all of these Sumai 3 QTL. SSR data also suggested that these QTL in Sumai 3 were derived from Chinese landrace Taiwan Xiaomai. Some highly resistant lines may carry novel QTL for FHB-resistance QTL, and need further investigation.

A mapping population of 139 recombinant inbred lines derived from the cross of Wangshuibai (resistant Chinese landrace)/Wheaton (susceptible cultivar) was genotyped with more than 1300 SSR and AFLP markers. Five QTL for type I resistance were detected on chromosome arms 3BS, 4BS, 5DL, 3AS, and 5AS; seven QTL for type II resistance on 3BS, 1AL, 5AS, 5DL, 7AL, and 3DL; and seven QTL for type III resistance on 3BS, 5AS, 1AS, 5DL, 1BL, and 7AL. These QTL together explained 31.7%, 64%, and 52.8% of the phenotypic variation for FHB type I, II, and III resistance, respectively. FHB resistance QTL identified in Wangshuibai can be used in developing wheat cultivars with enhanced FHB resistance by pyramiding FHB resistance QTL from other sources.

Table of Contents

List of Figures

List of Tables

Acknowledgements

I would like to thank my major professor Dr. Guihua Bai for his thoughtful guidance, friendship and consistent support to work out a long distance doctoral plan. I am grateful to the members of my advisory committee, Dr. Allan K. Fritz, Dr. Bikram S. Gill, and Dr. James C. Nelson, who took their precious time to serve on my thesis committee and provide great guidance and assistance throughout my graduate program. Thanks also go to Dr. Rollie J. Clem for serving as the outside chair of my final examining committee.

I would like to send my special thanks to our collaborators: Drs. Frederic L. Kolb, Wenchun Zhou, Tomohiro Ban, and Shibin Cai for their generous contributions of some genetic materials necessary for completion of this project as well as for assistance given throughout this project.

Many thanks are owed to Dr. Amy Bernardo, Dr. Paul St. Amand, Dr. Hongxiang Ma, Dr. Jun Yang, Benjamin Echalier, Shilpa Sood, and Lili Zhou for their help and friendship. Lastly, I would like to express my sincere gratitude and thanks to my family, my parents, my wife Hong, and my daughter Dandan for their love, understanding and support throughout this process.

CHAPTER 1 - Literature Review

The evolution of cultivated wheat

Wheat belongs to the Gramineae (Poaceae) family and consists of two genera, *Triticum* and *Aegilops* (van Slageren 1994). Wheat can be grouped into three groups based on ploidy level, diploid (2n = 2x = 14 chromosomes), tetraploid (2n = 4x = 28), and hexaploid (2n = 6x = 42), with the diploid and tetraploid groups including wild species. The wild wheat species *T. monococcum* ssp. *aegilopoides* (wild einkorn, diploid), *T. urartu* (diploid), and *T. turgidum* ssp. *dicoccoides* (wild emmer, tetraploid) are involved in domestication. The cultivated diploid is *T. monococcumm* ssp. *monococcum* (einkorn). Cultivated tetraploids are divided into two species, *T. timopheevii* and *T. turgidum*. Only the subspecies *timopheevii* within *T. timopheevii* is cultivated. Seven subspecies within *T. turgidum* are cultivated: ssp. *dicoccum* (emmer), ssp. *paleocolchicum* (Georgian), ssp. *durum* (macaroni), ssp. *turgidum* (rivet or cone), ssp. *polonicum* (Polish), ssp. *turanicum* (Khorassan), and ssp. *carthlicum* (Persian). There are two cultivated hexaploids, *T. zhukovskyii* and *T. aestivum* (known as common, bread or dinkel wheat). Five subspecies within *T. aestivum* are cultivated: ssp. *aestivum* (common or bread), ssp. *spelta* (dinkel or large spelt), ssp. *macha*, ssp. *compactum* (club), and ssp. *sphaerococcum* (shot) (Simons 2005).

The polyploid wheat species constitute two evolutionary lineages. *T. turgidum* (2n = 4x = 28, AABB genomes) and *T. aestivum* (2n = 6x = 42, AABBDD genomes) compose one lineage, and *T. timopheevii* (AAGG) and *T. zhukovskyii* (A^mA^mAAGG) comprise the other (Faris *et al*. 2002). Cytological data on the meiotic pairing behavior of interspecific hybrids between 2x/4x and 4x/6x wheats showed that *T. monococcumm* and *T. turgidum* share one genome and *T. turgidum* and *T. aestivum* share two genomes (Kihara 1924; Sax 1922). It was later discovered that *T. monococcum* was a mixture of two species, *T. monococcum* and *T. urartu* (Johnson and Dhaliwal 1976). The molecular evidence supports that *T. urartu* actually is the A genome donor of both tetraploid and hexaploid wheats (Dvorak *et al*. 1988, 1993). The contributor of the B genome is not clear, but evidence based on karyotype data (Riley *et al*. 1958), C-banding of

chromosomes (Friebe and Gill 1996), cytology (Kerby and Kuspira 1988), the geographical distribution of wheat populations (Witcombe 1983), and molecular genetics (Dvorak and Zhang 1990) support that the S genome of *Ae. speltoides* is the most likely donor of the B genome of bread wheat. However, whether *Ae. speltoides* is the only donor of B genome or B genome resulted from an introgression of several parental species remains uncertain (Zohary and Feldman 1962; Blake *et al*. 1999). The D genome of bread wheat was contributed by goat grass, *Aegilops tauschii* (Kihara 1944; McFadden and Sears 1946).

The Fertile Crescent is considered the birth-place of cultivated wheat about 8,000 to 10,000 years ago. Genetic evidence indicates that einkorn wheat (*T. monococcum*) may have been domesticated from wild einkorn (*T. monococcum* ssp *aegilopoides*) in the region of the Karacadag Mountains in southeast Turkey based on archeological findings dating from 7,500 to 6,200 BC (Heun *et al.* 1997). The remains of cultivated emmer (*T. turgidum* spp. *dicoccum*) have been discovered at several archaeological sites in Syria dating to 7,500 BC (Zohary and Hopf 1993). Bread wheat arose in the region from Armenia in Transcaucasia to the southwest coastal areas of the Caspian Sea in Iran (Dvorak *et al*. 1998). In this region, *Ae. tauschii* var. *strangulata* is predominant and evidently hybridized with cultivated emmer followed by spontaneous chromosome doubling to produce *T. aestivum*. Several independent hybridization events probably occurred during the process of forming *T. aestivum* (Talbert *et al*. 1998). The timeline of wheat evolution was also estimated based on gene sequence comparisons of the *Acc-1* (plastid acetyl-CoA carboxylase) and *Pgk-1* (plastid 3-phosphoglycerate kinase) genes, which indicated that the A genome of tetraploid and hexaploid wheat diverged from *T. urartu* less than half a million years ago, indicating a relatively recent origin of polyploid wheat. The D genome sequences of *T. aestivum* and *Ae. tauschii* are indistinguishable, consistent with the theory that *T. aestivum* arose from hybridization of *T. turgidum* and *Ae. tauschii* only 8,000 years ago (Huang *et al.* 2002).

Wheat genetic system

Genetic stocks

The plasticity of wheat genome allows various forms of aneuploids to be exploited for genetic investigations. Because of the polyploid nature of bread wheat, its genome is highly buffered and can tolerate structural and numerical changes to a much higher extent than diploid.

Several kinds of aneuploid stocks of wheat have been developed. The most widely used aneuploid stocks are those that are missing a single chromosome (monosomics, Sears 1954), a pair of chromosomes from one genome and compensated by additional two copies of a homoeologous chromosome from another genome (nullisomic-tetrasomic, Sears 1966), or a pair of chromosome arms (ditelosomics, Sears and Sears 1978). Using wheat aneuploid stocks, the 21 chromosomes of wheat were grouped into seven homoeologous groups with each consisting of one chromosome each from the A, B, and D genomes (Sears 1966). These aneuploid stocks also provide cytogenetic tools that allow physical mapping of wheat genes or markers to individual chromosome and chromosome arm.

Chromosome deletion lines are another unique genetic stock developed in wheat for genetic investigations (Faris *et al*. 2002). Wheat chromosome deletion lines were developed with the gametocidal (Gc) factors. The Gc factors have been identified in different related *Aegilops* species. Gc chromosomes can be introduced into wheat by interspecific hybridization with the related *Aegilops* species, and then backcrossed to wheat. Plants with monosomic Gc chromosome will produce two types of gametes: those possessing the Gc chromosome are normal and the others without Gc chromosome will undergo structural chromosome aberrations and are malfunctioned in most cases. However, if the aberration is not sufficient to kill the gamete, the gamete may still be functional and be transmitted to the offspring. More than 400 deletion stocks spanning all wheat chromosome regions have been developed with the Gc system (Endo and Gill 1996). These stocks have been used in physical mapping of wheat genes or markers to chromosome bins.

Cytogenetic and molecular mapping

Aneuploids provide useful tools for locating genes that behave qualitatively or quantitatively on particular chromosomes in wheat. One of the cytogenetic mapping methods in wheat involves the substitution of each of the 21 chromosomes from a donor variety for its homologue in a recipient variety. By this means, it is possible to make a series of 21 pure lines in which the genetic effects of individual chromosomes from a donor variety can be assayed in a constant genetic background and compared with the homologue of the recipient. Genetic contribution of each chromosome to a phenotypic trait can then be evaluated (Kuspira and Unrau 1957). After an association has been established between a particular chromosome and a

quantitative trait, the substitution line involving that chromosome can be used in crosses to detect recombination and estimate the number of genes controlling the trait in that chromosome (Law 1966).

Aneuploid and deletion lines have been used as powerful tools for physical mapping of gene into a small chromosome bin. Wheat deletion lines, using C-bands as reference markers, facilitate cytologics-based physical mapping (Werner *et al*. 1992; Gill *et al*. 1993). Nullisomic-tetrasomic lines can locate a gene of interest to a specific chromosome, ditelosomic stocks can map the gene on one arm of the corresponding chromosome, and deletion stocks can locate the chromosome bin that harbors the gene (Faris *et al*. 2002).

In the 1990s, molecular mapping in wheat took a leap forward with the application of DNA markers. Restriction fragment length polymorphisms (RFLPs) were the first DNA marker system to be used to construct the linkage maps of wheat in a recombinant inbred population from a cross of 'Opata' and a synthetic hexaploid, which was formed by crossing the durum cultivar 'Altar84' (AB genomes) with *Ae. tauschii* (accession 219) (D genome) followed by colchicine doubling (Devos *et al*. 1993; Marino *et al*. 1996; Nelson *et al*. 1995a, b, c; Van Deynze *et al*. 1995a). The maps have been supplemented with other kinds of molecular markers such as simple sequence repeats (SSRs) (Röder *et al*. 1998).

The aneuploid and deletion stocks also provide ideal materials for physical mapping of expressed sequence tags (ESTs) and other molecular markers, such as microsatellites, to small chromosomal regions. The chromosome bin map of ESTs is a unique resource for comparative mapping and structural and functional analysis of the wheat genome (Qi *et al*. 2004). The physically mapped ESTs that are linked to a QTL in a deletion bin can be potential candidate genes for the QTL (Sourdille *et al*. 2004).

Comparative mapping

Comparative mapping involves the alignment of chromosomes of related species based on genetic mapping of common DNA markers. The idea behind comparative mapping is that comparing the genomes of two related species can help locate important genes that have been identified in one species but not in another, and can provide clues about how both species evolved from a common ancestor. Synteny denotes the occurrence of two or more genetic loci on the same chromosome. It also describes the preserved similar linear order of genes between related species. The three genomes of common wheat are syntenic at the resolution of current

maps with few exceptions such as the well-documented 4AL-5AL-7BS translocation (Devos *et al*. 1995; Naranjo *et al*. 1987; Nelson *et al*. 1995c). This synteny has been extended to the A and D genome diploid progenitors as well as barley (Kam-Morgan *et al*. 1989; Van Deynze *et al*. 1995b; Dubcovsky *et al*. 1996). Functional genes located in syntenic regions of wheat relatives have emerged, such as the vernalization gene *Vrn-A1* in *T. monococcum* and *T. aestivum* (Dubcovsky *et al*. 1998).

Comparing the genomic relationships among wheat and other members of the Poaceae like rice, barley, rye, oats, and maize has also revealed remarkable similarities in gene content and marker synteny at the chromosome level. The presence of syntenic regions among these related species was demonstrated using a common set of anchor DNA probes that hybridize well to these species. These probes identify sets of orthologous loci that lie at approximately the same positions relative to each other (Van Deynze *et al*. 1998). The synteny with rice can be exploited as a means of providing markers to saturate the homologous regions in wheat. For example, the positions of ESTs of other grass species relative to rice bacterial artificial chromosome (BAC) insert can be used to construct synteny maps by aligning these EST markers. Such synteny maps will facilitate comparative mapping and genomic investigations of a large proportion of ESTs identified in the grasses by leveraging the information from all these species.

Direct use of rice genomic information for gene discovery in wheat may be difficult. The similarity observed at chromosome level among grass genomes led to the hope that information from rice genome could be directly applied to the much larger genome of wheat. However, studies of the degree of microcolinearity between wheat and rice have shown numerous chromosomal rearrangements (Sorrells *et al*. 2003; Liu *et al*. 2006).

Positional cloning

Since an efficient transposon-based tagging system has not yet been developed for wheat, cloning wheat genes mainly uses two methods: cloning based on a gene of similar function (candidate gene approach) and positional cloning (map-based cloning). The candidate gene approach is relatively easy. In this method, the primers based on homologues in other species are used to amplify (by PCR) the gene in wheat. For example, the reduced-height gene, *Rht1*, was cloned based on its homology with dwarfing genes in *Arabidopsis* and rice (Peng *et al*. 1999). Positional cloning typically involves the identification of partially overlapping DNA segments, which harbor the gene of interest, from a genomic library in an attempt to progress along the

chromosome toward the gene. Positional cloning usually is time consuming, but its feasibility has been demonstrated. For example, a QTL controlling tomato fruit weight, *fw2.2* (Frary *et al*. 2000), wheat leaf rust resistance gene *Lr21* (Huang *et al*. 2003), and the major wheat domestication gene *Q* (Faris *et al*. 2005, Simons *et al*. 2006) were isolated by means of positional cloning.

The steps of positional cloning in wheat can be summarized as follows: Initially, physical mapping locates the gene of interest to a specific chromosome bin, and molecular mapping can identify molecular markers tightly linked to the gene by use of a large mapping population. Second step is to develop high-resolution map with all available molecular markers within the chromosome bin and screen large-insert libraries developed from wheat diploid progenitors, such as the BAC library of *T. monococcum* (Lijavetsky *et al*. 1999), to construct a physical contig spanning the locus through the process called chromosome walking in which the closest known linked marker to the gene is used to probe the BAC library for overlapping BAC clones and search for closer linked markers. This process is repeated several times to walk across the chromosome and reach the gene of interest. Third step is to identify the candidate gene by looking for open reading frames (ORFs) in DNA sequence of the BAC clone that harbors the gene and the tightly linked markers. Finally, plant transformation using the cloned putative gene is performed to recover the wild-type phenotype. If transgenic wheat plant recovers the wild-type phenotype after candidate gene is transferred into the mutant, this will validate the function of the gene (Faris *et al*. 2005).

Wheat production and utilization

Conventionally, bread wheat is classified into two types, winter and spring, based on its growth habit. Winter wheat is sown in the fall and the plant needs to experience a certain period of cold temperature (0 to 12ºC), or vernalization for the plant to flower. Spring wheat is generally sown in the spring or in the fall without experiencing cold temperature during winter (Flood and Halloran 1986). Consequently, wheat can be grown in various climates all over the world and more of the world's farmland is devoted to wheat production than to any other food crop (Briggle and Curtis 1987). Wheat surpassed rice in the 1970s to become the most consumed food grain in the world. Currently about 240 million hectares are sown, with a total production of around 600 million metric tons annually (FAO 2006 http://faostat.fao.org/site/395/default.aspx).

The leading wheat producing countries are China, the United States, India, Russia, Kazakhstan, Canada, Argentina, and Australia (CIMMYT 1996).

Wheats can also be classified into two types based on their grain texture and protein content. The hardness of wheat is determined by expression of the *Hardness* (*Ha*) gene (Baker 1977; Giroux and Morris 1998). Hard wheat contains higher gluten than soft wheat. The flour of hard wheat is best for bread-making while the flour of soft wheat is suitable for cakes, crackers, cookies, and pastries (Johnson *et al*. 1978). Wheat is used mainly for human food but also for animal feed, and to produce starch, paste, malt, dextrose, gluten, alcohol, and other products.

In the United States, most wheat is grown in the Great Plains from Texas to Minnesota. Wheat is classified in six market classes according to the regions of production and end-use (milling and baking) characteristics. Hard red winter wheat is used to produce bread, rolls, and all-purpose flour. Most wheat of this class is grown in the central and southern Great Plains. Hard red spring wheat contains a high percentage of protein, has superior milling and baking characteristics, and is grown mainly in the northern Great Plains. Soft red winter wheat has relatively low protein and is used for flat breads, cakes, pastries, and crackers and is grown in the regions east of the Mississippi River. Hard white wheat is used mainly for yeast breads, hard rolls, bulgur, tortillas and oriental noodles and grown in the central states of the Great Plains. Soft white wheat is used for bakery products other than bread and grown primarily in the Pacific Northwest. Durum wheat is used to make semolina flour for pasta production and grown primarily in North Dakota. Kansas, North Dakota, and Oklahoma are the leading states for wheat production in the USA (USDA 2006 http://www.nass.usda.gov:8080/QuickStats).

Fusarium head blight of wheat

Pathogens

Fusarium head blight (FHB) of small grains is one of the most destructive crop diseases worldwide (Bai and Shaner 2004; Miedaner 1997). It was first recognized as a fungal disease in 1884 by W. G. Smith (Parry *et al*., 1984) and has emerged as a major threat to wheat and barley production in the early 20th century (Dickson and Mains 1929). Several species of the soil- and residue-borne fungus, *Fusarium*, can cause wheat FHB, and the symptoms caused by these different species are almost the same.

F. graminearum and F. culmorum are the major pathogens responsible for wheat FHB in different countries (Bai and Shaner 1994; Lu *et al*. 2001; Shaner 2003; Schroeder and Christensen 1963; Sutton 1982; Wiese 1987). *F. graminearum* has been the major pathogen in most of the recent outbreaks of FHB in the USA, Canada, and many other countries (Bai and Shaner 2004; Dubin *et al*. 1997; Parry *et al.* 1995). *Gibberella zeae* is the sexual stage of *F. graminearum* and was classified into two groups based on whether they were heterothallic (group 1) or homothallic (group 2). Aoki and O'Donnell (1999a, 1999b) reclassified group 1 (Burgess *et al.* 1981) as a new species called *Gibberella coronicola. F. culmorum* was found to be a major pathogen responsible for wheat FHB in some European countries (Mesterházy 2003b; Snijders 1990b; Wiese 1987).

Natural FHB infection and development

FHB epidemic potential is closely tied to the reproductive strategy of the pathogen. *F. graminearum* can survive in living or dead host tissues as mycelium, ascospores, macroconidia and chlamydospores. Ascospores are the propagules of the sexual stage (Reis 1990). Hyphal fragments, ascospores, macroconidia, and chlamydospores all can serve as inoculum (Zhu and Fan 1989). Ascospores released from crop debris on soil surface are the principal inocula that initiate epidemics because aerial dispersal is necessary for the fungus to reach the infection site (Sutton 1982). In addition, wheat planted after corn or wheat usually has significantly more FHB than wheat planted after other crops (Fig 1.1). Thus, reduced tillage for soil conservation increases the amount of inoculum that can infect wheat (Shaner 2003; Teich and Hamilton 1985; Zhu and Fan 1989).

The abundance of primary inoculum and weather conditions, mainly moisture and temperature, during and after anthesis determine FHB severity. Airborne ascospores are deposited on or inside wheat florets and subsequently germinate and initiate infection. The fungus rapidly infects the extruded anthers and then ramifies throughout the developing caryopsis, floral bracts, and rachis. The fungus may also infect wheat by direct penetration of the glume, palea or rachila. Soon after infection, dark brown spots appear on the infected florets and later the entire florets become blighted (Bai and Shaner, 1994; Bushnell *et al.* 2003). The fungus spreads internally through vascular bundles of the rachilla and rachis in susceptible wheat. Brownish chlorotic symptoms extend up- and downward and eventually the entire spike becomes blighted. If the weather is favorable, aerial mycelium can also spread externally from the infected

spikelet to nearby spikelets. Infected florets often fail to produce grain, or the kernel is poorly filled (Bushnell *et al.* 2003; Wiese 1987). In nature, wheat head infection can occur any time after the beginning of the flowering but anthesis is the most vulnerable period for infection. Owing to this brief period of vulnerability, the fungus is limited to one infection cycle per season in wheat (Strange and Smith 1987). Primary infection may occur on several florets of a spike in the field and the dark brown symptoms usually extend into the rachis. The clogging of vascular tissues in the rachis can cause the head to ripen prematurely, so that even grains not directly infected will be shriveled owing to a shortage of water and nutrients (Schroeder and Christensen 1963). The optimum temperature for infection and disease development is 25°C, with little or no infection occurring at 15°C. Incidence increases as temperature increases from 20 to 30°C and the moist period required for infection is about 36 to 72 hours (Anderson 1948).

Economic loss from FHB

FHB significantly reduces wheat grain yield and quality. Yield reduction results from shriveled grains, which may be light enough to be expelled from the combine with the chaff. In addition, the germination of infected seeds is low and causes seedling blight and poor stand (Bai and Shaner 1994). Over $2.6 billion of direct losses and $7.7 billion of total losses have been estimated for the cumulative economic impact of FHB to US agriculture during FHB epidemics in the 1990s (Johnson *et al.* 2003; Nganje *et al.* 2004). Moreover, this disease is increasingly becoming a threat to the world's food supply due to recent widespread head blight outbreaks in Asia, Canada, Europe and South America (Dubin *et al.* 1997). It is estimated that in China FHB can affect up to 7 million hectares of wheat and can cause yield losses of more than 1 million tons nationally in severe epidemics (Bai *et al.* 2003; Lu *et al.* 2001). FHB causes additional loss for agriculture because scabby grain is often contaminated with mycotoxins produced by *F. graminearum*, especially deoxynivalenol (DON). DON is also known as vomitoxin because it is responsible for causing feed refusal and vomiting in swine (Tuite *et al.* 1990). Contamination of DON makes the grain unsuitable for food or feed and it may be graded down or rejected entirely in commerce (McMullen *et al.* 1997).

Strategies for control of Fusarium head blight

Various cultural control measures for reducing FHB damage have been practiced. Appropriate methods of land preparation, good crop husbandry, timely harvest, proper storage

and crop rotation help reduce the disease by reducing primary inoculum but the adoption of minimum tillage for soil conservation reduces the options for the disease-management technique. In addition, adequate control by those methods is not possible because of the wide host range of *F. graminearum* (Bai and Shaner 1994; Reis 1990). Seed treatment and foliar application with fungicide at anthesis might provide some protection but the high cost, the difficulty of determining the optimum time of application, and the lack of registered fungicides that are highly effective make this means of control less attractive to farmers. Even if a fungicide can reduce direct yield loss, it may not reduce mycotoxin contamination to a tolerable level for food or feed (Martin and Johnston 1982; Mesterházy 2003a). Hence, breeding for durable resistance against this disease in wheat is the most economical and effective means of reducing yield loss as well as mycotoxin contamination. Considerable progress in the search for host resistance has been made in China, Japan, and other countries (Bai *et al.* 2003; Ban 2001; Mesterházy 2003b). Improving FHB resistance of wheat cultivar has become a major breeding objective worldwide. The current developments in genomics and biotechnology offer promise for understanding of the genetic mechanisms of FHB resistance and for developing new FHB resistant wheat cultivars.

Wheat resistance to Fusarium head blight

Types of resistance

Mesterházy (1995) proposed five types of FHB resistance: (1) resistance to initial infection, (2) resistance to spread of infection within a spike, (3) resistance to kernel infection, (4) tolerance, and (5) resistance to toxin accumulation. However, only three types are commonly accepted (Schroder and Christensen 1963, Miller *et al.* 1985): resistance to primary infection (type I), to spread of hyphae within a wheat spike (type II) and to mycotoxin accumulation in infected kernels (type III).

Different inoculation methods are used to evaluate these types of resistance in wheat. Type I resistance is estimated by spraying a spore suspension over flowering spikes and counting the diseased spikelets. Type II resistance is estimated by delivering conidia into a single floret of a spike and counting the blighted spikelets after a certain period of infection. Measurement of type III resistance is performed by chemical analysis of grain samples. These procedures are typically done in a greenhouse so that the conditions for FHB infection can be carefully controlled and genotype × environment effects can be minimized. Type II resistance has been

extensively studied in wheat and shown to be more stable and less affected by nongenetic factors than type I resistance (Bai and Shaner 1994). It may be difficult to distinguish type II from type I resistance in the field under conditions favorable for FHB. A genotype with type I resistance but no type II resistance may be highly susceptible because blight symptoms will subsequently spread throughout the spike from one infected spikelet. On the other hand, if a genotype has type II resistance without type I resistance, a high proportion of spikelets can be infected directly under heavy inoculum pressure and favorable environment, and blight symptoms may cover most of the spike regardless of type II resistance (Bai and Shaner 1996). Type III resistance may result from three possible causes: a low level of mycotoxin produced by the fungus, a degradation of mycotoxin by plant enzymes during kernel development, or the failure of mycotoxin to move into kernels during their development (Bai and Shaner 2004). The remaining two types, resistance to kernel infection and tolerance, have not been widely accepted because of some conceptual or operational weaknesses (Shaner 2002).

Mechanisms of resistance

Resistance mechanisms to FHB in wheat are classified as either active (physiological) or passive (morphological) (Mesterházy 1995; Rudd *et al.* 2001). Active resistance mechanisms include all resistance types mentioned above including inhibition of infection, restriction of colonization after infection, and metabolic degradation of chemicals produced by the pathogen. Currently, breeding efforts throughout the world are focused primarily on type II resistance. Reports have indicated that under epidemic conditions type I resistance is easily overcome, and thereafter type II resistance becomes the most promising type of defense to reduce the losses due to FHB infection (Mesterházy 1995). Passive resistance mechanisms are associated with phenotypic traits such as plant height, presence of awns, spikelet density, and time to flowering. For example, genotypes that flower concurrently with favorable environmental conditions for spore dispersal and infection are more likely to develop severe FHB symptoms. In addition, disease is decreased in genotypes with a shorter flowering period and in those genotypes with anthers barely released (Mesterházy, 1995). Waxy glumes may serve as a barrier to the fungal infection and help to exclude moisture, and tight glumes may limit access of airborne inoculum to wheat flower organs.

The molecular and biochemical mechanisms of wheat resistance to FHB are still unknown. Different expression patterns of several defense-related proteins and enzymes such as

ascorbic acid peroxidase and catalase were observed between resistant and susceptible wheat lines (Bai and Shaner 2004; Hill-Ambroz *et al*. 2006). DON produced by the fungus has been proposed as a virulence factor for FHB. Disruption of the gene encoding trichodiene synthase (*Tri*5) in *F. graminearum* reduced disease severity and restoration of *Tri5* gene resulted in increased FHB severity and DON production (Desjardins *et al.* 1996). This DON-nonproducing fungus could still infect the inoculated spikelets of wheat in both greenhouse and field (Bai *et al.* 2001a). This led to the suggestion that DON may not be essential for initiating infection but may involve in symptom development and spread of the fungus within a spike. Recently, the DON detoxification gene *Tri101* from *F. sporotrichioides* has been transformed into wheat and transgenic plants and transgenic plants were reported to show a significant reduction in FHB severity in greenhouse experiments (Okubara *et al.* 2002). Also, the major QTL for low DON content was mapped to the same location as the major QTL for type II resistance (Lemmens *et al.* 2005). Both studies suggest that DON may play an important role in disease development. Wheat resistance to FHB is a quantitative trait and probably involves a complex and interacting network of signaling pathways. Application of new technologies for large-scale gene analysis may facilitate discovery of critical pathways and key genes in these pathways (Bai and Shaner 2004).

Sources of resistance

Resistance to FHB in wheat and wheat alien species has been identified mainly from four origins: Europe, East Asia, South America, and North America (Table 1.2). Most of these sources have not been extensively evaluated, so their resistance may be uncertain and need further validation. Among the FHB-resistant wheat genotypes, most highly resistant wheat lines have Chinese resistance sources, mainly Sumai 3 or its derivatives, in their pedigrees. Only a few wheat lines appear to have different sources of resistance, without known relationship with Chinese sources, such as Chokwang from Korea (Shaner and Buechley 2001), Fundulea 201R from Romania (Shen *et al.* 2003a), and Ernie and Freedom from the USA (Rudd *et al*. 2001). To date, wheat with complete resistance has not been found (Ban 1997; Bai and Shaner 2004). The best-known resistance sources are spring wheats from China such as Sumai 3, Japan such as Shinchunaga, and Brazil such as Frontana (Bai *et al.* 2003; Ban 2000; Singh *et al.*1995). In addition, many wheat alien species have been screened to identify FHB resistance genes (Cai *et al*. 2005). Wan *et al.* (1997) reported that 13 grass species in the genera *Roegneria, Hystrix,* and

Psathyrostachys had better type II resistance than Sumai 3. Of these, *Roegneria tsukushiensis* var. *transiens* and *R. ciliaris* appeared to show complete resistance. These species may serve as alternative sources of resistance to FHB. Unfortunately, the resistance found in alien species usually does not surpass that in wheat cultivars such as Sumai 3 and is associated with many undesirable agricultural traits that are difficult to remove from the progenies of the wide crosses (Bai and Shaner 2004).

Inheritance of resistance

FHB resistance is quantitatively inherited under polygenic control of quantitative trait loci (QTL). A few major genes plus some minor genes may be responsible for type II FHB resistance with relatively high heritability (Bai *et al.* 1989; Bai and Shaner 1994; Snijders 1990a; Nakagawa 1995; van Ginkel *et al.* 1996). Additive genetic effects play the major role in general, but nonadditive gene effects might also be significant in some cases (Bai *et al.* 2000a). Of the nonadditive components, dominance appears to be the most important (Bai *et al.* 1990; Snijders 1990b), and epistatic effects are also seen (Bai *et al.* 2000a). The number and chromosome locations of FHB resistance QTL vary with the resistant wheat lines and genetic backgrounds used in molecular mapping studies. The inconsistent results in these studies may be due to the polygenic inheritance of FHB resistance, genetic background effects, different types of resistance evaluated, different inoculation methods used, genotype × environment interactions, and heterogeneous sources of a resistant parent (Kolb *et al.* 2001).

Durability of resistance

Durability of resistance is dependent on variation in pathogen virulence, host-pathogen interaction, mechanisms of resistance, and agricultural practices. Since its release 30 years ago, Sumai 3 and its derivatives have passed extensive tests in China, Japan, the United States, and many other countries with isolates of *F. graminearum* collected around the world (Bai *et al.* 2003; Ban 2001; Kolb *et al.* 2001; Mesterházy 2003b). These wheat lines are still the best source of type II resistance available for wheat breeding programs in China and other countries.

Although significant interaction between wheat genotypes and *F. graminearum* isolates has been observed, there is no evidence for stable pathogen races (Bai and Shaner 1996; Mesterházy 2003b; Snijders and van Eeuwijk 1991). Studies of resistant wheat lines all over the world concluded that resistance to certain strains of *F. graminearum* as well as to other species

of *Fusarium* was not strain- or species-specific. *Fusarium* species that cause head blight in wheat can also infect other cereals without showing specialization for any one host (Mesterházy 1981; Mesterházy 2003b; van Eeuwijk *et al.* 1995). For these reasons, FHB resistance is considered to be horizontal and potentially quite durable (Miedaner 1997), and the resistance genes in current resistant sources, such as Sumai 3, are not expected to be overcome by new *Fusarium* species in the near future.

Molecular markers

Molecular markers are DNA fragments that allow the detection of specific DNA sequence differences between two or more individual genotypes. Five properties of molecular markers distinguish them from morphological markers (Tanksley 1983): (1) genotypes can be determined with a small piece of plant tissue; (2) a relatively large number of naturally occurring alleles exist at many loci; (3) deleterious effects are not usually associated with different alleles; (4) codominant alleles at some loci can distinguish all different genotypes; (5) few epistatic or pleiotropic effects are produced, thus a very large number of segregating markers can be monitored in a single population. There are two major types of molecular markers: protein-based markers, such as isozymes, and DNA markers. DNA markers are superior to protein-based markers because the detection of variation is not limited to coding regions and all categories of mutational events may be detected. DNA markers can be subdivided into two major types: DNA-hybridization-based; and polymerase chain reaction (PCR) amplification-based.

RFLP markers

One of the earliest types of DNA-based molecular markers was restriction fragment length polymorphisms (RFLPs) (Grodzicker *et al*. 1974). DNA sequence polymorphisms can be caused by base substitution, insertion, or deletion that leads to the loss or gain of restriction sites for a restriction enzyme. Botstein *et al*. (1980) used RFLPs to construct a genome-wide genetic linkage map of human. The RFLP marker system is limited by low frequency of polymorphism, the requirement of relatively large amounts of DNA, time-consuming procedure, and the use of autoradiography, which make this technology relatively slow and expensive. Despite these disadvantages, RFLP has been used in wheat for genome mapping (Chao *et al*. 1989; Kam-Morgan *et al*. 1989), variety identification (Gupta *et al.* 1998) and marker-assisted selection

(Gale *et al.* 1995). RFLPs are still very useful for comparative mapping and gene cloning (Faris *et al*. 2002).

PCR-based markers

PCR-based markers share a number of general advantages over RFLP technology. The major advantages are the speed with which data are generated and the smaller amounts of genomic DNA template required. These markers can be based on arbitrarily primed reactions, such as random amplified polymorphic DNA (RAPD) markers (Williams *et al*. 1990) and amplified fragment length polymorphism (AFLP) markers (Vos *et al.* 1995), or on known sequences, such as simple sequence repeats (SSR) or microsatellites (Tautz and Renz 1984), sequence tagged sites (STS) (Weber and May 1989), sequence characterized amplified regions (SCAR) (Paran and Michelmore 1993), and single nucleotide polymorphisms (SNP) (Brookes 1999). SSR markers detect variation in the number of short repeat sequences, usually of one to six bases. SSR markers are mainly codominant and dispersed throughout the wheat genome (Röder *et al.* 1998). They are highly polymorphic and are easier to use than AFLPs. Several genetic linkage maps of wheat have been generated using different mapping populations and different molecular marker systems (Devos and Gale 1997; Röder *et al.* 1998; Pestsova *et al.* 2000; Gupta *et al.* 2002). A high-density consensus map of wheat was constructed from four of these maps (Somers *et al.* 2004). The availability of high-density linkage maps of wheat and numerous PCR-based molecular markers has greatly facilitated progress in genetic studies of wheat.

Major applications of molecular markers include identification and fingerprinting of genotypes, assessment of genetic diversity between germplasm and breeding materials, detection of monogenic and quantitative trait loci (QTL), and marker-assisted selection (MAS). In wheat, molecular markers have been linked to about 40 traits of economic importance (Gupta *et al.* 1999). An up-to-date list of the genes/traits that have suitable molecular markers for wheat MAS has been maintained at the wheat Coordinated Agricultural Project website (Wheat CAP, http://maswheat.ucdavis.edu/).

Mapping FHB resistance QTL in wheat

Briefly, every chromosome in wheat has been reported to be associated with FHB resistance (Table 1.1). The major QTL on 3BS has been identified in many Chinese lines and has the largest effect on type II resistance. The QTL on 5AS is another frequently reported QTL in both Chinese and European FHB resistant wheat cultivars. Other FHB resistance QTL were also identified but with smaller effect on FHB resistance and less consistency among reports than the 3BS QTL.

Several types of markers have been used to identify QTL for FHB resistance. Two QTL from Japanese resistant cultivar Fukuhokomugi were identified by means of random amplified polymorphic DNA (RAPD) markers (Ban 1997). Five QTL were detected with restriction fragment length polymorphism (RFLP) markers in a recombinant inbred line (RIL) population derived from Sumai 3 × Stao (Waldron *et al.* 1999). Two QTL, one on 3BS from Sumai 3 and the other on 2AL from Stoa, had a major effect on FHB resistance, explaining respectively 15.4% and 14.3% of phenotypic variation. In another study, 11 AFLP markers tightly linked to a major QTL for FHB resistance were identified in a RIL population developed from Ning 7840 × Clark (Bai *et al.* 1999). This QTL could explain up to 53% phenotypic variation of type II resistance and was also associated with low DON accumulation in infected kernels (Bai *et al.* 2000b). Later, this major QTL was mapped to chromosome arm 3BS (Zhou *et al.* 2002).

The major 3BS QTL, *Qfhs.ndsu-3BS* has been identified with SSR markers in Sumai 3 and its derivatives in different mapping populations such as Sumai 3 × DT 486 (Gilbert *et al.* 2000), Sumai 3 × Stoa (Waldron *et al.* 1999), Ning 7840 × Clark (Zhou *et al.* 2002), and CM-82036 × Remus (Buerstmayr *et al.* 2002; Buerstmayr *et al.* 2003a). This major QTL was also identified in other Chinese resistant sources, such as Wuhan-1 (Somers *et al.* 2003), Huapei 57-2 (Bourdoncle and Ohm 2003a), Wangshuibai (Lin *et al.* 2004; Zhou *et al.* 2004), and W14 (Chen *et al.* 2006).

Other QTL detected in these mapping populations were QTL on 2BL and 2AL in Ning 7840 × Clark population (Zhou *et al.* 2002); QTL on 5A and 1B in five different breeding populations with resistant parents CM-82036 (Anderson *et al.* 2001; Angerer *et al.* 2003) and Sumai 3 (Bai *et al.* 1999); QTL on 2DL, 4B, and 5A in the cross Wuhan-1 × Maringa (Somers *et al.* 2003); QTL on 5A in W14 (Chen *et al.* 2006); QTL on 5B, 1B, 6B, 3A, 7A, 3D, 2A, 2D, 4B, and 5A in mapping populations with Wangshuibai as the resistant parent (Lin *et al.* 2004; Lin *et al.* 2006; Mardi *et al.* 2005; Zhang *et al.* 2004; Zhou *et al.* 2004). In a study evaluating Sumai 3

substitution lines for type II resistance and DON accumulation, it was found that QTL on 7A, 3B, 2B, and 6B from Sumai 3 significantly reduced FHB symptoms; QTL on chromosomes 7A and 3B reduced both head blight severity FHB and DON accumulation in infected grains; and QTL on chromosomes 1B, 2D, and 4D appeared to increase DON accumulation (Zhou *et al.* 2002).

Quantitative trait loci from sources other than Chinese origins have also been reported. A QTL on 3A was identified in *T. dicoccoides* with SSR marker *Xgwm2* tightly linked to it (Otto *et al.* 2002). The same QTL was detected on 3A in a recombinant inbred population derived from cross Patterson/Fundulea 201R (Shen *et al.* 2003a). Nine QTL were detected in RILs derived from a resistant European winter wheat cultivar Renan. One QTL on 2B and two on 5A are stable and each explains 6% to 19% of phenotypic variation. QTL on 2A, 3A, and 3B have minor effects (Gervais *et al.* 2003). QTL on 3A and 5A in cultivar Frontana explained 26% of type I resistance (Steiner *et al.* 2004) and a QTL on 2B in cultivar Goldfield explained 29% of the phenotypic variation for FHB incidence (Gilsinger *et al.* 2005).

Breeding for FHB resistance in wheat

Conventional breeding strategies

The cost/gain effective way of controlling FHB disease in wheat is the use of FHB resistance. However, breeding FHB resistant wheat cultivars with desired agronomic traits has been difficult because of the polygenic control of FHB resistance, tight linkage of resistance QTL with undesired agronomic traits in the available resistance sources, laborious inoculation and disease evaluation procedures, and environmental effects on the resistance phenotype. In addition, at least three well-known types of FHB resistance are involved in wheat kernel invasion and DON accumulation. It still remains unknown whether these traits are under the control of common QTL (Bai and Shaner 2004). In last three decades, conventional breeding methods have succeeded in producing many wheat breeding lines with high levels of FHB resistance in wheat breeding programes of China and Japan, such as Sumai 3, Ning 7840, Tokai 63, and etc. (Bai *et al.* 2003; Ban 2000; Lu *et al.* 2001). Some of these lines have been widely used as resistant parents in wheat breeding programs in the United States, Europe, CIMMYT, and elsewhere (Buerstmayr *et al.* 2002; Mesterházy 2003; Rudd *et al.* 2001; Singh and van Ginkel 1997). However, since type II resistance of Sumai 3 alone may not provide adequate protection against

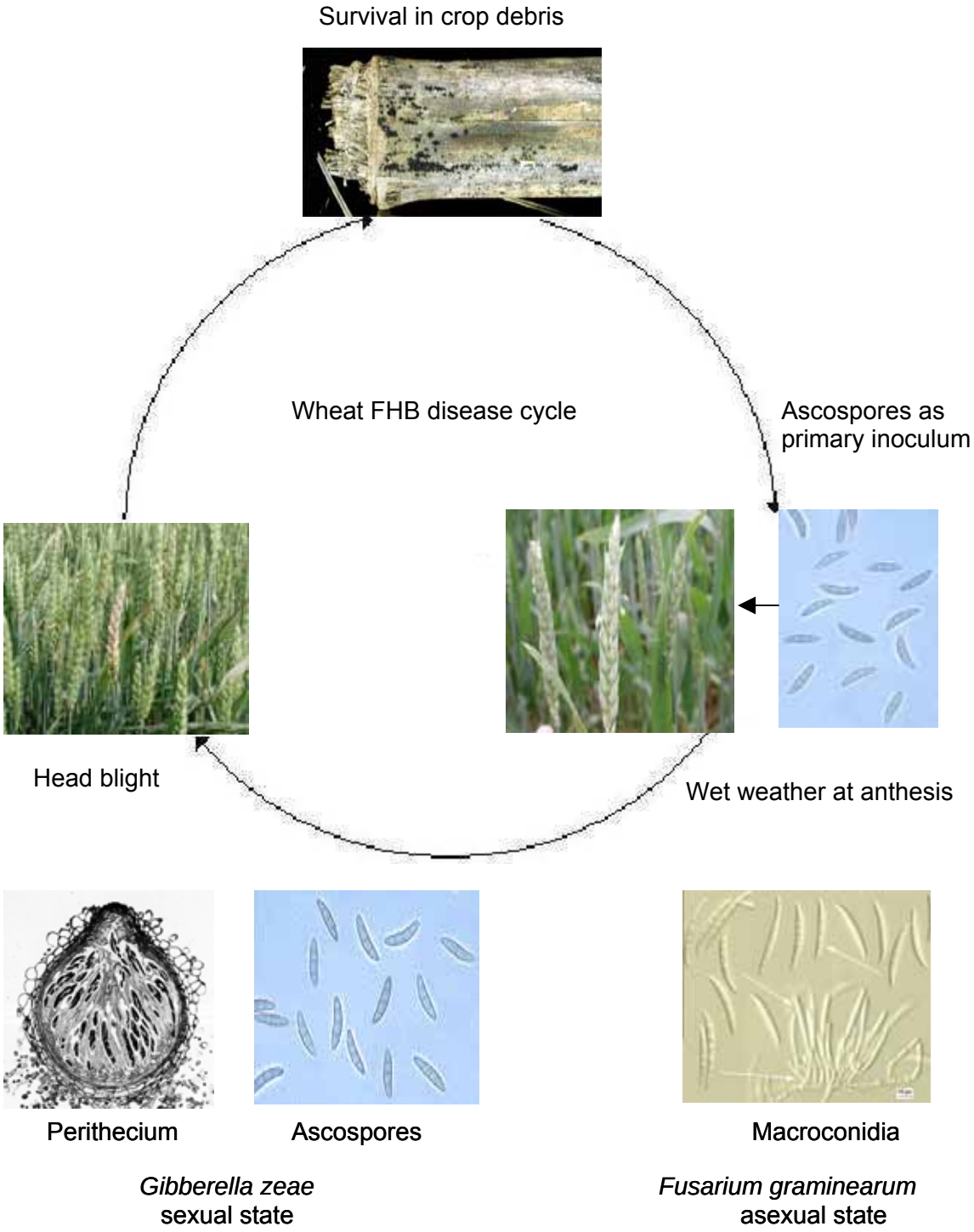
the fungal infection in severe FHB epidemics, most breeding programs now attempt to improve FHB resistance by recombining different sources and types of resistance and simultaneously selecting for resistance and desirable agronomic traits. Recurrent selection was reported to be successful for accumulating resistance genes of different types (Jiang *et al.* 1994).

Marker-assisted breeding

Breeding FHB-resistant wheat can be greatly facilitated by marker-assisted selection (MAS). Since FHB resistance appears to be controlled by a few major genes plus some minor genes, a nongenetic component of phenotype may make classification of individual plants in segregating generations uncertain (Bai and Shaner 2004). In contrast, once an association between a QTL and molecular markers is established, the QTL can be transferred into different genetic backgrounds through MAS. By use of MAS, selection for specific resistance gene(s) would eliminate susceptible materials earlier in the breeding process in order to combine different FHB resistance genes in a FHB-resistant cultivar.

Improving wheat FHB resistance with MAS is now being used in wheat breeding programs in Australia, Canada, and the USA with the help of the genotyping centers established in these countries. The effectiveness of the three SSR markers, *Xgwm389*, *Xgwm493*, and *Xgwm533*, which tightly linked to *Qfhs.ndsu-3BS*, was evaluated and validated in different populations (Chen *et al.* 2006; Yang *et al.* 2003). Molecular markers for FHB resistance can also be used for marker-assisted evaluation of FHB resistant wheat germplasm (Liu and Anderson 2003). Molecular markers linked to other minor FHB resistance QTL for type I resistance have also been reported (Bourdoncle *et al.* 2003a; Steiner *et al.* 2004), but they have not been widely used in MAS due to low repeatibility.

The routine use of MAS in wheat breeding programs for transferring QTL with major effects has not been extended to minor QTL. Because mapping methods remain insufficiently precise and QTL information is difficult to extrapolate from one mapping population to other breeding populations. MAS will remain a specialized breeding tool when the objective is to pyramid different genes or avoid difficult phenotypic screens, until QTL mapping can be extended to estimate breeding values across diverse breeding crosses and subpopulations such as those in typical plant breeding programs (Holland 2004).



Modified from http://www.apsnet.org/education/LessonsPlantPath/Fusarium/

Figure 1.1 Disease cycle of wheat FHB caused by *Fusarium graminearum/Gibberella zeae* and the pathogen's reproductive states

Table 1.1 QTL for FHB resistance in common wheat

QTL chromosome location	Marker / map interval	Resistance source	Reference
1A	*XmGTG.pAG225 – Xbarc28* ● *XFHBSTS1A-160* ●	Annong 8455 Ning 7840	Ma *et al*. 2006b Guo *et al*. 2006
1B	*Xbarc8 – Xgwm131* ● *XgluB1* ● *Xbarc312 – Xbarc302* ● *Xe38m50_10 – Xe32m65_10* ▲ *XP78M51_237 – XS26M23_365* ● *Xs12m25_14–Xs24m17_2* ▲ *Xgwm268 – Xwmc44* ● *XE38M52-378 – Xgwm131* ●	Fundulea 201R CM-82036 Wangshuibai Seri82 Lynx Remus Arina Cansas	Shen *et al*. 2003a Buerstmayr *et al*. 2002 Zhou *et al*. 2004 Mardi *et al*. 2006 Schmolke *et al*. 2005 Steiner *et al*. 2004 Paillard *et al*. 2004 Klahr *et al*. 2006
1D	*XS16M22-162 – Xwhs2001-1D* ▲	Ritmo	Klahr *et al*. 2006
2A	*Xgwm311 – Xgwm382* ● *XksuH16* ● *Xs13m26_4* ● *Xgwm614* ● *Xcfa2086 – Xgwm311* ● *Xgwm425-XmCCT.eAAG.2* ●	Renan Stoa Remus Ning 7840 Arina Wangshuibai	Gervais *et al*. 2003 Waldron *et al*. 1999; Anderson *et al*. 2001 Steiner *et al*. 2004 Zhou *et al*. 2002 Paillard *et al*. 2004 Ma et a*l. 2006*
2B	*Xgwm374* ● *XP74M53_272-XS25M12_206* ● *Xs13m25_8–Xs24m15_6* ▲ *Xbarc200 – Xgwm210* ▲ *Xgwm120* ● *Xs1021m* ● *Xgwm276b* ■	Renan Dream Frontana Goldfield Ning 7840 Nanda2419 Ernie	Gervais *et al*. 2003 Schmolke *et al*. 2005 Steiner *et al*. 2004 Gilsinger *et al*. 2005 Zhou *et al*. 2002 Lin *et al*. 2004 Abate and McKendry 2005
2D	*Xgwm296 – Xgwm261* ● *Xgwm539* ● *Xgwm539* ▲ *Xwmc144* ● *Xgwm261* ■ *Xglk302b – Xgwm539* ● *XmCGTA.pACT236 – XmACAG.pACT134* ● *XmCGAC.pTGC102–XmTGC.pTGC70* ● *Xgwm539 – Xs15/m24* ● *Xwmc181 – Xaf12* ▲ *Xwmc445-1 – Xgwm311-1* ▲ *Xgwm261 – Xgwm484* ● *Xgwm157 – Xgwm539* ●	Alondra Wuhan-1, CASS94 DH181 DH181 Maringa Arina Chinese Spring Chinese Spring Wangshuibai Wangshuibai Wangshuibai Wangshuibai CJ9306	Shen *et al*. 2003b Somers *et al*. 2003; Lewis *et al*. 2004 Yang *et al*. 2005b Yang *et al*. 2005b Somers *et al*. 2003 Paillard *et al*. 2004 Ma *et al*. 2006b Ma *et al*. 2006b Mardi *et al*. 2005 Lin *et al*. 2006 Lin *et al*. 2006 Jia *et al*. 2005 Jiang *et al*. 2005

▲ QTL for type I resistance; ● QTL for type II resistance; ■ QTL for type III resistance

Table 1.1 Cont.

QTL chromosome location	Marker / map interval	Resistance source	Reference
3A	*Xbcd941* ●	ND2603	Anderson *et al*. 1998
	Xgwm2 ●	Ning 7840	Gupta *et al*. 2000
	Xbcd372 ●	Recital	Gervais *et al*. 2003
	Xgwm5 ●	Huapei 57-2	Bourdoncle and Ohm 2003a
	Xgwm674 – Xbarc6 ●	Fundulea 201R	Shen *et al*. 2003a
	Xwmc264 – Xgwm155 ●	Arina	Paillard *et al*. 2004
	Xgwm720 – Xgwm112 ▲	Frontana	Mardi *et al*. 2006
	Xbcd941 ●	ND2603	Anderson *et al*. 2001
	Xdupw227 – Xgwm720 ▲	Frontana	Steiner *et al*. 2004
	Xwmc532 ●	Wangshuibai	Lin *et al*. 2004
	Xgwm369 – Xbarc045 ●	Wangshuibai	Jia *et al*. 2005
	Xwmc165 ▲	AC Foremost	Yang *et al*. 2005b
	XeagcMcta.1 ●	Sumai 3	Anderson *et al* 1998
	Xcdo981 ●	Sumai 3	Waldron *et al*. 1999
	Xgwm389 – Xgwm533 ●	Ning 7840	Gupta *et al*. 2000; Zhou *et al*. 2000
	Xgwm389 – Xgwm533 ●	W14	Chen *et al*. 2000
	Xgwm533 – Xgwm493 ●	Sumai3/ND2603	Anderson *et al*. 2001
		Chinese Spring	Ma *et al*. 2006b
	Xgwm533 – Xgwm493 ■	Maringa,	Somers *et al*. 2003
	Xgwm533 – Xgwm493 ●	CM-82036	Buerstmayr *et al*. 2002, 2003a
	Xbarc147 – Xgwm493■	CM-82036	Lemmens *et al*. 2005
	Xbarc133 ●	Huapei 57-2	Bourdoncle and Ohm 2003a
	Xbarc133 – Xgwm493 ●	Ning 894037	Shen *et al*. 2003b
	Xs23m15_3 ●	Remus	Steiner *et al*. 2004
	Xbarc147 – Xgwm493 ●	Ning 7840	Zhou *et al*. 2002
	Xbarc533 – Xgwm493 ●	Wangshuibai	Zhou *et al*. 2004; Mardi *et al*. 2005, Ma *et al*. 2006a; Jia *et al*. 2005
3B	*Xgwm533* ▲●	DH181	Yang *et al*. 2005b
	Xgwm493-Xgwm533-2 ●	CJ9306	Jiang *et al*. 2005
	E8M4_6■	Ernie	Abate and McKendry 2005
	Xgwm 533 ●	Chokwang	Yang *et al*. 2005a
	Xgwm533 – Xgwm493 ●■	W14	Chen *et al*. 2006
	Xbarc102-Xgwm533·1 ■	Wangshuibai	Ma *et al*. 2006a
	Xwmc054-1 ●	Wnagshuibai	Lin *et al*. 2004
	Xgwm533-3 ●	Wnagshuibai	Lin *et al*. 2004
	Xgwm247 ●	Huapei 57-2	Bourdoncle and Ohm 2003a
	Xwmc612 ▲	DH181	Yang *et al*. 2005b
	Xtam61 – Xgwm383 ●	Renan	Gervais *et al*. 2003
	Xgwm566 ▲	Maringa	Somers *et al*. 2003
	Xcfa2134b-Xgwm131b ●	Arina	Paillard *et al*. 2004
	Xbarc344 ●	Wnagshuibai	Zhou *et al*. 2004
	Xgwm533-2 – Xwmc054-1▲	Nanda2419	Lin *et al*. 2006
	XE35M59-107 – XE38M52-441 ●	Ritmo	Klahr *et al*. 2006

Table 1.1 Cont.

QTL chromosome location	Marker / map interval	Resistance source	Reference
3D	*Xgwm341 – Xgdm8* ●	Patterson	Shen *et al*. 2003a
	Xbcd907c – Xgwm161 ●	Arina	Paillard *et al*. 2004
	XE33M57-457 – Xgwm645 ●	Cansas	Klahr *et al*. 2006
4A	Xcdo545 – Xgwm160 ●	Arina	Paillard *et al*. 2004
	Xgbx3480b – Xbcd907g ●	Arina	Paillard *et al*. 2004
	mGCG.pGTG223-Xwmc617.2 ●	Annong 8455	Ma *et al*. 2006b
	Xwmc501-2 – Xwmc161 ▲	Nanda2419	Lin *et al*. 2006
4B	*Xwmc238* ▲	Wuhan-1	Somers *et al*. 2003
	Xwg909 ●	Stoa	Waldron *et al*. 1999, Anderson *et al*. 2001
	Xs13m25_9 ▲	Frontana	Steiner *et al*. 2004
	Xwmc349 – Xgwm149 ▲	Wangshuibai	Lin *et al*. 2006
	Xgwm513 – Xbarc20 ▲	Wangshuibai	Lin *et al*. 2006
	Xgwm368 – Xgwm149 ●	Wangshuibai	Jia *et al*. 2005
	Xgwm495 ●■	Ernie	Abate and McKendry 2005
	Xbarc 1096 ●	Chokwang	Yang *et al*. 2005a
4D	*Xcfd84-Xwmc331* ●	Chinese Spring	Ma *et al*. 2006b
	Xwmc331 ▲	DH181	Yang *et al*. 2005b
5A	*Xgwm156* ●	Ning 7840	Gupta *et al*. 2000
	Xgwm293 – Xgwm304 ●	CM-82036	Buerstmayr *et al*. 2002, 2003a
	Xgwm415 – Xgwm304 ●	Patterson	Shen *et al*. 2003a
	Xgwm96 ■	Maringa	Somers *et al*. 2003
	Xgwm186 – XmCCA.eAAG.2 ●	Wangshuibai	Ma *et al*. 2006a
	Xgwm293 ▲	DH181	Yang *et al*. 2005b
	Xpsr170a ●	Renan	Gervais *et al*. 2003
	Xgwm639b ●	Renan	Gervais *et al*. 2003
	B1 ●	Renan	Gervais *et al*. 2003
	Xgwm129–Xbarc197 ●	Frontana	Steiner *et al*. 2004
	Xs23m20_8 ●	Frontana	Steiner *et al*. 2004
	Xgwm291 – Xglk348c ●	Arina	Paillard *et al*. 2004
	Xfbb166a – Xpsr426 ●	Arina	Paillard *et al*. 2004
	XmCCA.eAAG.2 – Xgwm156 ■	Wangshuibai	Ma *et al*. 2006a
	Xgwm304 – Xbarc56 ▲	Wangshuibai	Lin *et al*. 2006
	Xbarc117 – Xbarc186 ▲	W14	Chen *et al*. 2006
	Xbarc56 ●■	Ernie	Abate and McKendry 2005
5B	*Xbarc59* ●	Patterson	Bourdoncle and Ohm 2003a
	Xgwm371 – Xpsr120a ●	Forno	Paillard *et al*. 2004
	Xpsr1201 – Xgwm371 ●	Forno	Paillard *et al*. 2004
	Xgwm1246 – Xpsr145a ●	Forno	Paillard *et al*. 2004
	Xgwm408 – Xbarc140 ▲	Wangshuibai	Lin *et al*. 2006
	Xgwm335 – Xgwm371 ●	Wangshuibai	Jia *et al*. 2005
	XE35M52-331 – XS25M20-245 ●	Cansas	Klahr *et al*. 2006
	Xbarc74-Xbarc408 ●	CJ9306	Jiang *et al*. 2005

Table 1.1 Cont.

QTL chromosome location	Marker / map interval	Resistance source	Reference
5D	*Xcfd29* • *Xgwm190 – Xgwm358* • *Xbarc 239* •	Renan Alondra Chokwang	Gervais *et al*. 2003 Jia *et al*. 2005 Yang *et al*. 2005a
6A	*XP77M51_430 – XS66M55_242* • *XksuH4* • *Xgwm169 – Xpsr966b* • *XmCTG.pACTG134 – XmCTG.pACT132* •	Dream Sumai 3/ND2603 Arina Chinese Spring	Schmolke *et al*. 2005 Anderson *et al*. 2001 Paillard *et al*. 2004 Ma *et al*. 2006b
6B	*Xbcd331* • *Xcdo524* • *Xgwm88 – Xgwm644* • *Xbarc101 – Xbcd1383* • *Xs23m14_4* ▲ *Xwmc539* • *Xgwm133 – Xgwm191* • *Xwmc397* ▲•	Sumai 3 Sumai 3 Ning 894037 Sumai 3/ND2603 Frontana Wangshuibai Wangshuibai DH181	Waldron *et al*. 1999 Waldron *et al*. 1999 Shen *et al*. 2003b Anderson *et al*. 2001 Steiner *et al*. 2004 Lin *et al*. 2004 Jia *et al*. 2005 Yang *et al*. 2005b
6D	*Xcfd42* • *Xgwm469* • *Xcfd19a – Xcfd47* • *Xpsr915 – Xcfd19a* • *Xcfd19b – Xgdm14b* •	Renan Nanda2419 Arina Arina Arina	Gervais *et al*. 2003 Lin *et al*. 2004 Paillard *et al*. 2004 Paillard *et al*. 2004 Paillard *et al*. 2004
7A	*Xwms1083* *Xe77m47_22–Xgwm233* ▲ *Xwmc338-2 – Xwmc83* ▲ *Xgwm276 – Xgwm282* • *XS23M21-271 – XS18M22-369* •	Wangshuibai Frontana Nanda2419 Wangshuibai Ritmo	Zhou *et al*. 2004 Mardi *et al*. 2006 Lin *et al*. 2006 Jia *et al*. 2005 Klahr *et al*. 2006
7B	*XS25M15_187-XS23M21_497* • *Xbarc126-2 – Xwmc476* ▲ *Xgwm146 – Xgwm611* • *Xwmc526* • *Xgwm46 – XE42M58-394* •	Dream Nanda2419 Alondra DH181 Cansas	Schmolke *et al*. 2005 Lin *et al*. 2006 Jia *et al*. 2005 Yang *et al*. 2005b Klahr *et al*. 2006
7D	*Xgwm437 – Xwmc488* ▲	Nanda2419	Lin *et al*. 2006

Table 1.2 FHB resistance sources in common wheat and other *Triticeae* species

Species	Origin	Genotype	Reference
Triticum aestivum	East Asia	AT2, Baihuamai, Baisanyuehuang, Caizihuang, Canlaomai, DaBaiPao, Dafaliuzhu, Dahongpao, Dataibo, Emai 14, Emai 6, F 5114, F 5125, F 60096, Fanshanxiaomai, FSW, Fumai 3, Fusuihuang, Haiyanzhong, Heshangmai, Hongheshang, Hongjianzi, Hongmai Hongxumai, Huangcandou, Huangfanzhu, Huapei 32-2, Huoshaobairimai, Huoshaomai, JC-3, JC-6, JG 1, Jiangzhou 1, Jianzimai, Lunhui 201, MaZhaMai, Mianyang 96-12, N894013, N894037, N962424, N983222, N991069, N991119, N991126 N991130, Nanda 2419, Ning 7840, Ning 7849, Ning 8026, Ning 8102 Ning 8331, Ning 8343, Ning 894013, Ning 894037, Ning 920292, Ning 962424, Ning 983222, Ning 991069, NTDHP, PC-2, Sanjianxiaomai, Sanyuehuang, SH19089, ShanghaiCaiZiHuang, Shanghai 3, Shenmai 2, Sumai 2, Sumai 3, Sumai 49, Taiwan Xiaomai, Wangshuibai, Wannian 2, Wuanmai 38, Wugongmai, Wuhan 1, Wuhan 2-37E, Wuhan 3, WZHHS, Xiangmai 1, Xiangmai 2, Xueliqing, Yangmai 1, Yangmai 4, Yangmai 5, Yangmai 9, Yangmai 158, Youmangmai, Zhen 7495	Liu 1984; Snijders 1990; Lu *et al*. 2001; Bai *et al*. 2001, 2003; Liu and Anderson 2003; McCartney *et al*. 2004; Yu *et al* 2006; Yang *et al*. 2006
		PingHuJianZiMai, HongHuDaTaiBao, ChongYangHongMai, anGangFangZhu	Yu 1991
		Ningmai 7, Longmai 19,Chuanmai 25	Gilchrist *et al*. 1997
		W14, CJ8806, TFSL037, CJ8805, CJ9047, CJ9049, CJ8809, HuW16, Emai 9	Jiang *et al*. 1997
		Shaan85-2, Futai 8944, Futai 9002, W14, Shaan 85, Changjiang 8809, Sho Chou	Griffey *et al*. 1998, 1999; Murphy *et al*. 1999
		Ning 89401, Ning 894037, Ningmai 9, Mutant AT 1, Mutant AT 2, Shengkang 1, Zhonghua 1, 85004/Mexico 354, SB 107, SB108, SB109, SB110, SB111, SB114, SB115, SB116	McKendry 2000a
		Shu Chou Wheat No. 3, Manchurian	Zhang *et al*. 2001

Table 1.2 Cont.

Species	Origin	Genotype	Reference
Triticum aestivum	East Asia	HC 540, Ning 68331, Wong Ju, H 281	Sun *et al*. 2002
		Yanzisanyuehuang, Qianxihuanglamai, Jishachangmangmai, Chikeguangtoumai, Pingbaniqiumai, Qingzhendatouhuang, Huangkeguangtoumai, Zaomangmai, Baiyuhua, Shuilizhan, Sanyuehuang, Huoshaotian, Huoshaomai, Jiulan, Changmangmai, Baipuxiaomai, Ning7640, Yibinwuyangmai	Wei *et al*. 2005
		CJ9306, CJ9403	Jiang and Ward 2006
		Saikai 165	Ban and Suenaga 2000
		Japon 2, Fujimi Komugi, Norin 43, Gogatsu-Komugi, Chuko, Froment Du Japon, Norin 61	Zhang *et al*. 2001
		Norin 50, Norin 96, Cltr 9506, Cltr 9507, Xin Dong No. 2, Ling Hai Mao Yang Mo, Yang La Zi, Seu Seun 6	McKendry *et al*. 2002
		Abura Komugi, Asakaze Komugi, Aso Zairai, Aso Zairai II, Chile, Chokwang, Itou Komugi, Kagoshima, Kikuchi, Nobeoka Bozu, Nobeokabozu Precoce, Nobeokabouzu Komugi, Nyu Bai, Qiaomai Xiaomai, Sanshukomugi, Sapporo H.K.J., Shinchunaga, Shirasaya No1, Shiro Nankin, Shou Komugi II, Soba Komugi IB, Soba Komugi IC, Sotome, Sotome A, Tokai 66, Yanggangfangzhu, Zairai Yuubou	Snijders 1990; Ban 2000; Bai *et al*. 2001, 2003; Liu and Anderson 2003; McCartney *et al*. 2004; Yu *et al* 2006; Yang *et al*. 2006
		Hayakomugi, Soujyukeakage, Norin 59, Norin 52, Norin 36, Norin 20, Norin 61, Tokai 62, Tokai 63, Saikai 95, Asakazekomugi, Saikai 104, Shiroganekomugi	Ban 2003
		Shiro Komugi, Okinawa Zairai Yuubou, Aso Zairai (Mubou), Ooita Komugi, Chikuzen,	Mckendry *et al*. 2004
	Europe	Erythrospermum 3086-30M	Schroeder and Christensen 1963
		Fundulea 29, F549, F 308, F 574	Ittu *et al*. 1994, 1997

Table 1.2 Cont.

Species	Origin	Genotype	Reference
Triticum aestivum	Europe	Arina, Praag-8, Kooperatorka, Arminda, Marathon, SVP 72017-17-5-10, Novokrumka 0102	Snijders 1990; Buerstmayr *et al*. 1996; Mentewab *et al*. 2000
		Senta, Sparta, Branka, Sofia	Stuchlikova and Sip 1996
		U-136.1	Buerstmayr *et al*. 1999
		VR95B717, VR95B295	Griffey *et al*. 1998, 1999
		81-F3-79, Arina, SVP-72017-17-5-10, SVP-C8718-5, UNG-136.1, UNG-226.1	Buerstmayr *et al*. 1999, 2000
		Mentana, NS 18-99, Luizia Strampelli, Inallettabile 3	Murphy *et al*. 1999
		Fundulea 01 R, Fundulea 183 P5, Fundulea 483, Fundulea 143-T3-103, Turda 95, Turda 195, Turda 2317-90, PI 221346, PI 345022, PI 350033, PI 350089, PI362463, PI 362676	McKendry 2000a, McKendry *et al*. 2000b
		193C, 69Z108.42, Prodigio Italiano, Chudoskaja, Newthatch Sel., 220, Estanzuela Young, Renacimiento, 274-1-118, III/14-B, Cluj 49-926, Belgrade 4, Academia 48	Zhang *et al*. 2000, 2001
		Cologna Veneto, Colorben 4, Quaderna, Trento	McKendry *et al*. 2002
		Academia 48, Artemowska, Belgrade 4, Chudoskaja, Prodigio I., 220, 193C, 69Z108.42, Cluj 49-926, III/14-B, Kooperatorka, Odesskaja 13, Stepnjachka	Liu and Anderson 2003
		Spartakus, Karat, Poncheau, Perlo, Livus, Extrem, Expert	Bai *et al*. 2001, 2003
		Bizel	Bourdoncle and Ohm 2003b
		Kimon, Fundulea 4, Arina, Orestis, F143 T3-103, Bussard, Kontrast, Bold, F-29, F 569U1-1, Ronos, F 92392 G3-3, Atlantis, F 91063 G3-21, Piko, F 508 U3-2, Turda-95, F 249T2-10U, Fundulea 201-R	Miedaner *et al*. 2003b
		14-3-C, Kooperatorka, Novokrymka	McCartney *et al*. 2004
		IPG-SW-14, IPG-SW-22, IPG-SW-28 IPG-SW-29, IPG-SW-30, IPG-SW-41, IPG-SW-44, Jasna, Santa, Eta, Henika, Omega, Ismena	Wiśniewska *et al*. 2004
		Hana, Samanta, Blava, Torysa, Regia, Košutka	Pavlová and Šrobárová 2004

Table 1.2 Cont.

Species	Origin	Genotype	Reference
Triticum aestivum	Europe	Biscay, Claire, Soissons, Solstice, Wizard, Consort, Deben, Einstein, Buchan	Browne and Cooke 2005
	North America	Ernie Freedom	McKendry *et al*. 1995 Gooding *et al*. 1997
		H821, HC374, H181, H185, H192, IL 95-1549, FHB143	Griffey *et al*. 1998, 1999
		NC96-13374, NC96-14629, NC96-13965	Murphy *et al*. 1999
		P92823A1-1-4-4-5, Agripro Foster, Coker 9803, IL94-1549, VA93-54-429, Agripro Patton	Chappell *et al*. 1999
		P93D1-10-2, IL 95-1966, IL 9634-24851, Roane, Bacup, IL 93-2283, IL 95-2066, IL 95-2909, Poncheau, OH569	Bai *et al*. 2001, 2003
		AW 495, 29AA28, AC Drummond, AW 478, AW 499, QW 628.5 Coker 9474, Truman, Foster, Patton, Roane, Hondo, Heyne, Wesley, Goldfield, McCormick, Tribute, Neuse, INW0304, IL 94-1653, Cecil, INW0411, Bess, NY88046-8138, Coker 9511, WestBred X00-1079, KS04WGRC48	Sun *et al*. 2002 Griffey 2005 Brown-Guedira *et al*. 2005
		Steele-ND	Mergoum *et al*. 2005;
		Roane, McCormick, NC-Neuse, Pat, VA01W476, B980582, B980416, B961378, Allegiance, Rcatl33, Patterson	Browne *et al*. 2005 Abate *et al*. 2006
	South America	Frontana, Encruzilhada, FT 83-326	Schroeder and Christensen 1963; Snijders 1990; Singh *et al*. 1995; Bai *et al*. 2003
		16-52-9, 274, Estanzuela Young, Renacimiento, 274-1-118, Tezanos Pintos Precoz, Centenario, Excelsior, Bahiense, Trintecinco, Rio Negro, PF 79782, Colotana 266/51, 16-52-2	Zhang *et al*. 2000, 2001
		113.92, 111.92, 117.92, Cooperacion Cabildo, Vilela-Sol, 38 M.A.	Bai *et al*. 2001, 2003

Table 1.2 Cont.

Species	Origin	Genotype	Reference
Triticum aestivum	South America	Abura, CItr 17427, CItr 5103, Excelsior, Colotana 266/51, Tezanos P.P., 16-52-9, PF79782 BRS 177, BRS 179, BRS Timbaúva, BRS Louro, BRS Umbu, BRS Camboim, BRS Tarumã	Liu and Anderson 2003 Lima *et al*. 2004
Triticum aestivum ssp. *macha*		JIC Acc. 1240001, HsTm4A	Grausgruber *et al*. 1998 Steed *et al*. 2005
Aegilops tauschii	Afghanistan		Gagkaeva 2003
Aegilops speltoides			Fedak *et al*. 2004
Aegilops sharonensis	Israel		Olivera *et al*. 2003
Triticum karamyschevii	Georgia		Gagkaeva 2003
Triticum monococcum			Fedak *et al*. 2004
Triticum militinae	Georgia		Gagkaeva 2003
Triticum persicum	Dagestan		Gagkaeva 2003
Triticum spelta	Switzerland		Gagkaeva 2003
Triticum turgidum L. var. *dicoccoides*	USA Israel	PI 272582, PI 466995, PI 343446, PI 362328 Mt. Gerizim-36, Mt. Hermon-22	Miller *et al*. 1998; Otto *et al*. 2002 Ban and Watanabe 2001 Buerstmayr *et al*. 2003b
Triticum turgidum subsp. *carthlicum*	USA	PI 283890, PI 94748, PI 352281, PI 94754, PI 286070, PI 61102	Oliver *et al*. 2005 Chen *et al*. 2005a
Triticum turgidum subsp. *dicoccum*	USA Germany	PI 79899, PI 41025, CI 14135, CI 7686	Oliver *et al*. 2005 Chen *et al*. 2005 Gagkaeva 2003
Triticum timopheevii subsp. *armeniacum*	USA Georgia	TA960	Brown-Guedira *et al*. 2005 Gagkaeva 2003
Hordeum vulgare L.		Chevron, Gobernadora, CMB 643, Hietpas 5, Fredrickson	de la Peña *et al*. 1999; Mesfin *et al*. 2003, Lamb *et al*. 2001
Agropyron cristatum	Romania	PI297870	Wan *et al*. 1997
Elymus fibrosa	Russia	PI43999	Wan *et al*. 1997
Elymus humidus	Japan	AG.91-35	Ban 1997; Fedak 2000 Cai *et al*. 2005
Elymus racemifer	Japan	AG.91-24	Ban 1997

Table 1.2 Cont.

Species	Origin	Genotype	Reference
Elymus giganteus (*Leymus racemosus*)			Mujeeb-Kazi *et al*. 1983 Chen *et al*. 1993, 2005 Chen and Liu 2000
Elymus tangutorum	China	NWC15-818-2	Wan *et al*. 1997
Elymus trachycaulus	Poland	Pr234	Wan *et al*. 1997
Hystrix duthiei	China		Wan *et al*. 1997
Leymus secalinus			Jauhar and Peterson 1998
Leymus angustus			Jauhar and Peterson 1998
Lophopyrum elongatum	USA		Shen *et al*. 2004
Psathyrostachys juncea	China	Y1603	Wan *et al*. 1997
Psatyrostachys huashanica	China		Cai *et al*. 2005
Roegneria ciliaris	China	Pr176, Pr166, Y83008	Wan *et al*. 1997
Roegneria ciliaris var. *japonensis*	China	Y83009, H19, Pr229, Pr330	Wan *et al*. 1997
Roegneria stricta	China	Y0936	Wan *et al*. 1997
Roegneria tsukushiensis var. *transiens*	China	Pr208, Pr219, Pr221, Pr237	Wan *et al*. 1997
Roegneria ciliaris	China	Pr247, Pr249	Wan *et al*. 1997 Chen and Liu 2000
Roegneria kamoji	China		Weng and Liu 1991 Chen and Liu 2000
Thinopyrum intermedium			Cai *et al*. 2005
Thinopyrum nodosum			Jauhar and Peterson 1998
Thinopyrum junceiforme		PI 414667	Jauhar and Peterson 2001
Thinopyrum ponticum			Cai *et al*. 2005

CHAPTER 2 - Identification of new FHB resistance sources from Asian wheat germplasm

Introduction

Identifying resistance genes and understanding the complex genetic structure of FHB resistance will greatly enhance wheat breeding for FHB resistance. FHB-resistant cultivars have been identified from different geographic regions (Chapter 1). Type II resistance is the predominant type of resistance in most of the varieties identified so far and a few resistance sources have been extensively used in breeding programs worldwide (Chapter 1), but other types of resistance sources and new type II resistance sources are needed to broaden genetic diversity and improve FHB resistance in wheat cultivars.

The relationship between FHB severity and DON content in harvested grain is uncertain. A negative correlation between FHB resistance and DON contamination was observed in some studies (Lemmens *et al.* 1994; Wang and Miller 1988, Bai *et al.* 2001), but not in others (Mesterházy *et al.* 1999; Wiśniewska *et al.* 2004). The discrepancy among studies may be due to factors such as the stage of plant development during FHB infection, the tissue and assay method used for DON evaluation, threshing method, and the degree of FHB resistance of evaluated genotypes. More studies are needed to reveal the relationships between DON accumulation and FHB severity resistance (type I and II).

In the present study, wheat genotypes with different levels of FHB resistance and origins were evaluated for FHB resistance under favorable FHB epidemic conditions in the greenhouse. The objectives were to identify new germplasm that expresses different types of FHB resistance and to explore the relationships among different types of FHB resistance.

Materials and methods

Plant materials and pathogen inoculum

Ninety-five landraces, cultivars, and breeding lines were evaluated for three types of resistance in repeated experiments. Among these accessions, 64 Chinese accessions were

provided by Jiangsu Academy of Agricultural Sciences, Nanjing, China and the National Small Grains Facility at Aberdeen, Idaho, USA; 24 Japanese accessions were provided by the Gene Bank of Japan; one Korean cultivar was obtained from Purdue University, Indiana, USA. The remaining six cultivars from the USA and other countries were used as references (Table 2.1). The inoculum was a field isolate of *F. graminearum* (GZ 3639) originating in Kansas. This isolate has been well characterized for its strong virulence and high quantity of DON production (Desjardins *et al*. 1996).

FHB evaluation

The FHB severity was evaluated in the greenhouse at Kansas State University from 2003 to 2005. To evaluate type II resistance, ~1000 conidiospores of *F. graminearum* were injected with a syringe into a central floret of a spike at anthesis. Each wheat spike was sprayed with 2 ml of liquid culture of *F. graminearum* (~500 conidiospores/ml) to evaluate type I resistance (Fig 2.1). The plants were prepared for inoculation as follows: after vernalization at 4ºC in a growth chamber for eight weeks, six seedlings were transplanted into a 5'x 5' Dura-pot (Hummert Int., St. Louis, MO) containing a soil mix (Hummert Int., St. Louis, MO), and grown in a greenhouse with 12-h daylight period. All plants in each pot were inoculated in a single head per plant, and incubated in a moist chamber for three days to initiate infection (Fig 2.1). The inoculated plants were then moved to their original bench positions and grown at 25ºC day and 22ºC night temperature. Type I resistance was evaluated by counting the infected and total numbers of florets in an inoculated spike on the seventh day after inoculation (7 d.a.i.) and calculating the proportion of infected florets (PIF). For type II resistance, the infected and total numbers of spikelets in an inoculated spike were counted at 21 d.a.i. and the proportion of symptomatic spikelets (PSS) was calculated as final disease severity. Mean values of the disease readings were used in the statistical analysis. Type I resistance was evaluated in two experiments (2004–2005) and type II resistance was evaluated in two experiments carried out in 2003 spring and fall with three replications in each experiment.

DON determination

For each accession, inoculated heads were carefully collected and threshed manually to save all grains including highly infected, shriveled, and degraded ones. Grains from the three replicates of a genotype in each experiment were combined for toxin analysis. DON content was

determined as the amount of DON (mg/kg, parts per million or ppm) in the wheat kernels harvested from *Fusarium*-inoculated spikes by gas chromatography–mass spectrometry (GC–MS) (Mirocha *et al.* 1998).

Statistical analysis

Disease scores on percentage of infected florets (PIF), proportion of symptomatic spikelets (PSS), and DON content were analyzed statistically. All analyses were based on plot means. Entry means of FHB severities followed a normal distribution and were used directly for statistical analysis. However, entry means of DON contents deviated from normality and were normalized by logarithmic transformation for further statistical analysis. Simple coefficients of correlation were calculated to estimate the relationships among PIF, PSS, and DON content. All statistical analysis was conducted with SAS software (SAS Institute, Inc., Cary, NC).

Results

Variation in three types of resistance among the wheat germplasm

All inoculated wheat landrace/cultivars showed FHB symptoms after single-point or spray inoculation. Significant differences among the wheat lines in PIF, PSS, and DON contents were observed among the accessions with mean PIF ranging from 9.1% (Nanda 2419) to 46.5% (ChanjiBaiDongMai), mean PSS ranging from 6.6% (Ning 7840) to 92.2% (Sanyuehuang), and mean DON contents ranging from 0.7 (Ning 7840) to 77.9 ppm (ChanjiBaiDongMai) (Table 2.1). Both PIF data and DON measurements showed poor correlation between experiments. Also, the PIF and DON data showed poor correlations between experiments. However, the PSS and DON data showed correlation between the two single-floret inoculation experiments in 2003 ($r = 0.61$, $P < 0.0001$).). In addition, the PSS and DON content were correlated in both single floret and spray inoculation experiments with correlation coefficients ranging from 0.27 to 0.71 (Table 2.2).

Based on their type II resistance, the tested wheat accessions could be separated into four categories: resistant (R), moderately resistant (MR), moderately susceptible (MS), and susceptible (S) lines. They accounted for 8.4%, 40%, 23.2%, and 8.4% of the wheat accessions evaluated, respectively (Table 2.3). Most of the accessions (63.2%) were MR or MS. The mean PSS for the four categories were significantly different with 15.6% for R, 37.3% for MR, 62.5%

for MS, and 83.1% for S. DON contents for the accessions in four different categories were 4.0, 8.3, 14.5, and 24.9 ppm, respectively. However, the differences were not significant for type I resistance among the four categories with mean PIF of 24.4%, 27.3%, 28.5%, and 27.7%, respectively (Table 2.3). Under the same conditions, the susceptible control Wheaton had PSS of 89.2%, DON of 84.5 ppm, and PIF of 28.2%, while the resistant control Sumai 3 had PSS of 18.8%, DON of 4.1 ppm, and PIF of 28.1%.

Among the 64 resistant or moderately resistant accessions, all, except the two USA cultivars (Ernie and Freedom), were landraces/cultivars from China or Japan. Seventeen of the 26 resistant accessions, including Ning 7840, F 60096, and Minamikyushu 69, showed lower mean DON accumulation than the control cv. Sumai 3 and nine of the 17 genotypes had less than 2 ppm DON in harvested diseased grains. Among all examined wheat genotypes, only six Chinese and Japanese accessions (Huoshaobairimai, NobeokaBozu, Asozaira III, Huang Fang Zhu, Huoshaomai, and Huangcandou) were found to have very low FHB incidence and severity (PIF < 28% and PSS < 25%) and low DON content (DON < 2 ppm), values superior to those of the resistant control Sumai 3 (Table 2.1). Five of the six highly resistant wheat landraces, Huoshaobairimai, Asozaira III, Huang Fang Zhu, Huoshaomai, and Huangcandou, are new and have not been thoroughly studied.

Association between DON contents and FHB severity

Wheat genotypes with low DON accumulation often had low PSS (Table 2.1 and 2.3). Positive correlation was observed between PSS and DON accumulation ($r = 0.50$, $p < 0.0001$) (Fig 2.1). Most (14 of 15) genotypes with low DON content had PSS less than 50% and nine of the 15 accessions had a PSS value less than 25%, including the well-known FHB-resistant lines Ning 7840, NobeokaBozu, and Wangshuibai. The average DON content for susceptible wheat accessions was 24.9 ppm and none had less than 2 ppm of DON (Table 2.1 and 2.3).

There was no correlation between PIF and DON accumulation ($r = 0.02$, $P = 0.85$) (Fig 2.1). Fifty-two accessions had lower PIF (< 28%) than both the resistant control (Sumai 3) and the susceptible control (Wheaton). Wheat accessions with low PIF were from all four FHB resistance categories, and even susceptible lines such as Sanyuehuang and TaFangShen showed lower PIF than that of the resistant control Sumai 3.

Discussion

In this study, most of the resistant wheat accessions were Chinese or Japanese wheat cultivars or landraces, except Ernie from the USA. Nine of the twenty-six accessions had less than 2 ppm DON accumulation and most have no known genetic relationship with Sumai 3 except Ning 7840 and Yangmai 1, suggesting that these wheat lines may have novel FHB resistance and low DON accumulation. It is rare for a wheat accession to be superior in both FHB severity and DON accumulation. Only six wheat accessions in this study showed a high level of resistance for all three types.

Results showed that type II resistance was significantly correlated with DON content. However, no correlation was observed between type I resistance and DON content, which agrees with the result of Lemmens *et al.* (2005) and others (Mesterházy *et al.* 1999; Wiśniewska *et al.* 2004). In general, moderately resistant and moderately susceptible accessions had higher DON content in harvested grain than resistant accessions with only a few exceptions.

Mild visual FHB symptoms on the infected spikes (type II resistance) usually indicates low DON contamination, but visual FHB symptoms may not always correlate with the levels of DON content for wheat lines with moderate FHB type II resistance. Fifteen lines in this study had DON content less than 2 ppm, and six of them were moderately resistant or moderately susceptible, showing PSS values from 26.3% to 71.4%. Wheat landrace KuangTuErhHsiaoMai was moderately susceptible with 71.4% PSS, but showed low DON content of 1.6 ppm. In contrast, Chinese landrace MeiQianWu showed low percentage of infected spikelets (16.1%) but relatively high (9.5 ppm) DON content.

It appears that three types of FHB resistance are present in these wheat lines and assessment of individual resistance components in these sources will facilitate their use in breeding FHB-resistant wheat cultivars. Resistance to FHB is most effective when a wheat line carries all the three types of resistance (Kolb *et al*. 2001). Different resistance sources identified in this study may possess different resistance genes that can be pyramided in improved lines.

Conclusions

In all ninety-five wheat landraces and cultivars were evaluated for the three types of FHB resistance. Deoxynivalenol content was significantly correlated with type II resistance, but not type I. All accessions could be classified into four categories based on their observed type II resistance. About 69% of the accessions were resistant or moderately resistant. Among them, 26 resistant accessions originated mainly from China and Japan, 15 had a DON content less than 2 ppm, and six Chinese and Japanese lines (Huoshaobairimai, NobeokaBozu, Asozaira III, Huang Fang Zhu, Huoshaomai, and Huangcandou) showed high levels of resistance for all three types of resistance. Since most of these resistant accessions do not have Sumai 3 in their pedigrees, they may carry QTL for FHB resistance and low DON accumulation different from those in Sumai 3.

Table 2.1 Mean values of visual disease ratings and DON contents for wheat accessions from different origins based on greenhouse-grown plants

Landrace/cultivar	Country	Source	PIF (%)		PSS (%)		DON (ppm)		Rating[†]
			Mean	Range	Mean	Range	Mean	Range	
Ning 7840	China	JAAS	30.1	22.7 - 45.0	6.6	5.3 - 16.3	0.7	0.1 - 1.5	R
Huoshaobairimai	China	JAAS	17.8	1.3 - 41.7	22	5.7 - 58.3	1.3	0.3 - 2.6	R
NobeokaBozu	Japan	PI382153	24.5	4.0 - 45.7	24.3	5.7 - 51.1	1.5	0.3 - 2.7	R
Asozaira III	Japan	JIRCAS	23.6	12.7 - 41.9	8.9	5.9 - 14.5	1.5	0.3 - 3.7	R
Huang Fang Zhu	China	JAAS	24.1	11.0 - 42.1	20.2	7.8 - 50	1.6	0.8 - 2.1	R
AsoZairai (YuubouKappu)	Japan	JIRCAS	32.1	2.0 - 43.1	12.7	6.1 - 23.1	1.6	0.3 - 3.0	R
Huoshaomai	China	JAAS	17.3	8.7 - 25.9	21.4	6.2 - 32.7	1.7	0.2 - 4.8	R
Huangcandou	China	JAAS	12.2	2.1 - 33.0	13.4	6.5 - 20.7	1.7	0.3 - 2.8	R
Fu 5114	China	JAAS	33.2	4.1 - 77.0	7.4	5.4 - 9.4	1.8	0.3 - 4.7	R
Qiaomai Xiaomai	Japan	JIRCAS	40.0	34.0 - 48.0	22.4	5 - 51.6	2.1	0.5 - 4.7	R
Baisanyuehuang	China	JAAS	23.3	4.3 - 44.7	12.5	6.4 - 22.1	2.3	1.3 - 3.3	R
F 60096	China	JAAS	20.9	7.7 - 38.0	6.9	5.8 - 8.8	2.6	0.3 - 5.7	R
Yangmai1	China	JAAS	23.6	4.3 - 34.3	22.5	7.3 - 60.1	2.6	0.1 - 6.9	R
Minamikyushu 69	Japan	PI382152	31.7	20.7 - 48.3	7.4	6.7 - 9.1	2.9	1.7 - 4.7	R
Wangshuibai	China	JAAS	15.7	1.6 - 32.7	8.7	6.6 - 16.4	3.6	0.3 - 9.5	R
MuTanChiang	China	PI70675	25.1	3.0 - 54.3	21.4	16.4 - 26.3	3.7	0.3 - 7.1	R
Haiyanzhong	China	JAAS	20.9	2.0 - 37.8	13.7	7.5 - 25.8	3.8	0.2 - 8.6	R
Fumai 3	China	JAAS	21.3	5.3 - 29.0	18.6	6.8 - 48.9	4.2	0.2 - 10.2	R
LingHaiMaoYangMo	China	PI435124	23.1	7.7 - 36.7	9.3	6.5 - 18.1	4.3	0.3 - 13.7	R
Taiwan Wheat	China	JAAS	23.8	1.3 - 49.3	13	10.4 - 16.6	6.0	0.3 - 21.6	R
Caizihuang	China	JAAS	28.8	18.3 - 37.7	22	6.3 - 34.5	6.6	0.4 - 9.3	R
Chukoku 81	-	-	27.0	3.3 - 45.3	19.5	9.1 - 38.9	8.4	0.2 - 16.9	R
Shirasaya No1	Japan	PI197129	19.8	1.6 - 64.3	22.4	7.3 - 44.5	8.6	1.3 - 19.4	R
Su 49	China	JAAS	20.3	4.0 - 33.3	7.8	6.3 - 10.3	8.6	0.3 - 20.3	R
Ernie	USA	PI592001	24.9	2.6 - 34.7	20.5	7.7 - 44.2	9.4	0.3 - 32.3	R
MeiQianWu	China	PI525071	25.3	12.3 - 38.5	16.1	5.9 - 27	9.5	2.4 - 16.6	R
Tokai 66	Japan	PI382161	25.7	15.7 - 36.2	35.4	5.1 - 66.7	1.0	0.5 - 2.6	MR
Nyubai	Japan	JIRCAS	32.0	4.6 - 48.8	40.2	15.8 - 74.4	1.1	0.2 - 1.6	MR
Shoukomugi II	Japan	JIRCAS	37.9	28.2 - 45.3	26.3	4.8 - 64.8	1.6	0.3 - 3.1	MR
Yangmai 158	China	JAAS	28.6	5.0 - 53.0	30.3	8.3 - 80.7	1.8	0.7 - 4.8	MR
Itoukomugi	Japan	JIRCAS	24.1	4.0 - 48.8	30.1	9.2 - 52.3	1.9	0.5 - 5.2	MR
Sotome	Japan	JIRCAS	24.8	16.0 - 34.3	42.7	22.8 - 70.2	2.2	0.4 - 3.2	MR
Sobakomugi 1B	Japan	JIRCAS	28.0	18.3 - 37.5	32.4	15.3 - 41.7	2.7	0.9 - 6.7	MR
Abura Komugi	Japan	JIRCAS	28.9	19.4 - 43.7	48	19.2 - 66.7	2.7	1.7 - 4.5	MR
Sotome A	Japan	JIRCAS	43.5	23.6 - 83.0	32.5	9.6 - 55	3.1	1.5 - 4.6	MR
Chokwang	Korea	JAAS	24.0	4.8 - 48.6	28.7	7.1 - 83.3	3.1	0.6 - 5.5	MR
NobeokabouzuKomugi	Japan	JIRCAS	40.0	29.0 - 44.9	32.3	11.4 - 75.6	3.3	0.7 - 7.3	MR
Wannin 2	China	JAAS	22.7	7.0 - 33.0	44.1	7.5 - 75.2	3.5	0.4 - 6.2	MR
YangLaZi	China	PI502932	31.2	5.3 - 46.3	39.1	25.6 - 70.4	3.5	0.5 - 6.2	MR
Hongjianzi	China	JAAS	17.4	1.7 - 28.3	28.6	7 - 49.9	3.7	0.5 - 8.2	MR

Table 2.1 Continued

Landrace/cultivar	Country	Source	PIF (%)		PSS (%)		DON (ppm)		Rating
			Mean	Range	Mean	Range	Mean	Range	
Freedom	USA	PI592002	35.2	4.1 - 74.7	27.8	4.3 - 59.3	3.8	0.7 - 8.7	MR
Kagoshima	Japan	JIRCAS	19.5	8.3 - 32.1	31.2	11.6 - 47.8	4.8	0.6 - 14.0	MR
ShuiLiZhan	China	PI502930	20.0	4.0 - 37.5	37.3	9.9 - 75.9	5.0	2.9 - 8.0	MR
Sanshukomugi	Japan	PI197130	12.2	1.0 - 37.5	49	32.6 - 69.9	5.1	1.6 - 10.3	MR
QiangShuiHuang	China	PI502931	26.5	15.3 - 37.3	28.1	6.5 - 53.3	5.1	0.5 - 12.3	MR
Kikuchi	Japan	JIRCAS	21.2	13.3 - 27.7	49.3	28.4 - 90.9	5.3	0.2 - 14.6	MR
FSW	China	JAAS	24.5	11.3 - 49.0	32.2	7.6 - 81.4	5.4	0.3 - 16.1	MR
HuiShanYangMai	China	PI462154	22.8	17.3 - 32.0	37.1	19.9 - 69.6	5.6	0.5 - 12.4	MR
Abura	Japan	PI382140	23.5	9.0 - 34.8	36.4	21 - 49.8	5.7	2.1 - 14.9	MR
Shinchunaga	Japan	PI197128	30.3	22.3 - 41.3	39.6	12.6 - 69.6	5.8	0.6 - 18.7	MR
Xueliqing	China	JAAS	21.1	3.6 - 48.8	47.8	21.3 - 68.8	6.0	0.2 - 12.7	MR
Sobakomugi 1C	Japan	JIRCAS	40.4	32.7 - 47.7	27.1	9.2 - 72.9	6.3	0.5 - 18.7	MR
WZHHS	China	JAAS	16.9	3.7 - 24.0	26.9	17.2 - 51.5	6.6	2.0 - 18.0	MR
Yangmai 4	China	JAAS	26.7	1.0 - 42.3	34.6	9.2 - 81.5	7.6	0.8 - 17.9	MR
Yangmai 5	China	JAAS	28.2	5.0 - 51.3	35	7.2 - 74.1	8.4	1.4 - 19.0	MR
JiangDongMen	China	PI462135	33.2	0.5 - 55.7	36.5	5.9 - 53.5	10.1	1.0 - 20.0	MR
DaHuangPi	China	PI502939	36.8	28.7 - 42.0	45.6	40.4 - 55.8	10.7	1.1 - 30.8	MR
LiangGuangTou	China	PI435109	26.0	19.1 - 34.6	49	45.9 - 53	10.9	0.9 - 29.7	MR
CanLaoMai	China	JAAS	18.4	2.0 - 29.5	39.4	21.6 - 72.2	12.8	3.4 - 20.1	MR
Emai6	China	JAAS	17.7	7.2 - 26.3	29	5.5 - 88.3	14.6	0.3 - 51.6	MR
Dahongpao	China	JAAS	31.5	14.3 - 47.7	43.3	10.7 - 79.2	16.7	0.1 - 38.8	MR
XingHuaBaiYuHua	China	PI462150	16.8	2.5 - 36.5	43.1	16.8 - 75	17.2	5.3 - 29.7	MR
YouBaoMai	China	PI524980	32.8	23.7 - 38.5	41.3	10.9 - 87.5	19.2	0.5 - 55.3	MR
Fusuihuang	China	JAAS	32.5	6.0 - 56.7	49.3	28.2 - 85.9	41.9	0.4 - 152.1	MR
YouZiMai	China	PI435110	42.3	26.4 - 54.3	48.1	7 - 63.7	46.6	0.5 - 158.6	MR
KuangTuErhHsiaoMai	China	PI57347	25.9	2.8 - 36.2	71.4	53.1 - 100	1.6	0.5 - 2.2	MS
SapporoHaruKomugiJugo	Japan	PI81791	21.6	3.4 - 45.7	52.3	23.6 - 68.1	2.1	0.3 - 3.7	MS
Funo	Italy	JAAS	29.5	11.7 - 35.6	51.8	39.5 - 70.6	3.3	0.6 - 9.8	MS
FangTouHongMang	China	PI502938	23.9	0.5 - 49.1	69.5	38.9 - 100	4.1	3.7 - 4.7	MS
PaiMaiTze	China	PI64285	-	-	69.9	53.7 - 100	8.3	8.2 - 8.5	MS
FangTouBaiMang	China	PI502935	-	-	68	48.7 - 100	9.5	3.0 – 21.0	MS
Dafanliuzhu	China	JAAS	27.5	1.0 - 43.6	54.4	16.3 - 88.5	9.6	4.9 - 18.4	MS
NTDHP	China	JAAS	39.7	4.7 - 75.0	66.6	52.3 - 100	9.9	2.0 - 24.3	MS
SanChaHo	China	PI70674	37.0	4.3 - 68.3	56.6	34.1 - 88	10.1	4.1 - 21.9	MS
HongHuaWu	China	PI502949	25.9	3.8 - 45.0	54.9	5.5 - 80	10.5	7.6 - 13.3	MS
ChuShanBao	China	PI524973	32.2	0.5 - 46.7	62.5	29.6 - 100	10.7	0.8 - 31.5	MS
Chile	Chili	JIRCAS	30.4	9.7 - 52.0	57.3	25.5 - 80.4	11.1	4.5 - 18.2	MS
ShanhaiCaiZiHuang	China	PI462140	11.2	4.3 - 26.7	53.5	16.9 - 78.6	11.2	3.3 - 26.7	MS
Heshangmai	China	JAAS	32.7	0.5 - 49.7	66.2	14.1 - 92.6	12.0	0.6 - 37.7	MS
Jingzhou 1	China	JAAS	23.8	2.3 - 43.6	69.1	44.7 - 100	13.6	3.2 - 29.0	MS

Table 2. 1 Continued

Landrace/cultivar	Country	Source	PIF (%)		PSS (%)		DON (ppm)		Rating
			Mean	Range	Mean	Range	Mean	Range	
Shironankin	Japan	JIRCAS	29.0	2.9 - 44.7	60.5	23.5 - 90.7	15.1	5.7 - 25.2	MS
HongMongBai	China	PI518598	-	-	64.8	7.7 - 100	24.8	0.8 - 48.8	MS
Zhen 7495	China	JAAS	38.4	31.1 - 51.0	54.5	30.2 - 80.4	29.5	3.9 - 72.0	MS
Avrora	Russia	JAAS	26.5	4.8 - 47.4	70.2	18.1 - 100	29.7	13.6 - 43.0	MS
HungGuangTou	China	PI447389	29.3	3.3 - 49.2	66.8	26.7 - 96.3	30.1	3.0 - 62.7	MS
Zairai Yuubou	Japan	JIRCAS	29.2	20.6 - 36.0	61.8	37.5 - 88.6	30.4	1.6 - 96.0	MS
YuLinBai	China	PI591997	-	-	72.3	51.1 - 90	32.5	31.2 - 33.8	MS
HongMangMai	China	PI525072	-	-	76	51.8 - 100	7.0	7.0 - 7.0	S
Nanda 2419	China	JAAS	9.1	2.0 - 14.7	81.5	46.4 - 100	7.7	1.3 - 20.8	S
DaBaiPao	China	PI525074	-	-	78.5	43.8 - 100	13.2	9.6 - 16.9	S
Chinese Spring	China	JAAS	30.9	4.4 - 46.1	76.5	66.1 - 86.7	22.7	3.2 - 79.9	S
Sanyuehuang	China	JAAS	26.1	13.0 - 47.3	92.2	71.9 - 100	24.2	6.5 - 39.0	S
TaFangShen	China	PI70666	25.2	2.7 - 44.6	82.6	82.6 - 82.6	37.8	8.8 - 98.9	S
ChanjiBaiDongMai	China	PI445868	46.5	18.0 - 83.0	-	-	77.9	2.9 - 188.9	S
Sumai 3	China	JAAS	28.1	19.0 - 38.2	18.8	10.6 - 26.2	4.1	0.1 - 10.3	R
Wheaton	USA	-	28.2	23.0 - 31.3	89.2	84.3 - 93.4	84.5	25.8 - 475.3	S

DON is value in ppm; PSS is the proportion of scabbed spikelets in a spike; PIF is the percent of infected florets in a spike.

[†]Wheat type II resistance to infection by *F. graminearum*: R = resistant with PSS ranging from 0-25%, MR = moderately resistant with PSS ranging from 26-50%, MS = moderately susceptible with PSS ranging from 51-75%, and S=susceptible with PSS ranging from 76-100%,

[§] Data missing

[¶]JAAS - Jiangsu Academy of Agricultural Science, Nanjing, P.R. China; JIRCAS – Japan International Research Center for Agricultural Sciences

Table 2.2 Correlations among FHB infection and DON levels for wheat accessions from different origins in greenhouse-evaluation experiments

Trait	2003 spring PSS (%)	2003 spring DON (ppm)	2003 fall PSS (%)	2003 fall DON (ppm)	2004 PIF (%)	2004 DON (ppm)	2005 PIF (%)	2005 DON (ppm)
2003 spring PSS	–							
2003 spring DON	0.71***	–						
2003 fall PSS	0.61***	0.48***	–					
2003 fall DON	0.35**	0.28*	0.27*	–				
2004 PIF	0.07	0.19	0.09	0.13	–			
2004 DON	0.53***	0.51***	0.65***	0.16	-0.05	–		
2005 PIF	0.09	0.21	0.05	-0.04	0.14	-0.02	–	
2005 DON	0.35**	0.29*	0.32**	0.36**	0.02	0.2	0.23*	–

Note: *, **, and *** represent significantly different from 0 at $p < 0.05$, $p < 0.01$, and $p < 0.001$, respectively.

Table 2.3 Number of accessions and means of PIF, PSS, and DON for wheat accessions in four different FHB resistance categories

Type II Resistance[†]	Number of accessions	Mean of resistance trait		
		PIF (%)	PSS (%)	DON (ppm)
R	26	20.0a	15.5a	4.0a
MR	39	27.3a	37.3b	8.3ab
MS	22	28.5a	62.5c	14.5b
S	7	27.6a	82.5d	27.2c

[†]Wheat type II resistance to the infection of *F. graminearum*: R=resistant with PSS ranging from 0–25%, MR=moderately resistant with PSS ranging from 26–50%, MS=moderately susceptible with PSS ranging from 51–75%, and S=susceptible with PSS ranging from 76–100%.

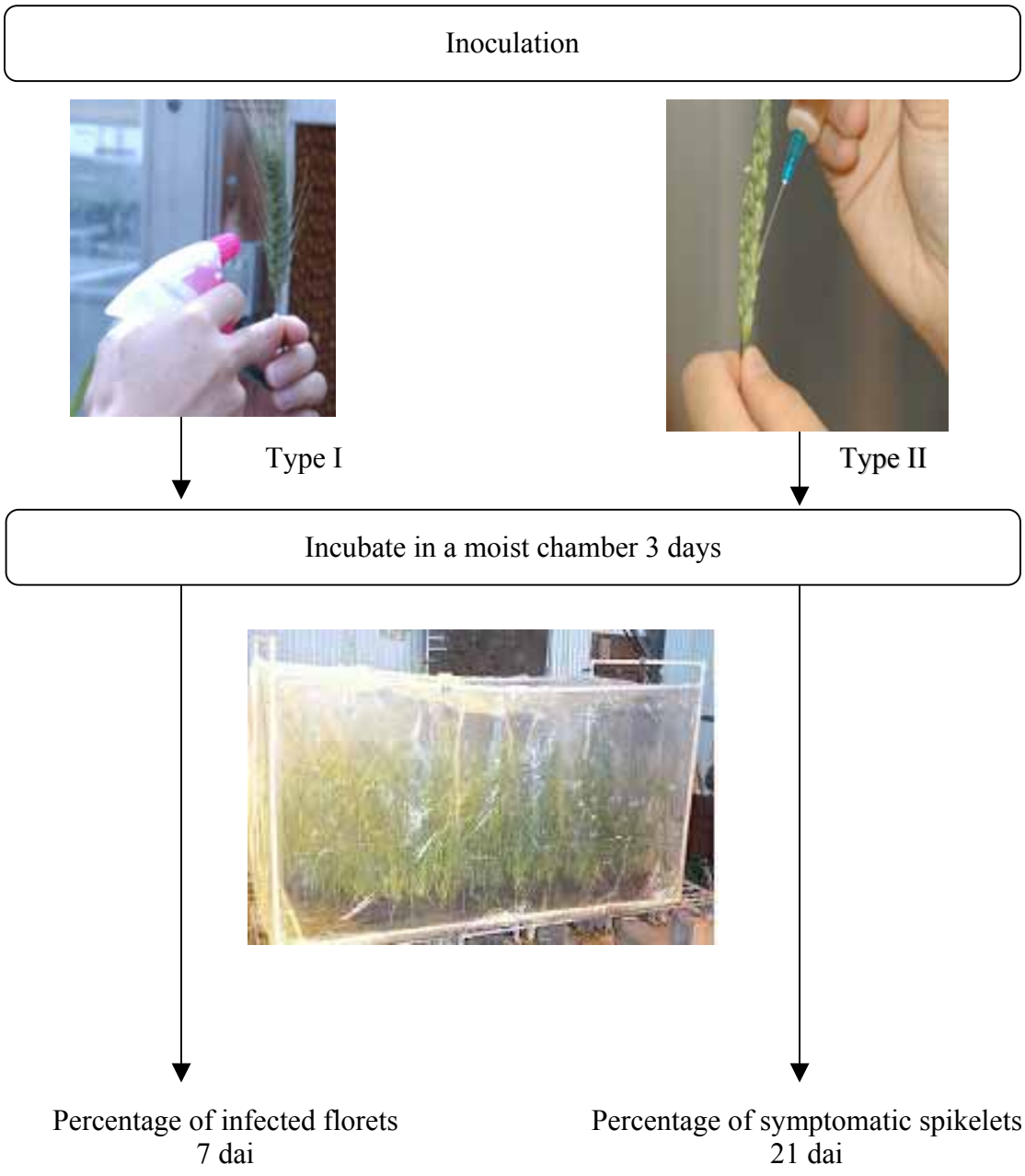


Figure 2.1 Evaluation of wheat type I and II resistance to FHB in greenhouse

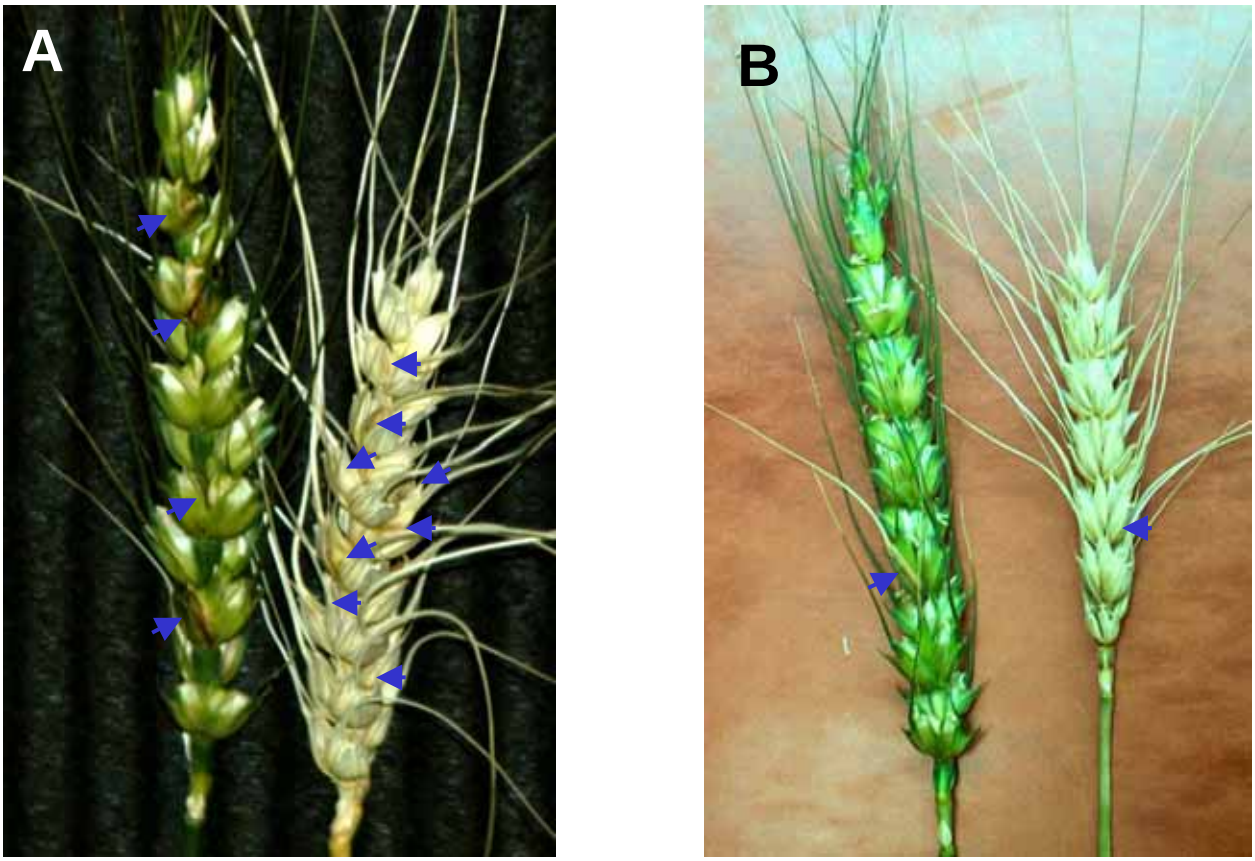


Figure 2.2 FHB symptoms resulting from artificial infections (left plant, resistant accession; right plant, susceptible accession). **A**, type I resistance evaluated with spray inoculation; **B**, type II resistance evaluated with single-spikelet inoculation. Arrows indicate infection sites.

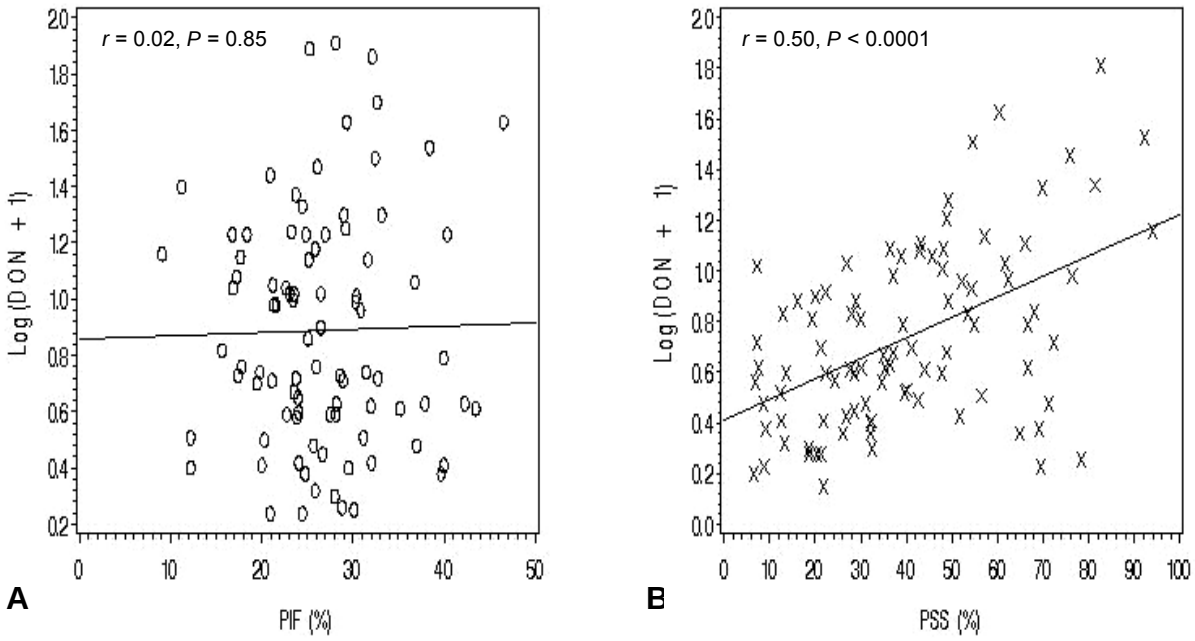


Figure 2.3 Association between visual disease ratings and DON contents for **A**, the proportion of infected florets (PIF) and **B**, the proportion of scabbed spikelets (PSS). The line marks the regression.

CHAPTER 3 - Marker-assisted characterization of Asian wheat lines for resistance to Fusarium head blight

Introduction

Haplotyping wheat accessions with SSR markers flanking the 3BS, 5AS, and 6BS FHB resistace QTL of Sumai 3 may provide useful information for predicting novel QTL by comparison of haplotypes of target accessions with known cultivars such as Sumai 3. Haplotype is a combination of alleles (for different markers or genes) which are located closely together on the same chromosome and which tend to be inherited together. The underlying assumption is that if a wheat line has the same allelic pattern for marker loci flanking the QTL as that in the known resistant line, the two lines most likely have the same QTL (Bai *et al*. 2003; Sun *et al*. 2003; McCartney *et al*. 2004); while, if a wheat line has a different allelic pattern from that in the known resistant line, the two lines most likely have different alleles of the QTL or different QTL.

Genetic diversity among wheat cultivars based on molecular markers has been effectively assessed with cluster analysis (CA) and principal component analysis (PCA) (Barrett and Kidwell 1998a, b; Liu and Anderson 2003). Cluster analysis and PCA are two of the most commonly used methods for analysis of genetic diversity irrespective of the types of data (morphological, biochemical, or molecular marker data). Both methods simultaneously analyze multiple measurements on each individual under investigation. CA seeks to identify a set of clusters that both minimize within-cluster variation and maximize between-cluster variation (Johnson and Wichern 1992). Thus, individuals within a cluster will genetically closer than those in different clusters. One of the predominantly used CA algorithms is unweighted pair-group method using average (UPGMA). It is a distance-based method that represents the distance or dissimilarity between two clusters as the average distance between all inter-cluster pairs. PCA involves a mathematical procedure that transforms a set of correlated variables into a set of uncorrelated variables called principal components. These principal components (PCs) are linear combinations of the original variables. The first step in PCA is to calculate eigenvalues, which define the amount of total variation that is displayed on the PC axes. The first PC summarizes

most of the variability present in the original data relative to all remaining PCs. The second PC explains most of the variability not summarized by the first PC and uncorrelated with the first PC, and so on (Jolliffe 1986). Since PCs are orthogonal and independent of each other, each PC reveals different properties of the original data and needs to be interpreted independently. The first two or three PCs usually summarize most of the total variability of the original data, so they can be utilized to derive a 2- or 3-dimensional scatter plot of individuals. The geometrical distances among individuals in the plot reflect the genetic distances among them. Aggregations of individuals in such a plot reveal sets of genetically similar individuals.

In this study, a panel of FHB-resistant germplasm was systematically characterized on the basis of both FHB phenotypic and molecular marker data. The objectives of this study were to identify new sources of FHB resistance from the Asian wheat gene pool, elucidate the genetic relationship among these accessions by analyzing molecular marker data with CA and PCA methods, and investigate the origin of QTL on 3BS, 5AS, and 6BS of Sumai 3. Information from this study may help breeders to select different sources of resistant materials for enlarging the genetic diversity of FHB resistance in their breeding programs.

Materials and methods

Plant materials

Fifty-nine wheat accessions originated in China (38 accessions), southwestern Japan (20 accessions), and Korea (one accession) (Table 3.1). The majority of the Asian accessions have some degree of FHB resistances based on previous FHB evaluation (Chapter 2). These materials include the well-known FHB resistant cultivars (Sumai 3, Wangshuibai, and Ning 7840) from China and three U.S. cultivars (Ernie, Freedom and Clark) as resistant, moderately resistant and susceptible controls, respectively. Funo from Italy and Avrora from Russia were also included because Funo is a parent of Sumai 3 and Avrora is a parent of Ning 7840. Both of them have been extensively used as parents in Chinese breeding programs from the 1950s to the 1970s (Bai *et al*. 2003).

Evaluation of FHB resistance

All wheat accessions were evaluated for FHB type II resistance in two experiments (2003 spring and fall) with three replications in each experiment in the greenhouse at Kansas State

University. The inoculum of *F. graminearum* and the whole evaluation procedure are described in Chapter 2. Infected and total spikelets in a spike were counted at the 21st day after inoculation, and the proportion of symptomatic spikelets (PSS) was calculated as a measure of disease severity.

Molecular marker analysis

DNA was isolated from seedling leaf tissue by the CTAB method (Saghai-Maroof *et al*. 1984). For AFLP analysis, DNA restriction digestion (with *Eco*RI and *Mse*I), adapter ligation, and PCR amplification were carried out as described by Bai *et al*. (2003). Pre-amplification was conducted with an *Eco*RI primer (5'-ACTGCGTACCAATTC) and an *Mse*I primer (5'-GATGAGTCCTGAGTAA). Selective PCR used 24 primer combinations between six IR-dye-labeled *EcoR*I primers with selective nucleotides of AGT, AAC, ACT, GCTG, CTCG, and CATG and five unlabeled *Mse*I primers with selective nucleotides of CAC, CAT, CAGT, TGC, and AGTG.

Twenty-five SSR markers (Table 3.2) associated with FHB-resistance QTL on 3BS (Anderson *et al*. 2001; Buerstmayr *et al*. 2002; Zhou *et al*. 2002), 3BSc (Somers *et al*. 2003), 5AS (Buerstmayr *et al*. 2002), 6BS (Anderson *et al*. 2001; Yang *et al*. 2003), 4B, and 2DL (Somers *et al*. 2003) were screened for polymorphisms among these accessions. The QTL on 4B and 2DL were originally detected from Wuhan-1, a Chinese breeding line with unknown pedigree (Somers *et al*. 2003), and five SSR markers linked to the two QTL (McCartney *et al*. 2004) were included in this study (Table 2). The SSR markers were amplified according to the protocol described by Bai *et al*. (2003). For PCR detection, an M13 tail sequence was added to the 5' of the forward SSR primers (5'-ACGACGTTGTAAAACGAC). A PCR with 10-µL reaction volume consisted of ~50 ng DNA, 1X PCR buffer, 0.2 m*M* dNTPs, 2 m*M* $MgCl_2$, 1 pmol each of tailed forward and reverse primers, and IR-dye-labeled M13 primer (Li-Cor, Inc. Lincoln, NE, U.S.A.). The SSR markers on 3BS were directly labeled with IR-dye on 5' ends without adding the M13 primer in PCR reaction mixture. To amplify SSR, a touchdown PCR profile started at 95°C for 5 min, followed by 5 cycles of 45 seconds at 95°C, 5 min at 68°C, and 1 min at 72°C; the annealing temperature was lowered by 2°C in each following cycle. Then 5 more cycles in which the annealing time was 2 min and the temperature was lowered 2°C in each following cycle. For the last 25 additional cycles, the annealing temperature was held constant at

50°C, with 5 min at 72°C for a final extension. AFLP and SSR fragments were analysized in a Li-Cor 4200 DNA Sequencer and scored with Saga^{GT} software (Li-Cor, Inc. Lincoln, NE)..

Data analysis

The molecular marker data used in this study are binary data. Thus, polymorphic DNA fragments are scored as either present (1) or absent (0) for each marker locus. The SAS software package was used for basic statistical analysis (SAS Institute Inc., Cary, NC, U.S.A.). Cluster analysis was performed using NTSYS-pc version 2.11a (Rohlf 1998). The genetic diversity among accessions, on the basis of the AFLP and SSR data, was estimated according to Jaccard's similarity coefficient and was calculated as $1 - a/(n - d)$, where a is the number of bands in common between two wheat accessions, n is the number of bands in the matrix, and d is the number of bands absent in both wheat accessions. The SIMQUAL routine of NTSYS-pc was used to generate the Jaccard's similarity coefficient matrix. The unweighted pair-group method with arithmetic mean (UPGMA) and SHAN routines of NTSYS-pc program were used to construct a dendrogram. Bootstrapping (500 iterations) was performed to evaluate the robustness of the branching points using Phyltools (Buntjer, 2001). The neighboring and consensus modules from the PHYLIP program (Felsenstein, 2005) were used to construct the consensus tree. Bootstrap values were percentages of number of runs showing a specific branch point in the consensus tree when the data were randomly resampled for 500 times. DCENTER and EIGEN modules of the same program were used for principal component analysis (PCA). Haplotypes of the 64 wheat cultivars were determined on the basis of the allelic distribution pattern of SSR markers linked to 3BS, 5AS, 4B, and 6BS QTL in Sumai 3. Polymorphism information content (PIC) refers to the ability of a given marker to detect polymorphism within a population (Anderson *et al.* 1993). The PIC depends on the number of detectable alleles and their frequency. In this study, the simplified version (Lagercrantz *et al.* 1993) is used, which assumes that the wheat accessions are all homozygous: $PIC_i = 1 - \sum_{j=1}^{n} p_{ij}^2$ where p_{ij} is the proportion of the jth allele for marker i, and n is the total number of alleles.

Results

Type II FHB resistance of Asian wheat accessions

Asian wheat accessions differed in FHB severity, as reflected by their PSS (Table 3.1). The correlation coefficient for PSS between the two experiments was highly significant (r = 0.66, $p < 0.0001$). Wheat responses to FHB infection ranged from highly resistant (PSS < 10%, F 60096 and Fu 5114) to highly susceptible (PSS > 85%, Sanyuehuang). Approximately 67% of the Asian wheat accessions tested showed a high or moderate level of FHB resistance, with a mean PSS of less than 50% under the favorable epidemic conditions. More than half of the highly resistant accessions originated from China. Ernie from the U.S.A. also showed a high level of FHB resistance. The remaining highly resistant lines originated in Japan, including Aso Zairai II, Aso Zairai (Yuubou Kappu), Itou Komugi, and Shirasaya No 1. Taiwan Xiaomai, one of the parents of Sumai 3, had a PSS of less than 15%, which was similar to Sumai 3. Another parent of Sumai 3, Funo, showed moderate susceptibility to FHB in two experiments.

Genetic relationships among the wheat accessions

The genetic distance between the Chinese and the Japanese landraces was closer than between some of the Chinese landraces. A total of 483 SSR and AFLP polymorphic alleles were scored. Allele variation of the SSR marker *Xwmc397* and the AFLP primer combination mAGTG-eACT are shown as examples (Fig 3.1). The AFLP and SSR data illustrated that the groups resulting from cluster analysis of these accessions agreed with their geographic distribution and/or available pedigrees, with only a few exceptions (Fig. 3.2). In general, the cluster analysis roughly separated the 64 accessions into three major clusters, a Chinese/Japanese landrace cluster, an Avrora-related cluster, and a Funo-related cluster. Avrora- and Funo-related clusters consisted of all improved cultivars from China, with Avrora and Funo, respectively, as one parent. Most of the southwestern Japanese landraces (17 of 19) formed a closely related subgroup within the Chinese/Japanese landrace cluster (Fig. 3.2, Fig. 3.3). The Japanese subgroup was separated from the Chinese landraces at a similarity coefficient of about 0.82, whereas the most distant Chinese landrace, Chinese Spring, was separated from the other Chinese landraces at a similarity coefficient of about 0.77. It was interesting that Japanese landraces Shironankin and Shinchunaga were closer to Chinese accessions than to Japanese landraces.

The information on relationships among the 64 genotypes from PCA cluster analysis corresponded very well to those from Figure 3.3. The first principal component (PC1) clearly separated Funo-related accessions and Avrora-related accessions from Chinese/Japanese landraces; the second principal component (PC2) clearly separated Funo-related accessions from Avrora-related groups; and third principal component (PC3) corresponded to genetic relationships among accessions within each of the three groups (Fig. 3.3). The PCA results for most accessions were also consistent with their pedigrees. For example, Sumai 3 (accession 55) was located between Funo (accession 6) and Taiwan Xiaomai (accession 51), whereas Ning7840 (accession 43) was located between Sumai 3 and Avrora (accession 16) (Fig. 3.3).

The closest accessions in this study were Japanese accessions Nyubai, Nobeokabouzu and Nobeokabouzu Komugi, separated at a similarity coefficient of about 0.99. Two Chinese landraces, Huoshaobairimai and Huacandou, were also very close, at a similarity coefficient of about 0.96. The Avrora-related cluster and the Funo-related cluster were separated from the Chinese/Japanese landrace cluster at similarity coefficients of 0.71 and 0.73. No Japanese landraces were classified into the Avrora-related cluster or the Funo-related cluster. The Avrora-related cluster (6 lines) and the Funo-related cluster (11 lines) agreed well with the pedigrees of their accessions. As for FHB resistant sources, the genetic diversity within the Chinese landraces was broader than that of the Japanese landraces (Fig. 3.2, Fig. 3.3).

Allelic variation in SSR marker loci linked to QTL for FHB resistance

Twenty-five SSR markers linked to six putative QTL on five chromosome arms of wheat were highly polymorphic among the wheat accessions evaluated (Table 3.2). The PIC values for these SSRs ranged from 0.23 (*Xgwm113*) to 0.92 (*Xwmc612*). Two (*Xbarc75* and *Xgwm508*) to 18 (*Xwmc612*) alleles per SSR locus were detected across all 64 accessions (Table 3.2). Haplotypes based on Sumai 3/non-Sumai 3 alleles were identified from 4 on 3BSc to 24 on 5AS (Table 3.2).

The overall results indicated a trend in which the more putative Sumai 3 marker alleles for 3BS QTL an accession carried, the more likely the accession showed a lower average PSS (Table 3.4). However, 15 accessions did not follow the trend, which suggested that the resistance in these accessions may be controlled by different QTL or alleles from that on 3BS of Sumai 3. Twenty-one haplotypes were identified for the five SSR markers linked to the major QTL on

3BS among the 64 accessions when the marker alleles were analyzed as Sumai 3 and non-Sumai 3 alleles. Only two accessions, Ning 7840 and Taiwan Xiaomai, shared the haplotype of Sumai 3 (Table 3.2, Table 3.3). Two Japanese accessions (Sanshukomugi and Shinchunaga) and one Chinese accession (FSW) carried four of the five Sumai 3 SSR alleles; 34 accessions carried 2 to 3 Sumai 3 SSR alleles; 11 accessions carried only one Sumai 3 SSR allele; and the remaining 12 lines, including Funo (another parent of Sumai 3) and Avrora (one of Ning 7840's parents), carried none of any of the five Sumai 3 SSR alleles for the major QTL on 3BS (Table 3.2). Among those 23 accessions with none or only one Sumai 3 allele, 7 lines expressed a level of FHB resistance similar to that of Sumai 3, and another 8 accessions showed moderate FHB resistance, with less than 50% PSS under high disease pressure. The 15 wheat lines are unlikely to carry the major QTL for FHB resistance.

Discussion

Genetic relationships among Asian FHB resistant germplasm

Cluster analysis and principal component analysis uncovered the relatively limited genetic diversity among the accessions in this collection. This result was not surprising because most of FHB resistant wheat landraces originated in southeast China and in the Kyushu area of southwestern Japan, where FHB epidemics have been frequent and severe, and the improved cultivars were mainly related to Avrora from Russia, Mentana, and its relative Funo from Italy. The results on genetic similarities of Chinese/Japanese FHB resistance accessions from this study agreed with a previous study (Bai a*t al.* 2003).

The genetic relationships based on cluster analysis matched well with their pedigree information and their geographic origins. The dendrogram clearly separated the Funo-related accessions and the Avrora-related accessions from the Chinese/Japanese landraces. This may be because the Russian cultivar Avrora and Italian cultivar Funo and Mentana were far from Asian landraces and were extensively used in early wheat breeding programs in China. Introduction of cultivars Funo and Avrora from Europe broadened the genetic diversity of Chinese wheat. The Chinese landrace Taiwan Xiaomai and the Italian cultivar Funo are two parents of Sumai 3, and Sumai 3 falls between the Funo and Taiwan Xiaomai, although it is closer to Taiwan Xiaoma in the Chinese/Japanese landrace cluster. Sumai 3 and Avrora are the parents of Ning 7840, and Ning 7840 is closer to Avrora than to Sumai 3 (Fig. 3.1, Fig. 3.2). The Funo cluster is further

separated into three subgroups. Cultivars Nanda 2419, Emai 6, Jingzhou 1, and Wannian 2 form a subgroup, which share a common Italian ancestor Mentana (Fig. 3.1). Yangmai 1, Yangmai 4, Fumai 3, and Yangmai 158 form another subgroup in which Funo serves as a common ancestor. Fusuihuang and Zhen 7495 form the third subgroup because Zhen7495 was derived from a cross between Fusuihang and Youyimai, a derivative of Funo. These results clearly confirmed the pedigree relationships within each of the three subgroups.

Results for the Avrora-related cluster were similar. In this cluster, Ning 7840, Sumai 49, and Fu 5114 shared the common ancestor Avrora. But the two Chinese landraces FSW and WZHHS were close to Ning 7840, and their pedigree information was not available to verify this relationship.

It was unexpected that the three accessions from the U.S.A., Freedom, Ernie, and Clark, did not form a separate group in the dendrogram. These three cultivars were in a completely different cluster from the Chinese landraces and cultivars in a previous study when more cultivars from the U.S.A. were used (Bai *et al*. 2003). This could be because the small number of accessions with diverse genetic backgrounds from the U.S.A. could not provide sufficient genetic information to form their own group. This result suggests that interpretation of genetic relationships between Asian landraces and the cultivars from the U.S.A. should be cautious.

The Japanese landraces form a sub-cluster within Chinese landrace cluster, indicating that the genetic bases of FHB resistant landraces from southwestern Japan is narrower than that of the Chinese landraces. It is interesting that the Japanese landraces Shinchunaga and Shironankin are closer to Sumai 3 and Caizihuang, respectively, than to the other Japanese landraces (Fig. 3.1). Shironankin may originate in China, because Shironankin means 'White Nanjing' in Japanese and Nanjing is a Chinese city where many FHB resistant Ning lines were developed. The same might also be true for Shinchunaga, which has been a major source of FHB resistance widely used in Japanese breeding programs for decades (Ban 2000). Shinchunaga had similar banding patterns to those in Sumai 3 at most SSR marker loci in the three QTL regions of Sumai 3 (Table 3.2). The results suggest that some of the FHB resistance QTL in Japanese germplasm might originate from Chinese landraces.

It is possible that Nobeokabouzu Komugi, Nobeokabouzu, and Nyubai are the same landrace with different identifications. Japanese landrace Nobeokabouzu Komugi was reported to have the best resistance in Japanese germplasm (Ban 2000). In our study, two other

accessions, Nyubai and Nobeokabouzu are very close to Nobeokabouzu Komugi, with 99% identity according to AFLP marker data and 100% identity according to the 25 SSR marker alleles scored in this study. In our collection, Nyubai and Nobeokabouzu Komugi were originally from the Gene Bank of Japan, and Nobeokabouzu was obtained from China. Komugi means 'wheat' in Japanese and can be omitted from the name. Thus, any one of the three should be able to represent the same accession in breeding programs.

Origin of QTL for FHB resistance from Sumai 3

Taiwan Xiaomai was likely the donor of the QTL on 3BS, 5AS and 6BS in Sumai 3. The major FHB resistance in Sumai 3 was once assumed to be from Funo, or from transgressive segregation of resistance genes from both parents (Liu and Wang 1990). More recent studies suggested that Taiwan Xiaomai might be the donor of the 3BS major QTL from Sumai 3 on the basis of haplotypes of 3BS markers from Sumai 3 and Funo (Bai *et al*. 2003; Liu and Anderson 2003). But marker data for other QTL and phenotypic data from Taiwan Xiaomai were not available in those studies. The FHB and SSR marker data from this study provided more solid evidence to support the proposed relationship. Taiwan Xiaomai showed the same high level of FHB resistance as Sumai 3, whereas Funo, the other parent of Sumai 3, was moderately susceptible to FHB. The five SSR marker alleles closely linked to the 3BS FHB resistant QTL in Taiwan Xiaomai were the same as those of Sumai 3, whereas those from Funo were all different. In addition, Taiwan Xiaomai had most of the Sumai 3 SSR alleles at 5AS and 6BS loci, whereas Funo has only a few Sumai 3 SSR alleles in the two QTL regions.

Haplotype pattern and FHB resistance

The more the markers in the haplotype pattern information, the more accurate the prediction of the FHB-resistance performance of an accession. The haplotypes of SSR markers that flank QTL can help to predict whether an accession carries known or different QTL. The results from this study show that the more the putative Sumai 3 marker alleles an accession has for QTL on 3BS, 5AS and 6BS, the more likely the accession has lower average PSS, suggesting that QTL on 3BS, 5AS and 6BS are important in most of the resistant accessions. For example, accessions with fewer than six Sumai 3 SSR alleles for the three QTL had an average PSS of 43%, whereas those with more than 10 Sumai 3 alleles had an average PSS of 17%. Haplotype information can only roughly predict whether an accession have one or more of the putative

QTL(s) and predicts its FHB-resistance performance in general. For instance, Fu 5114, a descendant of Sumai 3, carried three Sumai 3 SSR alleles at 3BS, three Sumai 3 SSR alleles at 5AS, and one Sumai 3 SSR allele at 6BS. The haplotyping data suggested that Fu 5114 might inherit both the 3BS and the 5AS QTL (Table 3.2). Thus its good FHB resistance was consistent with prediction based on its haplotype information.

If an accession is not genetically related to the QTL donor, haplotype information may not provide a reliable prediction for the presence of target QTL in the accession and its FHB-resistance performance. Several factors may affect the accuracy of QTL predictions, including the genetic relationship between a target line and the line with known QTL, the phenotypic effect of the target QTL, imprecise locations of QTL, and the genetic distance between the QTL and the markers used for the prediction. For example, the Chinese landrace Sanyuehuang carries two Sumai 3 3BS alleles, including *Xgwm533*, the SSR marker most closely linked to the 3BS QTL in Sumai 3, and four Sumai 3 5AS alleles, but it showed high susceptibility to FHB. In contrast, Huoshaomai, which carries one 3BS marker allele (*Xgwm389*) of Sumai 3 and four 5AS marker alleles of Sumai 3, showed high FHB resistance. Although both had similar haplotypes and were not related to Taiwan Xiaomai or Sumai 3, their reactions to FHB were completely different.

Potential new QTL for FHB resistance

Novel FHB-resistance QTL may contribute to the high level of FHB resistance in Fumai 3, Yangmai 1, Haiyanzhong, Huoshaomai, Ernie, and Huangcandou. These six accessions carried no or only one Sumai 3 SSR allele at 3BS and 6BS, and no more than four Sumai 3 alleles at 5AS. Thus these lines may not have the 3BS and 6BS QTL and may have the 5AS QTL in some of them. 5AS QTL only had a minor effect on Type II resistance (Somers *et al*. 2003), so new FHB resistance QTL may be responsible for their high level of FHB resistance. In addition, Huoshaomai and Huangcandou showed a high level of all three types of FHB resistance (Chapter 1). These accessions can be used to enlarge the wheat FHB resistance gene pool and enhance genetic diversity by incorporating different types of resistance in FHB resistant cultivars.

Conclusions

Fifty-nine Asian wheat landraces and cultivars differing in the levels of FHB resistance were evaluated for FHB type II resistance and for genetic diversity on the basis of AFLPs and SSRs. Genetic relationships among these wheat accessions estimated by cluster analysis of molecular marker data were consistent with their geographic distribution and pedigrees. Chinese resistant landraces had broader genetic diversity than that of accessions from southwestern Japan. The haplotype pattern of the SSR markers linked to FHB resistance quantitative trait loci (QTL) on chromosomes 3BS, 5AS and 6BS of Sumai 3 suggested that only a few lines derived from Sumai 3 may carry all the putative QTL from Sumai 3. About half of the accessions may have one or two FHB resistance QTL from Sumai 3. Some accessions with a high level of resistance may carry different FHB resistance loci or alleles from those in Sumai 3, and invite further investigation. SSR data also clearly suggested that FHB resistance QTL on 3BS, 5AS, and 6BS of Sumai 3 were derived from Chinese landrace Taiwan Xiaomai.

Table 3.1 Origin, pedigree, and FHB severity scores of 64 wheat accessions

ID	Name	Origin	[a]Source	Pdigree	[b]PSS	[c]FHB reaction
1	DaBaiPao	China	PI 462150	Landrace	78.5 ± 24.4	S
2	Nanda 2419	China	JAAS	Selection of Mentana	81.5 ± 21.0	S
3	Sanyuehuang	China	JAAS	Landrace	92.2 ± 12.3	S
4	Clark	USA	PI 512337	Beau//65256A1-8-1/67137B5-16/Sullivan/Beau//5517B8-5-3-3/Logan	94.1 ± 9.0	S
5	MaZ[h]aMai	China	PI382153	Landrace	-[d]	-
6	Funo	Italy	JAAS	Duecentodieci/Demiano	51.8 ± 11.6	MS
7	ShanghaiCaiZiHuang	China	PI 435110	Landrace	53.5 ± 26.3	MS
8	Dafanliuzhu	China	PI 447402	Landrace	54.4 ± 28.0	MS
9	Zhen 7495	China	JAAS	Youyimai/ Fusuihuang	54.5 ± 23.9	MS
10	Chile	Japan	ACC.26869	Chili	57.3 ± 24.6	MS
11	Shironankin	Japan	ACC.23277	Landrace	60.5 ± 27.4	MS
12	Zairai Yuubou	Japan	ACC.22130	Landrace	61.8 ± 27.9	MS
13	Heshangmai	China	JAAS	Landrace	66.2 ± 28.6	MS
14	NTDHP	China	JAAS	Landrace	66.6 ± 19.8	MS
15	Jingzhou 1	China	JAAS	Nanda 2419/Sereal	69.1 ± 18.0	MS
16	Avrora	Russia	JAAS	Neuzucht/Bezostaja 4//Bezostaja 1	70.2 ± 35.9	MS
17	ChineseSpring	China	CItr 14108	Landrace	76.5 ± 10.0	MS
18	Shou Komugi II	Japan	ACC.23653	Landrace	26.3 ± 21.7	MR
19	WZHHS	China	JAAS	Landrace from China	26.9 ± 14.0	MR
20	Freedom	USA	PI 562382	GR876/OH217	27.8 ± 22.7	MR
21	Hongjianzi	China		Landrace	28.6 ± 17.5	MR
22	Chokwang	Korea	JAAS	unknown	28.7 ± 30.2	MR
23	Emai 6	China	JAAS	Selection from radiated Nanda 2419	29.0 ± 33.5	MR
24	Itou Komugi	Japan	ACC.23647	Landrace	30.1 ± 35.3	MR
25	Yangmai 158	China	JAAS	St1472/506//Yangmai4	30.3 ± 27.6	MR
26	Kagoshima	Japan	ACC.23542	Landrace	31.2 ± 15.0	MR
27	FSW	China	JAAS	Landrace from China	32.2 ± 25.5	MR
28	Nobeokabouzu Komugi	Japan	PI 382153	Landrace	32.3 ± 24.1	MR
2[9]	Soba Komugi 1B	Japan	ACC.23662*	Landrace	32.4 ± 9.1	MR
30	Sotome A	Japan	ACC.23660	Landrace	32.5 ± 34.5	MR
31	Yangmai 4	China	JAAS	(Nanda2419/Triumph)F5/Funo	34.6 ± 29.5	MR
32	CanLaoMai	China	JAAS	Landrace	39.4 ± 28.5	MR
33	Shinchunaga	Japan	PI 197130	Selection from landrace Nakanaga	39.6 ± 18.4	MR
34	Nyubai	Japan	ACC. 22957	Landrace	40.2 ± 36.1	MR
35	Sotome	Japan	ACC.23595	Landrace	42.7 ± 32.8	MR
36	Dahongpao	China	JAAS	Landrace	43.3 ± 39.0	MR
37	Wangnian 2	China	JAAS	Selection of Mentana	44.1 ± 32.8	MR
38	Xueliqing	China	JAAS	Landrace	47.8 ± 18.5	MR
39	Abura Komugi	Japan	ACC.23516	Landrace	48.0 ± 18.5	MR
40	Sanshukomugi	Japan	PI 592001	Landrace	49.0 ± 13.8	MR
41	Fusuihuang	China	PI 213833	Landrace	49.3 ± 23.2	MR
42	Kikuchi	Japan	ACC.22952	Landrace	49.3 ± 21.5	MR
43	Ning 7840	China	JAAS	Avrova/Anhui 11/Sumai 3	6.6 ± 1.4	R

Table 3.1 Continued

ID	Name	Origin	[a]Source	Pdigree	[b]PSS	[c]FHB reaction
44	F 60096	China	JAAS	Jingzhou 1/Sumai 2	6.9 ± 1.2	R
45	Fu 5114	China	JAAS	LongXi 18/(Avrora/Anhui11//Sumai 3)	7.4 ± 1.6	R
46	Sumai 49	China	PI 447405	N7922/(Avrova/Anhui 11/Sumai 3)	7.8 ± 1.5	R
47	Wangshuibai	China	PI 197129	Landrace	8.7 ± 3.8	R
48	Aso Zairai II	Japan	ACC.23524	Landrace	8.9 ± 3.7	R
49	Baisanyuehuang	China	JAAS	Landrace	12.5 ± 6.9	R
50	Aso Zairai (Yuubou Kappu)	Japan	ACC.23521	Landrace	12.7 ± 7.7	R
51	Taiwan Xiaomai	China	Purdue University	Landrace	13.0 ± 3.2	R
52	Huangcandou	China	JAAS	Landrace	13.4 ± 7.6	R
53	Haiyanzhong	China	JAAS	Landrace	13.7 ± 6.3	R
54	Fumai 3	China	PI 447404	Orofen/Funo	18.6 ± 16.4	R
55	Sumai 3	China	PI 462149	Funo/Taiwan Xiaomai	18.8 ± 11.9	R
56	HuangFangZhu	China	JAAS	Landrace	20.2 ± 17.4	R
57	Ernie	USA	PI 584525	PI584525 PIKE/3/Stoddard/Blueboy// Stoddard/D1707	20.5 ± 13.7	R
58	Huoshaomai	China	JAAS	Landrace	21.4 ± 13.7	R
59	Caizihuang	China	JAAS	Landrace	22.0 ± 14.2	R
60	Huoshaobairimai	China	JAAS	Landrace	22.0 ± 22.3	R
61	Shirasaya No 1	Japan	PI 197128	Landrace	22.4 ± 14.5	R
62	Qiaomai Xiaomai	Japan	ACC.24142	Landrace	22.4 ± 18.4	R
63	Yangmai 1	China	PI 447403	Selection of Funo	22.5 ± 22.0	R
64	NobeokaBouzu	Japan	JAAS	Landrace	24.3 ± 19.3	R

[a] JAAS – seeds were provided by Jiangsu Academy of Agricultural Science, Nanjing, P.R. China and all these accessions were selected based on their good resistance to FHB in China; PI–seeds were provided by the National Small Grains Research Facility at Aberdeen, ID, U.S.A. and were selected based on their diverse geographic distribution in China without knowledge of their FHB resistance; ACC.-accession number in Gene Bank of MAFF, JAPAN and all these accessions were selected based on FHB resistance tested in Japan

[b] Average of proportion of symptomatic spikelets in a spike (PSS) from replication means of two season greenhouse evaluations in 2003 ± standard deviation

[c] *R* resistant (PSS < 25%), *S* susceptible (PSS > 75%), *MR* moderately resistant (50% > PSS > 25%), *MS* moderately susceptible (50% < PSS < 75%)

[d] – Data are not available

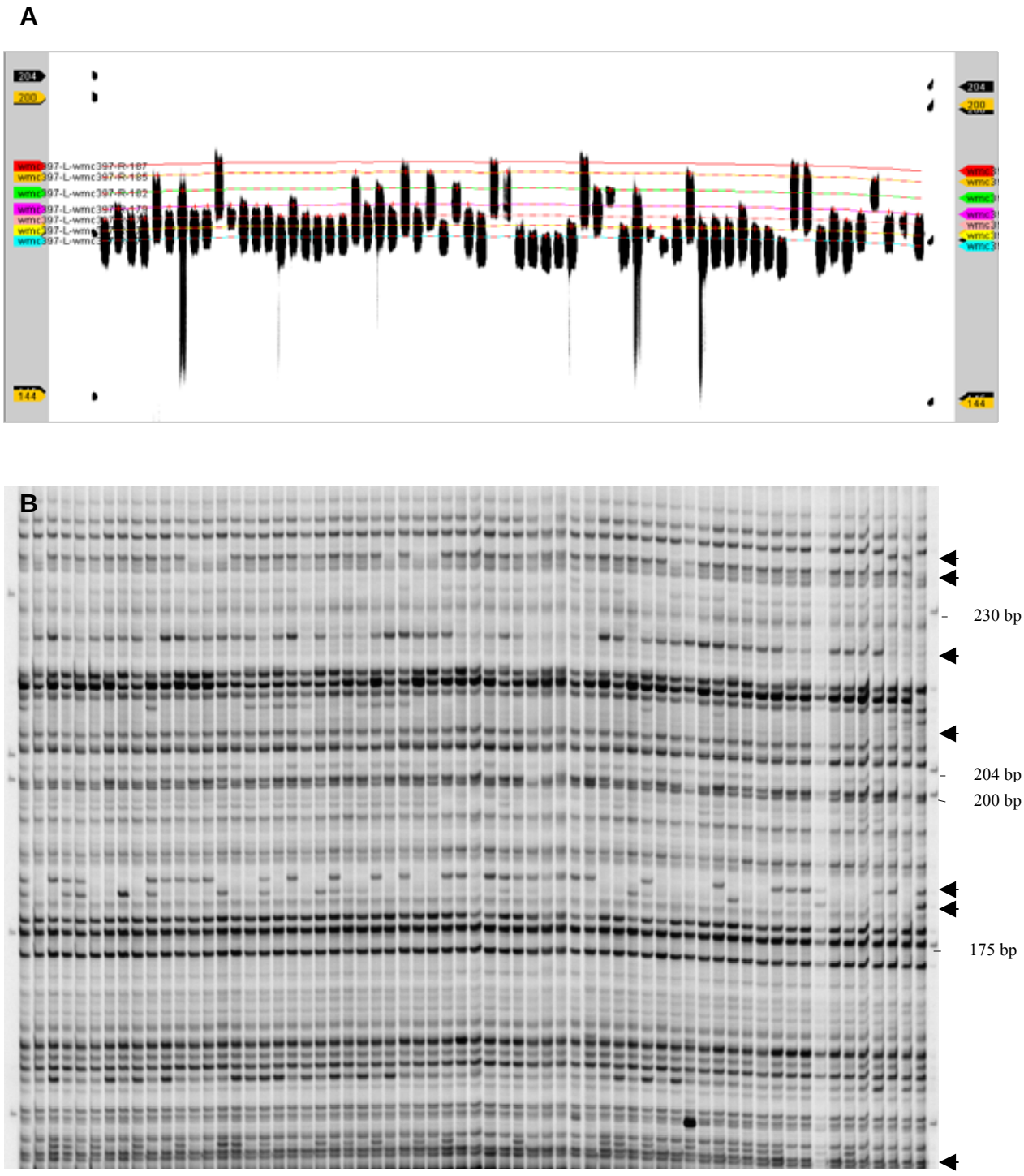


Figure 3.1 Banding patterns of SSR and AFLP markers separated on 6.5% denaturing polyacrylamide gel and visualized by Li-Cor DNA sequencer. **A**, The 7 alleles of SSR marker *Xwmc397* scored with Saga software. **B**, AFLP primer combination mAGTG-eACT. Solid arrows indicate polymorphic AFLP fragments.

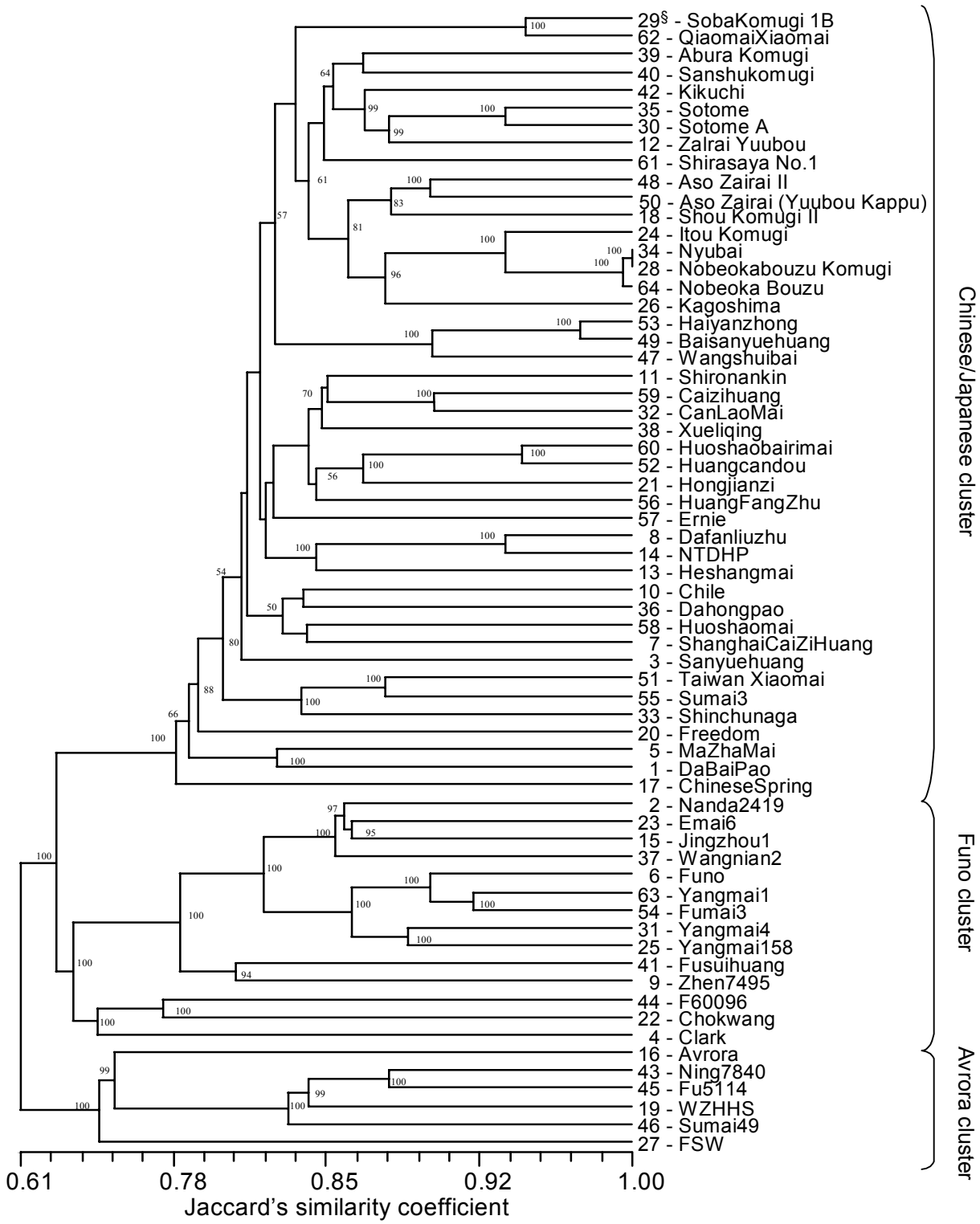


Figure 3.2 UPGMA dendrogram based on Jaccard's similarity coefficient from AFLP and SSR marker data showing genetic relationships among the 64 wheat accessions used in this study. Bootstrap values (%) for each branch point are indicated if they are >50%. [§]Accession ID

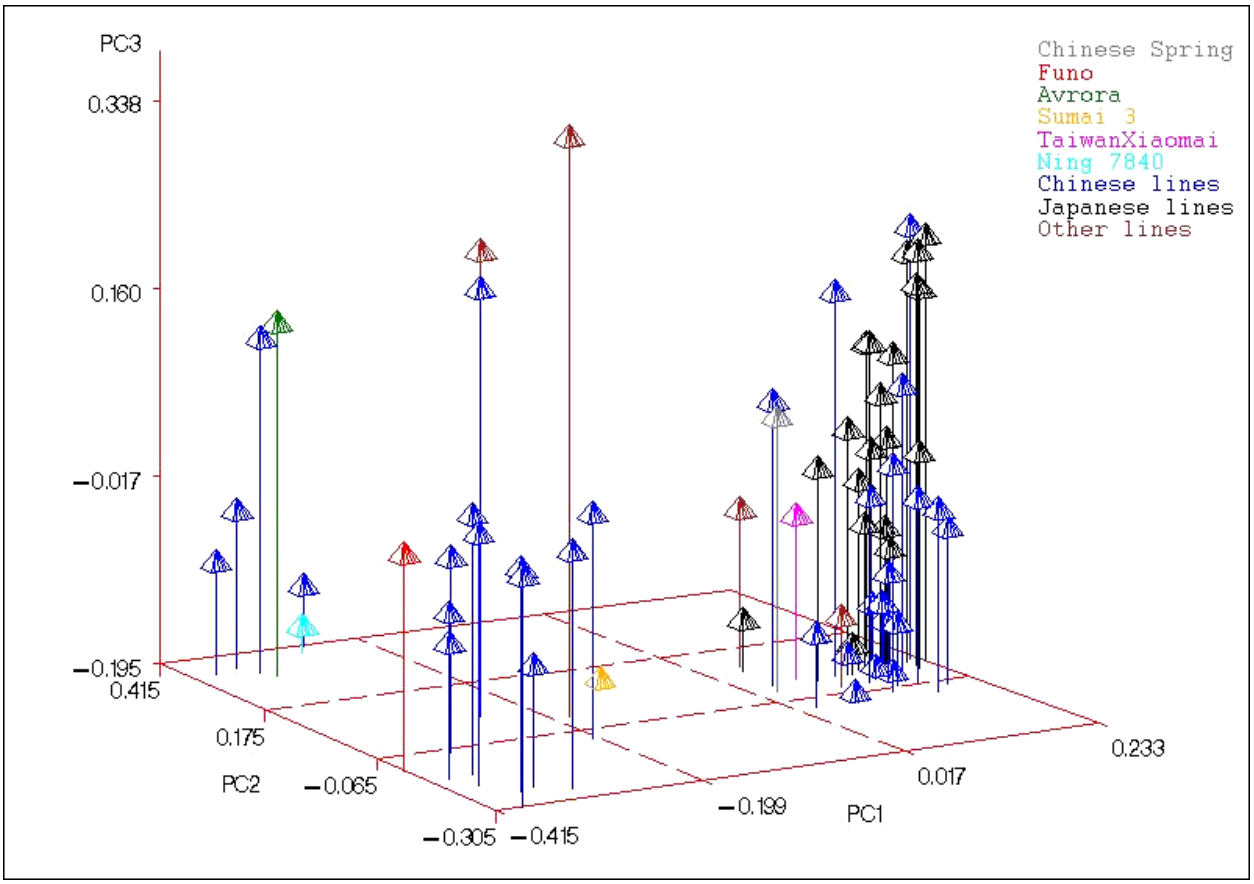


Figure 3.3 Genetic relationships among 64 accessions with different levels of FHB resistance revealed by principal component analysis based on AFLP and SSR marker data.

Table 3.2 Allele sizes of the SSR markers that have been reported to be tightly linked to QTL for FHB resistance in Sumai 3 and Wuhan-1 for 64 wheat accessions.

ID	Accession	2DL		3BSc		4B			5AS						6BS							3BS				
		Xwmc144	*Xwmc245*	*Xwmc777*	*Xwmc612*	*Xwmc48*	*Xgwm113*	*Xgwm165*	*Xgwm293*	*Xgwm415*	*Xwmc705*	*Xgwm129*	*Xbarc117*[6]	*Xgwm304*	*Xgwm518*	*Xwmc494*	*Xgwm508*	*Xwmc398*	*Xwmc105*[6]	*Xwmc397*	*Xgwm219*	*Xgwm389*	*Xbarc75*	*Xgwm533*[6]	*Xbarc133*	*Xgwm493*
1	DaBaiPao	159	165	156	274	216	166	274	218	150	188	241	239	231	185	231	200	159	353	177	208	158	128	156	140	212
2	Nanda 2419	161	166	133	296	209	168	279	214	152	160	239	235	217	201	241	200	159	377	177	206	134	nd	160	142	nd
3	Sanyuehuang	161	166	133	280	218	168	281	218	150	186	247	243	233	178	276	200	175	367	185	199	150	128	160	109	162
4	Clark	159	166	111	302	218	168	276	214	152	158	235	239	235	187	220	N	166	nd	179	199	134	128	132	134	180
5	MaZhaMai	157	165	147	284	216	168	272	218	148	198	249	nd	229	185	226	200	159	349	175	194	150	nd	156	130	212
6	Funo	161	168	108	298	209	168	276	214	150	156	239	243	237	180	231	200	166	353	187	194	nd	nd	134	138	160
7	ShanghaiCaiZiHuang	162	165	150	nd	nd	166	nd	nd	148	184	nd	239	231	209	235	N	nd	343	173	182	150	128	160	145	212
8	Dafanliuzhu	159	166	131	284	216	168	274	216	150	184	244	239	233	209	241	200	168	343	179	192	150	128	160	130	212
9	Zhen 7495	157	166	111	302	218	168	274	218	150	156	239	239	237	183	226	200	166	nd	173	208	134	nd	109	145	nd
10	Chile	159	165	144	268	216	168	264	218	150	184	241	239	233	185	226	200	176	nd	185	192	154	128	156	109	216
11	Shironankin	159	165	131	282	216	168	279	218	150	193	249	239	233	175	272	200	174	353	179	194	154	128	160	152	214
12	Zalrai Yuubou	159	166	133	282	214	168	272	218	150	193	241	239	231	209	233	N	172	343	177	199	152	126	163	140	214
13	Heshangmai	159	166	131	280	216	168	276	216	148	195	244	243	233	187	230	N	168	343	179	199	152	128	162	109	200
14	NTDHP	157	166	133	282	216	168	274	216	150	184	247	239	231	209	239	200	172	343	177	192	150	128	157	130	212
15	Jingzhou 1	159	165	111	302	209	168	276	216	150	156	239	239	235	201	239	N	159	nd	173	204	137	128	157	138	182
16	Avrora	161	166	111	292	216	168	261	214	150	153	237	243	231	185	226	N	159	nd	182	189	nd	nd	132	145	162
17	Chinese Spring	157	165	150	300	214	168	264	216	150	193	241	239	235	181	235	200	176	336	179	194	152	126	171	109	214
18	Shou Komugi II	159	166	150	239	216	168	274	218	150	191	244	239	233	209	237	200	159	343	179	202	154	128	162	140	214
19	WZHHS	159	166	129	280	216	166	276	218	150	191	241	239	237	178	272	200	172	nd	179	199	152	128	160	nd	160
20	Freedom	155	166	144	302	214	168	270	212	152	156	237	239	233	164	223	200	159	nd	185	171	134	126	152	138	162
21	Hongjianzi	161	168	133	287	218	170	274	218	150	195	241	239	231	187	237	N	168	nd	177	209	152	126	157	142	218
22	Chokwang	162	166	163	307	216	168	274	216	150	153	249	243	231	183	230	N	159	353	182	206	150	128	157	140	214
23	Emai6	159	166	131	298	209	168	279	216	150	156	244	239	237	185	229	200	159	377	173	204	134	nd	157	145	nd
24	Itou Komugi	155	166	144	276	209	168	nd	216	150	189	244	239	229	209	239	200	159	339	179	206	152	128	159	140	214
25	Yangmai 158	159	166	111	302	209	168	276	216	150	156	239	239	235	180	231	200	166	349	187	194	nd	nd	132	138	160
26	Kagoshima	155	166	133	282	216	168	270	216	152	191	244	239	233	175	272	200	174	343	182	208	152	128	157	140	210
27	FSW	157	165	153	307	212	168	272	220	150	178	247	239	237	175	272	200	172	341	177	199	152	128	160	140	214
28	Nobeokabouzu Komugi	157	166	147	284	212	168	272	218	150	188	244	239	233	209	239	200	159	341	179	206	150	128	157	140	214
29	SobaKomugi 1B	157	166	133	279	nd	168	281	220	150	195	249	239	235	183	272	N	172	343	177	199	154	128	160	109	210
30	Sotome A	159	166	133	282	216	168	272	207	150	198	241	239	233	178	270	N	172	346	179	204	150	126	157	140	212
31	Yangmai 4	161	168	108	300	209	168	276	216	150	156	239	239	235	178	230	200	159	nd	173	202	nd	nd	132	138	160
32	CanLaoMai	161	166	131	284	216	170	274	218	150	193	244	239	233	183	243	200	175	nd	185	189	154	128	162	140	220
33	Shinchunaga	157	166	131	280	216	168	272	216	150	188	244	239	235	209	231	N	172	343	175	194	152	128	160	138	212
34	Nyubai	157	166	147	284	212	168	272	218	150	188	244	239	233	209	239	200	159	341	179	206	150	128	157	140	214
35	Sotome	159	166	133	282	218	168	274	218	150	195	244	239	233	178	270	N	159	nd	179	204	158	126	109	138	214

Numbers are amplicon sizes (in bp) for the respective marker in wheat lines. Amplicon sizes of the five 3BS SSR marker are sizes from directly labeled primers plus the 18bp M13 tailing primer. N - null allele. nd - no data. PIC - polymorphism information content

Table 3.2. Continued

ID	Accession	2DL		3BSc		4B			5AS						6BS							3BS				
		wmc144	*wmc245*	*wmc777*	*wmc612*	*wmc48*	*Xgwm113*	*Xgwm165*	*Xgwm293*	*Xgwm415*	*wmc705*	*Xgwm129*	*Barc117*[§]	*Xgwm304*	*Xgwm518*	*wmc494*	*Xgwm508*	*wmc398*	*wmc105*[§]	*wmc397*	*Xgwm219*	*Xgwm389*	*Barc75*	*Xgwm533*[§]	*Barc133*	*Xgwm493*
36	Dahongpao	159	165	131	279	218	168	276	216	150	193	241	239	231	189	241	200	176	nd	182	202	152	128	159	145	210
37	Wannian 2	159	168	129	298	209	168	276	216	150	156	237	239	237	219	239	N	159	377	173	206	134	nd	160	140	214
38	Xueliqing	159	166	150	279	218	168	272	218	152	184	241	239	233	209	243	200	168	343	179	192	152	126	163	147	210
39	Abura Komugi	157	166	133	289	212	168	276	216	150	189	249	239	233	nd	233	200	168	339	179	199	137	128	159	140	212
40	Sanshukomugi	157	166	129	289	209	168	272	216	150	186	239	239	235	183	235	200	175	349	185	204	152	128	154	140	212
41	Fusuihuang	159	166	133	298	216	168	274	212	152	158	241	235	215	187	237	200	198	349	187	202	134	nd	157	142	nd
42	Kikuchi	157	nd	129	284	214	166	272	218	150	186	241	nd	233	180	266	N	172	339	179	204	154	128	160	140	214
43	Ning 7840	157	166	111	292	218	168	276	218	150	186	239	239	233	209	231	N	172	343	175	194	152	128	160	140	212
44	F 60096	159	166	111	292	209	168	276	218	150	186	241	239	233	209	231	N	174	343	175	194	150	128	159	140	212
45	Fu 5114	157	166	108	287	209	168	276	nd	152	186	249	239	233	178	nd	N	166	nd	179	nd	152	128	160	142	214
46	Sumai49	157	166	111	292	218	168	270	216	150	156	241	239	237	209	231	N	174	341	175	194	137	126	163	nd	212
47	Wangshuibai	157	166	156	311	214	168	274	220	150	176	244	239	235	180	272	200	172	343	175	199	150	128	159	136	212
48	Aso Zairai II	155	166	150	239	212	168	279	216	152	195	249	239	237	209	237	200	159	343	177	204	158	128	159	140	200
49	Baisanyuehuang	159	166	156	274	214	168	276	218	150	178	244	239	231	183	276	200	172	346	175	199	150	128	160	109	214
50	Aso Zairai (Yuubou Kappu)	157	166	150	239	214	168	276	216	150	195	244	239	237	209	237	200	159	343	177	209	154	128	159	140	214
51	Taiwan Xiaomai	157	166	129	282	216	168	274	218	148	186	241	239	233	209	231	N	172	nd	175	194	152	128	160	140	212
52	Huangcandou	161	166	133	287	218	168	274	218	150	184	244	239	233	175	239	200	172	346	177	206	150	128	162	142	214
53	Haiyanzhong	159	166	156	287	216	168	276	218	150	178	247	nd	231	183	276	200	172	346	177	199	150	128	159	109	214
54	Fumai 3	159	168	nd	298	209	168	nd	214	150	156	237	239	235	180	230	200	166	349	187	204	134	nd	132	138	160
55	Sumai 3	157	166	131	282	209	168	276	218	150	186	239	239	233	209	231	N	172	343	175	194	152	128	160	140	212
56	HuangFangZhu	161	166	131	284	216	170	276	216	150	193	241	239	235	209	237	N	168	nd	179	192	152	128	156	109	214
57	Ernie	155	166	147	302	216	168	272	212	152	156	239	239	237	185	229	N	166	349	179	204	134	128	109	142	160
58	Huoshaomai	161	168	147	272	218	168	274	218	150	186	244	239	229	175	243	200	159	346	182	192	152	126	157	142	210
59	Caizihuang	159	166	133	284	218	168	276	218	150	186	247	239	231	185	243	200	174	367	182	194	150	128	160	142	220
60	Huoshaobairimai	161	168	133	287	216	170	274	218	152	186	244	239	233	175	241	200	174	343	177	206	152	128	159	140	216
61	Shirasaya No.1	157	166	133	282	218	168	274	220	150	184	241	239	233	185	230	200	168	341	175	194	152	128	160	136	162
62	Qiaomai Xiaomai	159	166	133	280	212	168	276	214	150	156	239	239	235	180	231	200	166	349	187	206	150	128	160	109	210
63	Yangmai 1	159	168	113	298	209	168	276	216	150	156	237	239	237	180	231	200	166	349	187	206	nd	nd	132	138	160
64	Nobeoka Bozu	157	166	147	284	212	168	272	218	150	188	244	239	233	209	239	200	159	341	179	206	150	128	157	140	214
PIC		0.70	0.43	0.87	0.92	0.77	0.23	0.77	0.68	0.36	0.88	0.80	0.28	0.75	0.85	0.91	0.45	0.78	0.82	0.82	0.86	0.81	0.56	0.85	0.82	0.84
No. of alleles		5	3	12	18	5	3	8	6	3	14	7	3	7	12	16	2	8	9	7	11	6	2	12	10	11
Haplotypes[¶]		nd		4		nd			24						23							21				

[¶]Haplotype based on Sumai 3/non-Sumai 3 alleles. [§]Marker closest to the QTL for FHB resistance.

Table 3.3 Haplotypes of 59 Asian accessions and five cultivars from other countries based on allele variation of five SSR markers tightly linked to the 3BS QTL from Sumai 3.

	Haplotypes based on Sumai 3/non-Sumai 3 alleles																						
SSR marker	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	**No. of alleles**	**PIC***
Xgwm389	■	■	■	■	■	■	□	□	□	■	■	□	□	□	□	□	■	□	□	□	□	6	0.81
Xbarc75	■	■	■	■	■	■	■	■	■	□	■	□	■	■	□	■	□	□	■	□	□	2	0.56
Xgwm533	■	□	■	■	□	■	□	■	■	□	□	□	□	□	■	■	□	□	□	■	□	12	0.85
Xbarc133	■	■	□	■	■	□	■	□	■	■	□	■	□	■	■	□	□	□	□	□	□	10	0.82
Xgwm493	■	■	■	□	□	□	■	■	□	□	□	■	■	□	□	□	□	■	□	□	□	11	0.84
No. of lines	3	1	1	1	3	3	3	2	1	1	3	1	2	8	1	6	4	2	6	1	11		

Darkened boxes represent Sumai 3 alleles and open boxes represent non-Sumai 3 alleles

* Polymorphism information content

Table 3.4 Number of accessions with Sumai 3 SSR alleles at 3BS, 5AS and 6BS and their relationship with FHB resistance

Chromosomal region	No. of Sumai 3 allele	No. of accessions*	PSS¶	No. of accessions and FHB resistance			
				R	R	MS	S
3BS	≤ 1	23	41.9 ± 24.2	8	6	6	3
	2 or 3	34	36.1 ± 21.3	14	10	8	2
	> 3	6	26.5 ± 16.4	3	3	0	0
5AS	≤ 1	8	56.6 ± 27.8	1	1	3	3
	2 or 3	35	34.5 ± 19.4	15	11	8	1
	> 3	20	34.6 ± 21.6	9	7	3	1
6BS	≤ 1	38	40.5 ± 22.6	13	13	7	5
	2 or 3	18	36.5 ± 20.0	7	5	6	0
	> 3	7	22.1 ± 21.0	5	1	1	0
All of 3BS, 5AS and 6BS	≤ 5	25	43.4 ± 23.0	7	6	9	3
	> 5 and ≤10	33	35.8 ± 20.8	14	12	5	2
	> 10	5	17.0 ± 13.6	4	1	0	0

* Exclude MaZhaMai, which did not have phenotypic data

¶ Mean ± standard deviation of proportion of symptomatic spikelets of the lines

CHAPTER 4 - QTL for three types of resistance to Fusarium head blight in a wheat population from Wangshuibai/Wheaton

Introduction

Various statistical methods have been developed for QTL mapping. The two most commonly used methods for QTL mapping are interval mapping (IM) and composite interval mapping (CIM). Powerful computer software programs are now available to map QTL with IM, CIM, and other statistical methods (Nelson, 1997; Manly *et al.* 2001; Broman *et al.* 2003; Wang *et al.* 2006). These software packages have been commonly used in wheat FHB resistance QTL mapping studies, for example, Lin *et al.* 2004 and Zhou *et al.* 2004. IM uses two observable flanking markers to define an interval within which to search for QTL along the chromosome. A map function, such as Kosambi (Kosambi, 1944), is used to translate the recombination frequency to distance or vice visa. A logarithm of odds ratio (LOD) score is calculated at each increment in the interval. These scores are plotted as a profile along the chromosome. When a peak has exceeded a threshold value, it indicates that a QTL may be at this location (Lander and Botstein 1989). Composite interval mapping method is an extension of IM. CIM is a combination of maximum likelihood-based interval mapping and multiple regression. CIM selects certain markers into the model as cofactors to control the effects of non-target QTL. It fits parameters for a target QTL in one interval while simultaneously fitting partial regression coefficients for background markers to account for variance caused by non-target QTL. In theory, CIM gives more power and accuracy than simple IM because the effects of other QTL are not present as residual variance (Jansen and Stam 1994; Zeng 1994). Both methods were used in present QTL mapping study.

In this study, a Wangshuibai/Wheaton recombinant inbred population was used to validate the FHB resistance QTL detected in other Wangshuibai-derived populations under field conditions. Also, new FHB resistance QTL in Wangshuibai may be detected under different genetic background and under greenhouse conditions. So far, most of Wangshuibai QTL

mapping experiments were conducted under field conditions and mainly used to detect type II resistance QTL (Lin *et al.* 2004; Lin *et al.* 2006; Mardi *et al.* 2005; Zhang *et al.* 2004; Jia *et al.* 2005). Environmental factors in these mapping experiments may have affected the disease evaluation and the detection of QTL with minor effects. In addition, the low DON content type of resistance has been characterized only under field conditions in one previous study (Ma *et al.* 2006a). In the present study, the Wangshuibai/Wheaton population was repeatedly evaluated for all three types of FHB resistance under controlled greenhouse conditions to detect all three types of resistance QTL, especially QTL for low DON content.

Materials and methods

Plant materials and pathogen

A population of 139 $F_{5:6}$ recombinant inbred lines (RILs) was developed by single seed descent from a cross between Wangshuibai and Wheaton. Wangshuibai is a highly FHB-resistant Chinese landrace and Wheaton is a hard red spring FHB-susceptible cultivar released from the University of Minnesota, USA. The inoculum of *F. graminearum* was a field isolate (GZ 3639) originating in Kansas. This isolate has been well characterized for its high aggressiveness and DON production (Desjardins *et al*. 1996).

Evaluation of FHB and DON content

All RILs were evaluated for FHB resistance in the greenhouse at Kansas State University from 2003 to 2005. The evaluation procedures were described in Chapter 2. Type I resistance was evaluated in two experiments in 2004 and 2005 with the spray inoculation method and measured as the proportion of infected florets (PIF). Type II resistance was evaluated in three experiments in 2003 (spring and fall) and 2005 (spring) by single-spikelet inoculation and measured as the proportion of symptomatic spikelets (PSS). DON content was determined in parts per million (ppm) by gas chromatography–mass spectrometry (GC–MS) in the wheat kernels harvested from Fusarium-inoculated spikes (Mirocha *et al.* 1998). DON data were collected from two single spikelet inoculation experiments in 2003 and two spray inoculation experiments in 2004–2005.

AFLP analysis

Genomic DNA of each RIL was isolated from leaf tissue by the CTAB method (Saghai Maroof *et al*. 1984). For AFLP analysis, DNA restriction digestion (with *Pst*I and *Mse*I), adapter ligation, and PCR amplification were carried out as described by Bai *et al*. (2003). Pre-amplification was conducted with a *Pst*I primer (5'- ACTGCGTACATGCAG) and an *Mse*I primer (5'-GATGAGTCCTGAGTAA). In total 28 IRD700/800-dye-labeled *Pst*I primers were combined with 30 *Mse*I primers with 2 to 4 selective nucleotides at the 3' ends of both primers for selective amplification. DNA from the parental lines, a bulk of the 10 most resistant RILs, and a bulk of the 10 most susceptible RILs were screened with 110 primer combinations between 15 *Pst*I primers (with selective nucleotides of ACA, ACT, ACTG, AG, AGC, AGG, AGT, CAG, CAT, CATG, CGA, CGT, CTC, GCTG, GTG, and TGC) and 29 *Mse*I primers (with selective nucleotides of ACAG, ACGC, ACGT, ACTG, AGAC, AGC, AGCT, AGGC, AGTG, CAA, CACG, CAG, CAT, CGAT, CGCT, CGTA, CTA, CTCG, CTG, CTGA, CTT, GAC, GCAG, GCAT, GCG, GTG, TCGA, TGC, and TGCG). The polymorphic primers were chosen to screen the population. The IRD-labeled AFLPs were analysized in a Li-COR 4200 DNA analyzer. After addition of 5 µl of formamide dye, the AFLP product was denatured at 95°C for 5 min and loaded into wells of a 25-cm sequence gel made from 6.5% Gel Matrix (Li-Cor, Inc. Lincoln, NE). The gel was run for 210 min in 1×TBE at constant power of 40 w and voltage of 1500 V. The collected gel images were stored in an attached computer and were scored with Saga software (Li-Cor, Inc. Lincoln, NE).

Analysis of simple sequence repeats (SSR)

About 1200 SSRs from different research groups including WMC (Somers *et al*. 2004), GWM (Röder *et al*, 1998), BARC (Song *et al*, 2005), CFA and CFD (Guyomarc'h *et al*. 2002; Sourdille *et al*. 2003), and GDM (Pestsova *et al*. 2000) were screened for polymorphism between the parental lines. A total of 248 polymorphic SSR markers were selected and used for genotyping the mapping population, as described by Bai *et al*. (2003) and as modified in chapter 3. The SSR fragments were determined in a Li-Cor 4200 DNA Analyzer under the same conditions as described for AFLP. The gel images were collected, printed, and scored by visual inspection.

Experimental design and data analysis

All experiments were arranged in a randomized complete block design. The variance analyses of disease severities from each experiment (greenhouse cycle) and across all three experiments were performed using the GLM procedure of the software package SAS (SAS Institute Inc, Cary, NC). Both genotypic and experimental effects were treated as random effects. Broad-sense heritabilities and their 90% confidence intervals for line means were estimated according to Shen *et al.* (2003a) and Knapp *et al.* (1985), respectively.

Linkage maps of AFLP and SSR were constructed with JoinMap 3.0 (van Ooijen and Voorrips, 2001) with the Kosambi mapping function (Kosambi, 1944). The QTL analyses were conducted separately for FHB and DON data. Simple (SIM) and composite interval mapping (CIM) were performed with WinQTLCart 2.5 (Wang *et al.* 2006) on the individual line means from each individual experiment and on the overall line means across all experiments. Five markers and a 10-cM window size were used as a background control in CIM analysis. Permutation tests were performed to estimate appropriate significant threshold for both SIM and CIM (Churchill and Doerge 1994). Based on 1,000 permutations, a LOD threshold of 2.0 was set to declare a significant QTL in both SIM and CIM. A multiple regression model was used to estimate the total phenotypic variation of a trait explained by the additive effects of all detected QTL for that trait. This model included a single marker with the highest determination coefficients (r^2) value from each QTL detected in CIM. The statistical analyses were performed with the SAS REG procedure. The regions of chromosome locations coresponding to the detected QTL were determined with the 1-LOD support interval method (Lander and Botstein 1989).

Results

FHB infection and DON content in RILs

The frequency distributions for mean PIF (type I), mean PSS (type II) and DON content were continuous, with broad phenotypic variation among the RILs (Fig. 4.1). Mean PSS ranged from 12.8% to 100% across the three experiments in which single spikelet inoculation was used. Mean PIF ranged from 9.6% to 39.5% over the two experiments in which plants were spray-inoculated. DON content in inoculated spikes varied from 2.72 to 243 ppm among RILs over the

four experiments (Fig. 1). Normality tests using the PROC UNIVARIATE procedure of SAS indicated that DON content deviated from normal distribution whereas type I and type II resistance scores did not (data not shown), so logarithm-transformed DON data were used in further statistical analysis.

Mean DON content in single-floret inoculation experiments (14.2 ppm) was about half of that in spray inoculation experiments (27.9 ppm). The correlation coefficient was 0.23 ($P < 0.01$) for PIF between the two experiments. The correlation coefficients ranged from 0.53 to 0.61 ($P < 0.0001$) for PSS among the three single spikelet inoculation experiments and from 0.34 to 0.52 ($P < 0.0001$) for DON content among these experiments. The variances of experiment, genotype, and experiment by genotype interaction were significant for all traits. The broad-sense heritability calculated from line means was low for type I resistance (0.36), but was high for type II (0.75) and DON accumulation (0.71) resistance (Table 4.1).

QTL for type I resistance

Type I resistance, as reflected by PIF, was evaluated in two independent experiments. Composite interval mapping of PIF from a single experiment or combined from the two experiments detected five QTL, on chromosome arms 3BS, 4BS, 3AS, 5DL, and 5AS (Fig. 4.2 and Fig. 4.3). Each of the five QTL was significant in only one of the two experiments. The QTL on 3BS and 5DL were also significant when the mean over the two experiments was used (Fig. 4.2, Fig. 4.3, and Table 4.2). The QTL on 3BS and 4BS have relatively larger effects than the others, based on a single year's data, and explained 19.7% and 15.6%, respectively (Table 4.2). Four QTL for type I resistance were from Wangshuibai and one on 4BS was from Wheaton. In addition, a small QTL was observed on 3BS near the centromere region, but it was not significant in either of the two experiments at LOD = 2.

QTL for type II resistance

Type II resistance was evaluated in three experiments. Composite interval mapping using PSS from a single experiment, or combined PSS from the three experiments, detected seven significant QTL for type II resistance, on chromosome arms 3BS (Fig. 2), 1AS, 5AS, 5DL, 3DL, and 7AL (Fig. 4.3). All of them were from Wangshuibai. The QTL on distal end of 3BS showed the largest effect in all three experiments (Table 4.2). This QTL explained 17.6% to 26.5% of the phenotypic variation in individual experiments and 33.9% of the phenotypic variation when data

were combined from all three experiments. Another QTL significant for all three experiments was on 3DL and explained 5.2% to 9.7% of the phenotypic variation in the three experiments. The other five QTL were significant in only one or two experiments, and showed relatively smaller effects on type II resistance (Table 4.2). The QTL on 5DL was significant in only one experiment and not significant when the mean over three experiments was used, thus this QTL may be the least reliable one for type II resistance. Of the seven putative QTL detected for type II resistance, three QTL (on 3BS, 3BSc, and 5AS) were in the same genomic regions as those for type I resistance and DON accumulation resistance (Table 4.2). In addition, the QTL on 7AL also showed small effects on DON accumulation resistance.

The three markers linked to QTL that affected all three types of resistance on 3BS, 5AS, and 5DL were chosen to evaluate their genotypic effects on type II resistance (Table 4.3). This was done by comparison of phenotypes for type II resistance among RILs carrying the FHB-resistant or -susceptible alleles of the three markers. All phenotype comparisons refer to change in % symptomatic spikelets among specific genotypes. The resistance allele of *Xbarc*147 showed the largest effect on FHB resistance among the three markers. The mean PSS of the RILs with one of the three resistance marker alleles was lower than the mean PSS of those lines with none. The mean PSS value of RILs with all three resistance marker alleles (R/R/R) was 28.9%, whereas the mean PSS for the RILs with all three susceptible alleles (S/S/S) was 73.7%.

QTL for low DON content

Seven QTL were detected, on chromosome arms 3BS (2 QTL), 5AS, 1AS, 5DL, 1BL, and 7AL, for low DON content in four experiments (Table 4.2, Fig. 4.2, and Fig. 4.3). The QTL at the distal end of 3BS showed the largest effect in all the experiments and explained 9.6% to 30.6% of phenotypic variation across four experiments, with an R^2 of 35.6% when means of RILs across four experiments were used. The other six QTL showed smaller effect, with R^2 values ranging from 3.6 to 12.2%. The QTL on 1AS, 1BL, and 7AL were detected once in these experiments. Four QTL (on 3BS, 1AS, 5DL, and 1BL) were significant when average DON content from all four experiments was analyzed. The QTL on 3BS, 3BSc, 5AS, 5DL, and 7AL also showed significant effects on type I and/or type II resistance (Fig. 4.2, Fig. 4.3, and Table 4.2). The location of the QTL on 1A for low DON content was different from that for type II resistance on the same chromosome. The QTL for type II resistance on 1AL was from

Wangshuibai, whereas the QTL for low DON on 1AS was from Wheaton. The other six QTL for low DON content were from Wangshuibai (Table 4.2).

Three QTL-linked marker loci (Table 4.4) were chosen to estimate their effectiveness for marker-assisted selection. Genetic effects of QTL on 3BS, 5AS, and 5DL were estimated in the same way as for type II resistance. All phenotype comparisons refer to change in the mean DON content among RILs carrying specific genotypes. When RILs carried all three resistance alleles (R/R/R), their mean DON content was 6.7 ppm, in contrast to 66.5 ppm for the RILs carrying all three susceptible alleles (S/S/S) (Table 4.4). The allele of *Xbarc*147 associated with resistance showed the largest DON-reducing effect.

Discussions

Type I resistance is unstable while type II and type III resistance are relatively stable and not easy affected by nongenetic factors. The high broad-sense heritabilities for type II and III resistance indicated high reproducibility of data from different experiments. A relatively lower heritability for type I resistance implied that type I resistance was unstable and more vulnerable to nongenetic variation (Bai and Shaner 2004; Kolb *et al*. 2001).

Based on cross-referenced markers, the 3BS and 5AS QTL in Wangshuibai were most likely the same as *Qfhs.ndsu-3BS* and *Qfhs.ndsu-5AS* in Sumai 3. QTL on 3BS and 5AS detected in this study were reported in previous studies as the major FHB resistance QTL in many Chinese wheat lines (Chen *et al*. 2006; Somers *et al*. 2003). These two QTL showed effects on all three types of FHB resistance in this study.

The 3BS QTL in Wangshuibai and Sumai 3 may be different alleles of *Qfhs.ndsu-3BS*. The 3BS QTL in Wangshuibai had a smaller effect in different genetic backgrounds than that of Sumai 3, and the haplotype pattern of the SSR markers linked to the QTL in Wangshuibai was different from that of Sumai 3 (McCartney *et al*. 2004; Yu *et al.* 2006). This QTL showed the largest effect on type II resistance and explained up to 33.9% of the phenotypic variation, which was larger than that generated from field experiments (9.0% to 17.0%) in other Wangshuibai-derived mapping populations (Lin *et al.*, 2004; Ma *et al.* 2006a; Mardi *et al.* 2005). This may be due to larger environmental effects on type II resistance in the field than under greenhouse conditions.

Effects from environmental factors, genetic backgrounds, and the low heritability of type I resistance may be responsible for the difference of detected type I resistance QTL between this study and previous studies. The QTL on 3AS was reported for type I resistance in Frontana (Steiner *et al.* 2004), but the relationship between the 3AS QTL detected in Frontana and Wangshuibai is unknown because of the lack of common markers between the two maps from the two studies. QTL on 4BS originated from Wheaton and showed minor effects only on type I resistance in this study. The QTL on 4B (*Qfhi.nau-4B*) and 5A (*Qfhi.nau-5A)* showed large effects on type I resistance in Wangshuibai/Nanda 2419 population (Lin *et al.* 2006), which is different from the result in this study. The QTL on 5DL identified in this study has not been reported previously. Neither the QTL on 3BS nor that on 5DL for type I resistance was detected in Wangshuibai/ Nanda 2419 (Lin *et al* 2006).

The type II resistance QTL on 3BSc and 3DL were reported in previous studies, while three QTL with minor effects on type II resistance detected in this study seemed to be new. The 3BSc QTL was detected in Wangshuibai and other cultivars such as Sumai 3, CM-82036, Maringa, and W14 (Buerstmayr *et al.* 2002; Chen *et al.* 2006; Lin *et al.* 2004; Shen *et al.* 2003b; Somers *et al.* 2003; Zhou *et al.* 2004). The QTL on 3DL detected in this study may be the same QTL as *Qfhs.nau-3D* reported in Wangshuibai based on cross-referenced markers (Lin *et al.* 2004). QTL on chromosome arms 7AL, 1AL, and 5DL have not been reported before.

Environmental conditions of FHB resistance evaluation may determine the set of FHB resistance QTL detected. Previous studies indicated that type I and II resistance might be controlled by different QTL under field conditions (Lin *et al.* 2006; Buerstmayr *et al.* 2003a; Steiner *et al.* 2004). In this study, the QTL on 3BS had the largest effect on both type I and type II resistance under greenhouse conditions. This differed from Lin *et al.* (2006) in that 3BS QTL was not significant for type I resistance in their study under field conditions. QTL on 5AS also showed effects on both type I and II resistance in this study and other previous studies (Chen *et al.* 2006; Somers *et al.* 2003). Thus, the major QTL on 3BS and some other minor QTL for type II resistance also have effects on type I resistance. The QTL on 4BS, 3AS, 1A, 3DL, and 7AL were detected for only one type of resistance, indicating that the expression of the two types of resistance may be modified by different QTL. These modifying QTL usually showed minor effects and may be more vulnerable to nongenetic variation. They may differ between the parents in some studies and not in others. For this reason, these minor QTL should be verified in

multiple tests across different genetic backgrounds and in different environments before they can be widely used in marker-assisted breeding.

Results in this study suggested that type III resistance may be the consequence of type II and type I resistance. Previous study showed that FHB resistance and DON accumulation might be controlled by independent genetic factors (Somers *et al.*, 2003), whereas other studies suggested that type II resistance might be closely associated with low DON content (Abate and McKendry 2005; Chen *et al.* 2006; Lemmens *et al.* 2005). Most FHB resistance QTL detected in this study showed a pleiotropic effect on both low DON and FHB severity. The 3BS and 5AS QTL in Wangshuibai showed effects on both type II resistance and low DON content in this and a previous studies (Ma *et al.* 2006a). Four of the seven QTL for type III resistance on 3BS (2 QTL), 5AS, and 7AL were coincident with the QTL for type II resistance. The QTL on 3BS, 5AS, and 5DL also showed type I resistance. The 1BL QTL showed only type III resistance in this study but it was determined to have a minor effect on type II resistance (Zhou *et al*. 2004). Only minor QTL on 1AS for low DON content showed no association with FHB severity. Thus, QTL for FHB resistance, especially type II, seemed to play an important role in lowering DON content and low DON in infected grain. Breeding selection for FHB resistance may lead to reduction in DON content in wheat cultivars.

Resistance to FHB of Wangshuibai is complex, as indicated by the five to seven QTL that were identified for each of the three types of FHB resistance in the current study. These QTL jointly explained more than 90% of genetic variation for type I resistance and 80% for type II resistance, suggesting that most of the QTL were identified for both types of resistance in this study. It is possible that more QTL for type II resistance are present in Wangshuibai to account for the unexplained portion of genetic variation; however, those undetected QTL most likely have minor effects and are difficult to detect due to environmental variation. For DON accumulation resistance, all QTL together could explain only about 65% of the genetic variation. This may be because a large portion of nongenetic variation is associated with the trait evaluation. For instance, plant growth stage at inoculation, inoculation method, threshing method, and DON testing technique may all affect the accuracy of the DON measurement in a plant (Bai and Shaner, 2004), as well as QTL identification. For this reason, a better protocol is still needed for accurately measuring DON content for QTL mapping of DON accumulation. Based on results from this study, type II resistance is a more stable type of resistance than type I

and III resistance. Also, type I and III resistance were mainly coincident with type II resistance. Marker-assisted selection for QTL on 3BS could gain about 50% FHB resistance from Wangshuibai, but selection for QTL on 3BS, 5AS, and 5DL may increase all three types of resistance, especially DON accumulation resistance.

Conclusions

QTL for the three types of FHB resistance were detected with the 139-RIL population from Wangshuibai/Wheaton. Five QTL for type I resistance were detected, on chromosome arms 3BS, 4BS, 5DL, 3AS, and 5AS; seven QTL for type II resistance were located, on 3BS, 1AL, 5AS, 5DL, 7AL, and 3DL; and seven QTL for low DON content were detected, on 3BS, 5AS, 1AS, 5DL, 1BL, and 7AL. These QTL jointly explained up to 31.7%, 64%, and 52.8% of the phenotypic variation for the three types of FHB resistance, respectively. The QTL on 5AS, the distal end of 3BS, and 5DL contributed to all three types of FHB resistance. QTL on 7AL, 1A, and the proximal end of 3BS showed effects on both type II and III resistance. The broad-sense heritabilities were low for type I resistance (0.36), but high for type II (0.75), and for type III resistance (0.71). Results also suggested that selection for type II resistance may simultaneuously improve type I and DON content (type III) resistance as well. The QTL for FHB resistance identified in Wangshuibai have potential to be used to enhance FHB resistance by pyramiding FHB resistance QTL from different sources.

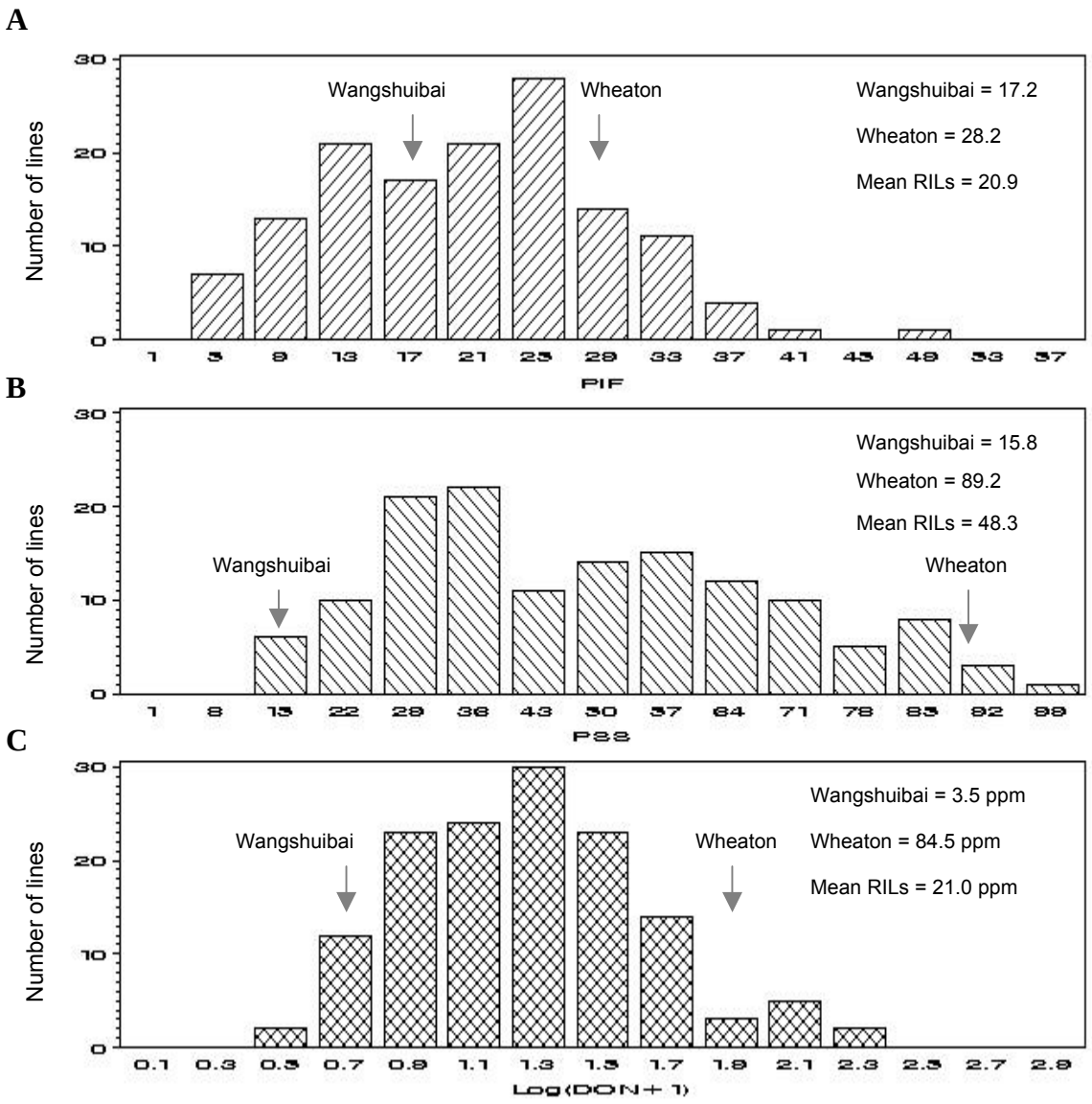


Figure 4.1 Frequency distribution of mean PIF, PSS, and DON content in 139 RI lines. **A**, PIF over two experiments. **B**, PSS over three experiments. **C**, log(DON+1) over four experiments. Arrows indicate values of the parental lines.

Table 4.1 Analysis of variance for type I, type II, and DON accumulation resistance and the broad-sense heritabilities of the three traits estimated for 2003 to 2005 experiments.

FHB resistance	Variable	df	Mean square	F-value	*P*-value	Heritability (90% CI[a])
Type I						0.36
						(0.18 – 0.50)
	Experiments	1	624.7	6.27	< 0.0129	
	RILs	137	283.8	2.85	< 0.0001	
	RILs x experiments	273	182.9	1.84	< 0.0001	
	Error	394	99.6			
Type II						0.75
						(0.68 – 0.80)
	Experiments	2	25648.42	72	<.0001	
	RILs	137	2378.19	6.68	<.0001	
	RILs x experiments	267	606.77	1.7	<.0001	
	Error	527	356.24			
DON content						0.71
						(0.63 – 0.77)
	Experiments	3	11.582	76.25	<.0001	
	RILs	137	0.8934	5.88	<.0001	
	RILs x experiments	409	0.2635	1.73	0.0001	
	Error	534	0.1519			

[a] Confidence interval

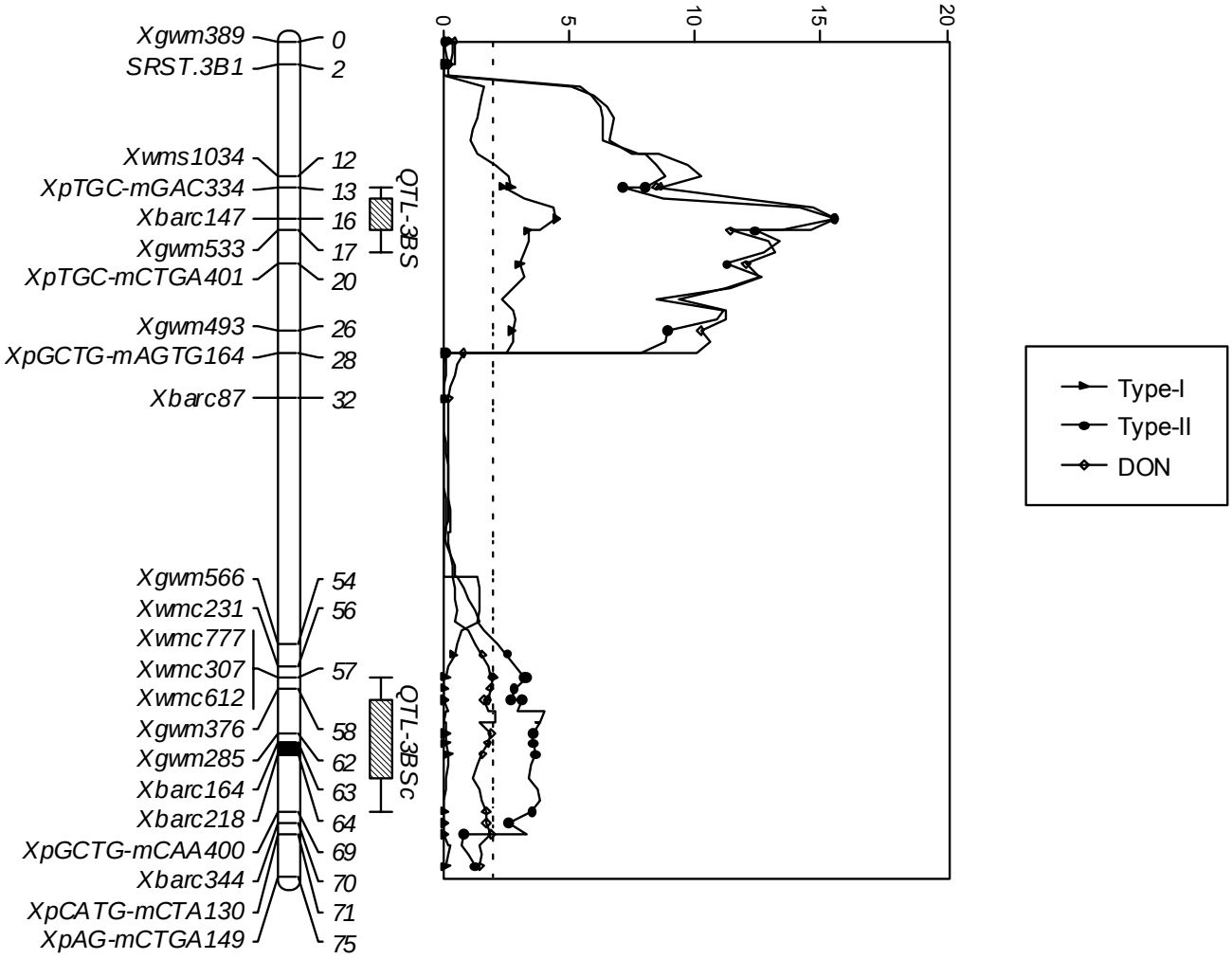


Figure 4.2 Log likelihood ratio (LOD) contours obtained from composite interval mapping with means across two type I experiments, three type II experiments and four DON experiments that show QTL locations for the three FHB resistance traits on chromosome arm 3BS. The black solid bar indicates the approximate location of the centromere.

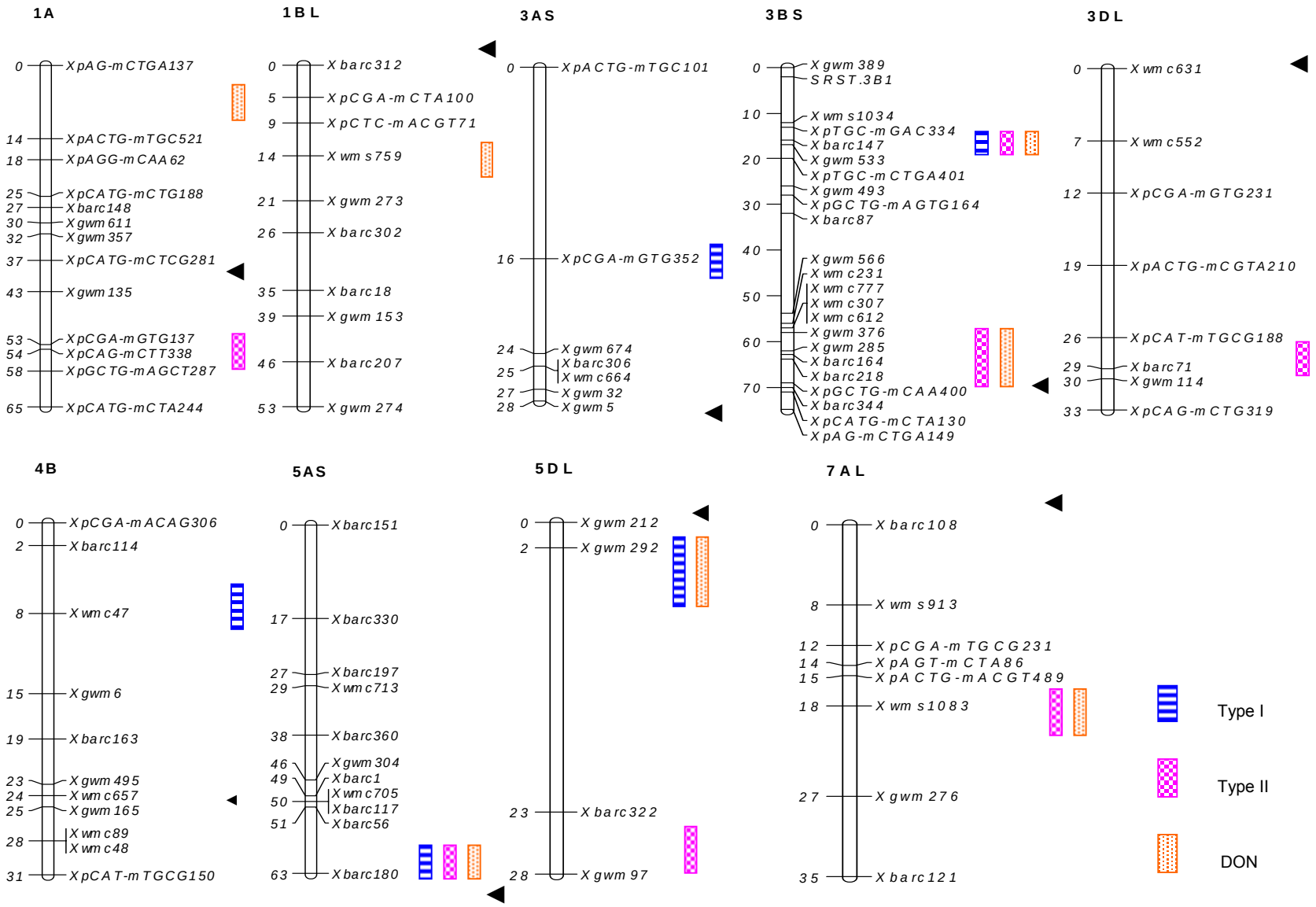


Figure 4.3 FHB resistance QTL detected by composite interval mapping. The bars indicate QTL locations and their lengths represent 1-LOD support interval. The approximate centromere locations are indicated with solid arrows

Table 4.2 Effects of QTL on FHB resistance in the RIL population from Wangshuibai × Wheaton

Type	Chrom.	Marker	Source[a]	03 Spring		03 Fall		2004		2005		Combined[b]	
				LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2	LOD	R^2
Type I													
	3BS	*Xbarc*147	WSB	-	-	-	-	-	-	6.9	19.7*	4.5	13.6*
	4B	*Xwmc*47	WTN	-	-	-	-	5.3	15.6*	-	-	-	-
	3AS	*XpCGA-mGTG*352	WSB	-	-	-	-	-	-	2.6	11.4*	2.2	8.1*
	5DL	Xgwm292	WSB	-	-	-	-	3.1	8.5*	-	-	2.5	6.8*
	5AS	*Xbarc*180	WSB	-	-	-	-	2.1	6.9*	-	-	-	-
Total R^2									24.8		31.7		30
Type II													
	3BS	*Xbarc*147	WSB	11.6	24.6*	11.5	26.5*	-	-	6.1	17.6*	15.6	33.9*
	3BSc	*Xgwm*376	WSB	4.1	9.0*	6.0	11.8*	-	-	-	-	3.8	8.1*
	7AL	*Xwms*1083	WSB	2.3	4.1*	-	-	-	-	-	-	2	3.2
	3DL	*XpCAT-mTGCG*188	WSB	5.3	9.7*	2.5	5.5*	-	-	2.3	5.2*	3.8	7.3*
	5DL	*Xgwm*97	WSB	-	-	2.9	5.5*	-	-	-	-	-	-
	5AS	*Xbarc*180	WSB	2.6	5.5*	1.8	4.6	-	-	-	-	2	3.5*
	1A	*XpAG-mTCGA*338	WSB	-	-	-	-	-	-	2.6	6.6	-	-
Total R^2					64		45.6				24.8		59.8
DON content													
	3BS	*Xbarc*147	WSB	14.7	30.6*	4.5	9.6*	6.5	18.6*	10.2	25.8*	15.6	35.6*
	3BSc	*Xbarc*376	WSB	1.9	2.9	5.1	12.2*	-	-	-	-	2.1	3.4
	1A	*XpACTG-mTGC*521	WTN	-	-	4.0	10.1*	1.5	4.2	-	-	3.0	6.5*
	5AS	*Xbarc*180	WSB	2.9	5.1*	-	-	-	-	1.9	4.0	-	-
	5DL	*Xgwm*212	WSB	-	-	2.3	7.7*	-	-	4.7	10.0*	2.1	3.6*
	1BL	*Xwms*759	WSB	3.5	7.8*	-	-	-	-	-	-	-	-
	7AL	*Xwms*1083	WSB	-	-	3.3	7.1*	-	-	-	-	-	-
Total R^2					52.8		46.1		20		41.4		43.8

[a] WSB, resistance allele from Wangshuibai; WTN, QTL allele from Wheaton
[b] Mean over experiment
* Significant QTL

Table 4.3 Individual and combined genotypic effects of marker loci linked to QTL for type II FHB resistance on wheat chromosome arms 3BS, 5AS, and 5DL

Locus	Genotype	2003 Spring	2003 Fall	2005	Mean
Xbarc147 (3BS)	A (R)[§]	34.5	26.6	51.7	35.3
	B (S)	66	56.6	71.3	60.4
	dif	31.5	30	19.6	25.1
Xbarc180 (5AS)	A (R)	40.5	33.9	58.9	42.2
	B (S)	53.3	43.9	61.8	49.6
	dif	12.8	10	2.9	7.4
Xgwm292 (5DL)	A (R)	40.5	33.9	58.9	42.2
	B (S)	53.3	43.9	61.8	49.6
	dif	12.8	10	2.9	7.4
Xbarc147/*Xbarc180*	A/A (R/R)	29.9 a[¶]	26.3 a	54.2 a	35.1 a
	A/B (R/S)	37.4 a	26.8 a	51.0 a	36.0 a
	B/A (S/R)	62.9 b	48.8 b	70.2 b	56.3 b
	B/B (S/S)	70.0 b	60.3 b	72.9 b	63.4 b
	dif	40.1	34	21.9	28.3
Xbarc147/*Xgwm292*	A/A (R/R)	28.5 a	24.1 a	49.5 a	32.4 a
	A/B (R/S)	44.5 b	30.8 b	55.5 ab	40.2 ab
	B/A (S/R)	58.8 c	49.1 c	64.4 b	55.2 c
	B/B (S/S)	74.8 d	65.1 d	79.3 c	66.5 d
	dif	46.3	41	29.8	34.1
Xbarc147/*Xbarc180*/*Xgwm292*	A/A/A (R/R/R)	24.4 a	19.2 a	46.6 a	28.9 a
	A/A/B (R/R/S)	41.2 abc	23.2 ab	48.1 a	36.0 ab
	A/B/A (R/S/R)	33.4 ab	35.1 abc	54.4 ab	38.1 abc
	A/B/B (R/S/S)	48.1 bcd	38.4 bc	67.6 abc	46.3 bc
	B/A/A (S/R/R)	44.7 bc	28.1 ab	52.9 ab	41.6 abc
	B/A/B (S/R/S)	72.6 ef	61.5 de	79.3 d	64.3 de
	B/B/A (S/S/R)	66.0 de	57.7 d	71.7 bc	61.0 de
	B/B/B (S/S/S)	84.9 f	78.2 e	81.0 d	73.7 e
	dif	60.5	59	34.4	44.8

[§]A = Wangshuibai, B = Wheaton, and dif = phenotypic difference between genotype means.
A and B are resistant (R) and susceptible (S) for the flanking markers.
[¶]Different letter indicates significant difference at $P < 0.05$

Table 4.4 Individual and combined genotypic effects of marker loci linked to QTL for low DON content resistance on wheat chromosome arms 3BS, 5AS, and 5DL

Locus	Genotype	03 Spring	03 Fall	2004	2005	Mean
Xbarc147 (3BS)	A (R) [§]	5	6.8	20.9	12.3	11.2
	B (S)	25.2	25.7	48.9	36	33.8
	dif	20.2	18.9	28	23.7	22.6
Xbarc180 (5AS)	A (R)	6.9	9.7	19.7	18.4	13.7
	B (S)	19.2	17.2	44.5	25.9	26.6
	dif	12.3	7.5	24.8	7.5	12.9
Xgwm212 (5DL)	A (R)	10.1	10.4	15.7	15.2	12.7
	B (S)	18.1	19.9	52.3	31.5	30.3
	dif	8	9.5	36.6	16.3	17.6
Xbarc147/*Xbarc180*	A/A (R/R)	2.6 a[¶]	6.1 a	16.0 a	13.2 a	9.5 a
	A/B (R/S)	7.0 a	8.4 ab	29.1 ab	12.2 a	14.1 a
	B/A (S/R)	14.8 ab	18.0 ab	30.7 ab	29.3 ab	23.2 ab
	B/B (S/S)	32.5 b	26.7 b	63.8 b	40.3 b	40.8 b
	dif	29.9	20.6	47.8	28.1	26.7
Xbarc147/*Xgwm212*	A/A (R/R)	4.1 a	3.6 a	10.7 a	8.7 a	6.8 a
	A/B (R/S)	6.3 a	11.7 ab	36.9 ab	18.0 a	18.2 a
	B/A (S/R)	18.4 ab	20.8 bc	23.6 ab	24.6 a	21.9 a
	B/B (S/S)	32.8 b	31.1 c	78.6 b	48.8 b	47.3 b
	dif	28.7	27.5	67.9	40.1	40.5
Xbarc147/*Xbarc180*/*Xgwm212*	A/A/A (R/R/R)	1.8 a	4.1 a	12.1 a	8.3 a	6.7 a
	A/A/B (R/R/S)	3.5 a	8.7 a	20.9 a	19.4 ab	13.1 a
	A/B/A (R/S/R)	5.2 a	3.8 a	10.4 a	8.9 a	7.0 a
	A/B/B (R/S/S)	9.9 a	15.4 ab	59.8 ab	17.5 ab	25.6 a
	B/A/A (S/R/R)	16.9 ab	24.4 ab	19.7 a	15.6 ab	19.1 a
	B/A/B (S/R/S)	14.1 ab	15.7 ab	35.2 ab	34.2 b	24.6 a
	B/B/A (S/S/R)	20.2 ab	13.3 a	26.9 a	24.4 ab	21.2 a
	B/B/B (S/S/S)	48.7 b	44.1 b	112.0 b	61.2 c	66.5 b
	dif	46.9	40	101.6	52.9	59.8

[§] A = Wangshuibai, B = Wheaton, and dif = phenotypic difference between genotype means
A and B are resistant (R) and susceptible (S) for the flanking markers.
[¶] Different letter indicates significant difference at $P < 0.05$

REFERENCES

Abate ZA and McKendry AL (2005) Quantitative trait loci associated with reduced deoxynivalenol in the soft red winter wheat 'Ernie'. p. 3–5 *In*: Canty SM, Lewis J, Siler L, and Ward RW (eds), Proceedings of the 2005 National Fusarium Head Blight Forum; 2005 Dec 11-13; Milwaukee, Wisconsin. East Lansing: Michigan State University, MI.

Abate ZA, Tague DN, and McKendry AL (2006) Diallel analysis of Fusarium head blight resistance in genetically diverse winter wheat germplasm. p. 73–77 *In*: Canty SM, Lewis J, Siler L, and Ward RW (eds), Proceedings of the 2005 National Fusarium Head Blight Forum; 2006 Dec 10-12; Research Triangle Park, NC. East Lansing: Michigan State University, MI.

Anderson AL (1948) The development of *Gibberella zeae* head blight of wheat. Phytopathology 38: 595–611

Anderson JA, Churchill GA, Autrique JE, Tanksley SD, Sorrells ME (1993) Optimizing parental selection for genetic linkage maps. Genome 36:181–186

Anderson JA, Waldron BL, Moreno-Sevilla B, Stack RW, and Frohberg RC (1998) DNA markers for Fusarium head blight resistance QTL in two wheat populations. p. 57–59. *In*: Hart P *et al*. (eds) 1998 National FHB Forum Proceedings. University Printing, East Lansing, MI.

Anderson JA, Stack RW, Liu S, Waldron BL, Fjeld AD, Coyne C, Moreno-Sevilla B, Fetch JM, Song QJ, Cregan PB, Frohberg RC (2001) DNA markers for *Fusarium* head blight resistance QTL in two wheat populations. Theor Appl Genet 102:1164–1168

Angerer A, Lengauer D, Steiner B, Lafferty J, Loeschenberger F, *et al*. (2003) Validation of molecular markers linked to two Fusarium head blight resistance QTLs in wheat. p. 1096–1098. Proc Int Wheat Genet Sym. Paestum, 10th, Rome: 1st. Sper. Cerealicolt. Press

Aoki T, O'Donnell K (1999a) Morphological and molecular characterization of *Fusarium pseudograminearum* sp. nov., formerly recognized as the Group 1 population of *F. graminearum*. Mycologia 91:597–609

Aoki T, O'Donnell K (1999b) Morphological characterization of *Gibberella coronicola* sp. nov. obtained through mating experiments of *Fusarium pseudograminearum*. Mycoscience 40:443–53

Bai GH, Xiao QP, Mei JF (1989) Genetic analysis of resistance to scab in six wheat cultivars. Acta Agric. Shanghai 5:17–23

Bai GH, Zhou CF, Qian CM, Ge YF (1990) Genetic analysis of genes for resistance to scab spread in wheat cultivars. p. 171–177. *In*: Lu HZ (ed), Advances in Genetic Researches on Resistance to Diseases in Major Crops. Nanjing, China: Jiangsu Sci. Tech. Publ. House

Bai GH, Shaner GE (1994) Wheat scab: perspective and control. Plant Dis. 78:760–766

Bai GH, Shaner GE (1996) Variation in *Fusarium graminearum* and cultivar resistance to wheat scab. Plant Dis 80:975–979

Bai GH, Kolb FL, Shaner GE, Domier LL (1999) Amplified fragment length polymorphism markers linked to a major quantitative trait locus controlling scab resistance in wheat. Phytopathology 89:343–348

Bai GH, Shaner GE, Ohm HW (2000a) Inheritance of resistance to *Fusarium graminearum* in wheat. Theor Appl Genet 100:1–8

Bai GH, Plattner R, Shaner GE, Kolb FL (2000b) A QTL for deoxynivalenol tolerance in wheat. Phytopathology 90(6):S4 (Abstr.)

Bai GH, Plattner RD, Desjardins AE, Kolb FL (2001) Resistance to Fusarium head blight and deoxynivalenol accumulation in wheat. Plant Breeding 120:1–6

Bai GH, Desjardins AE, Plattner RD (2002) Deoxynivalenol-nonproducing *Fusarium graminearum* causes initial infection, but does not cause disease spread in wheat spikes. Mycopathologia 153:91–98

Bai GH, Chen LF, Shaner GE (2003) Breeding for resistance to Fusarium head blight of wheat in China. p. 296–317. *In*: Leonard KJ and Bushnell WR (eds) Fusarium Head Blight of Wheat and Barley. St Paul, MN: APS Press

Bai GH, Guo PG, Kolb FL (2003) Genetic relationships among head blight resistant cultivars of wheat assessed on the basis of molecular markers. Crop Sci 43:498–507

Bai GH, Shaner GE (2004) Management and resistance in wheat and barley to Fusarium head blight. Ann Rev Phytopath 42:135–161

Baker RJ (1977) Inheritance of kernel hardness in spring wheat. Crop Sci 17:960–962

Ban T (1997) Evaluation of resistance to Fusarium head blight in indigenous Japanese species of *Agropyron* (*Elymus*). Euphytica 97:39–44

Ban T (2000) Review-study on the genetics of resistance to Fusarium head blight caused by

Fusarium graminearum in wheat. p. 82–93. *In*: Raupp WJ, Ma Z, Chen PD, and Liu DJ (eds), Proceeding of International Symposium on Wheat Improvement for Scab Resistance. May 5–10, 2000 Suzhou and Nanjing, China

Ban T, Suenaga K (2000b) Genetic analysis of resistance to Fusarium head blight caused by *Fusarium graminearum* in Chinese wheat cultivar Sumai 3 and the Japanese cultivar Saikai 165. Euphytica 113:87–99

Ban T (2001) Studies on the genetics of resistance to Fusarium head blight caused by *F. graminearum* Schwabe in wheat (*Triticum aestivum* L.). Bull. Kyushu Natl Agric Exp Stn 38:27–78

Ban T and Watanabe N (2001) The effects of chromosomes 3A and 3B on resistance to Fusarium head blight in tetraploid wheat. Hereditas 135:95-99

Ban T (2003) Comparative genetic analysis of FHB-resistant germplasm for wheat improvement. p. 215–217. *In*: Canty SM *et al*. (eds) Proceedings of the 2003 National Fusarium Head Blight Forum. Michigan State University, East Lansing, MI.

Barrett BA and Kidwell KK (1998a) AFLP-based genetic diversity assessment among wheat cultivars from the Pacific Northwest. Crop Sci 38:1261–1271

Barrett BA, Kidwell KK, Fox PN (1998b) Comparison of AFLP and pedigree-based genetic diversity assessment methods using wheat cultivars from the Pacific Northwest. Crop Sci 38:1271–1278

Blake NK, Lehfeldt BR, Lavin M, and Talbert LE (1999) Phylogenetic reconstruction based on low copy DNA sequence data in an allopolyploid: the B genome of wheat. Genome 42:351–360

Botstein D, White RL, Skolnick M, and Davis RW (1980) Construction of a genetic linkage map in man using restriction fragment length polymorphisms. Am J Hum Genet 32:314–331.

Bourdoncle W, Ohm HW (2003a) Quantitative trait loci for resistance to Fusarium head blight in recombinant inbred wheat lines from the cross Huapei 57-2/Patterson. Euphytica 131:131–136

Bourdoncle W and Ohm HW (2003b) Fusarium head blight-resistant wheat line 'Bizel' does not contain rye chromatin. Plant Breeding 122:281–282

Briggle LW, Curtis BC (1987) Wheat Worldwide. p. 1–32. *In*: Heyne EG (ed) Wheat and Wheat Improvement. Madison, WI

Brookes AJ (1999) The essence of SNPs. Gene 2341:177–186

Broman KW, Wu H, Churchill GA (2003). R/qtl: QTL mapping in experimental crosses.

Bioinformatics 19:889–890

Brown-Guedira GL, Bockus WW, Davis MA, Gill BS, Van Sanford DA, and Murphy JP (2005) Notice of release of KS04WGRC46 Fusarium head blight-resistant hard red winter wheat germ plasm. Ann Wheat Newslett 51:189

Browne RA, Murphy JP, Cooke BM *et al*. (2005) Evaluation of components of Fusarium head blight resistance in soft red winter wheat germ plasm using a detached leaf assay. Plant Dis 89:404–411

Buerstmayr H, Lemmens M, Grausgruber H, and Ruckenbauer P (1996) Scab resistance of international wheat germplasm. Cereal Res Comm 24:195-202

Buerstmayr H, Lemmens M, Ruckenbauer P (1997) Chromosomal location of Fusarium head blight resistance genes in wheat. Cereal Res Commun 25:727–728

Buerstmayr H, Lemmens M, Berlakovich S, and Ruckenbauer P (1999) Combining ability of resistance to head blight caused by *Fusarium culmorum* (W.G. Smith) in the F_1 of a seven parent diallel of winter wheat (*Triticum aestivum* L.). Euphytica 110:199–206

Buerstmayr H, Steiner B, Lemmens M, and Ruckenbauer P (2000) Resistance to Fusarium head blight in winter wheat: heritability and trait associations Crop Sci 40:1012–1018

Buerstmayr H, Lemmens M, Hartl L, Doldi L, Steiner B, Stierschneider M, Ruckenbauer P (2002) Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. resistance to fungal spread (type II resistance). Theor Appl Genet 104:84–91

Buerstmayr H, Steiner B, Hartl L, Griesser M, Angerer N, Lengauer D, Miedaner T, Schneider B, Lemmens M (2003a) Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor Appl Genet 107:503–508

Buerstmayr H, Stierschneider M, Steiner B, Lemmens M, Griesser M, Nevo E, and Fahima T (2003b) Variation for resistance to head blight caused by *Fusarium graminearum* in wild emmer (*Triticum dicoccoides*) originating from Israel. Euphytica 130:17-23

Buntjer, J.B. (2001). PhylTools: Phylogenetic Computer Tools V.1.32. Wageningen University and Research Centre, The Netherlands. http://www.dpw.wau.nl/pv/PUB/pt/

Burgess LW, Dodman RL, Pont W, Mayers P (1981) Fusarium disease of wheat, maize and grain sorghum in eastern Australia. p.64-76. *In*: Nelson PE, Toussoun TA, and Cook RJ (eds) *Fusarium*: Disease, Biology, and Taxonomy. University Park, PA: Penn. State Univ. Press

Bushnell WR, Hazen BE, Pritsch C (2003) Histology and physiology of Fusarium head blight. p. 44–83. *In*: Leonard KJ and Bushnell WR (eds) Fusarium Head Blight of Wheat and

Barley. St Paul, MN: APS Press

Cai X, Chen PD, Xu SS, Oliver RE, and Chen X (2005) Utilization of alien genes to enhance Fusarium head blight resistance in wheat — A review. Euphytica 142:309–318

Chao S, Sharp PJ, Worland AJ, Warham EJ, Koebner RMD, and Gale MD (1989) RFLP-based genetic maps of wheat homoeologous group 7 chromosomes. Theor Appl Genet 78:495–504

Chappell M, Griffey C, Pridgen T, Chen JL, and Shaw J (1999) Assessment and reaction of soft red winter wheat genotypes to *Fusarium graminearum* and effects on traits related to yield and seed quality. p. 143–146. *In*: Wagester JA *et al*. (eds) Proceedings of the 1999 National Fusarium Head Blight Forum. Michigan State University, East Lansing, MI.

Chen J, Griffey CA, Saghai Maroof MA, Zhao W, Xie W, and Pridgen T (2000) Genetic analysis of resistance to Fusarium head blight in common wheat. p. 19–24. *In*: Ward RW *et al*. (eds) 2000 National Fusarium Head Blight Forum Proceedings, Erlanger, KY. 10–12 Dec. 2000. Michigan State University, East Lansing, MI.

Chen J, Griffey CA, Saghai Maroof MA, Stromberg EL, Biyashev RM, Zhao W, Chappell MR, Pridgen TH, Dong Y, Zeng Z (2006) Validation of two major quantitative trait loci for Fusarium head blight resistance in Chinese wheat line W14. Plant Breeding 125:99–101

Chen PD, Wang ZT, Wang SL, Huang L, Wang YZ, and Liu DJ (1993) Transfer of wheat scab resistance from *Elymus giganteus* into common wheat. p. 153–157. *In*: Li ZS and Xin ZY (eds), Proc 8th Int Wheat Genet Symp, China Agri Sci Press, Beijing, China.

Chen PD and Liu DJ (2000) Transfer of scab resistance from *Leymus racemosus*, *Roegneria ciliaris*, and *Roegneria kamoji* into common wheat. p. 62–67. *In*: Raupp WJ, Ma Z, Chen PD, and Liu DJ (eds), Proc Int Symp Wheat Improv Scab Resist, Suzhou and Nanjing, China.

Chen P, Liu W, Yuan J, Wang X, Zhou B, Wang S, Zhang S, Feng Y, Yang B, Liu G, Liu D, Qi L, Zhang P, Friebe B, and Gill BS (2005). Development and characterization of wheat-*Leymus racemosus* translocation lines with resistance to Fusarium head blight. Theor Appl Genet 111:941-948

Churchill GA and Doerge RW (1994) Empirical threshold values for quantitative trait mapping. Genetics 138:963–971

CIMMYT (1996) CIMMYT 1995–96 world wheat facts and trends: understanding global trends in the use of wheat diversity and international flows of wheat genetic resources. Mexico, DF. Mexico

de la Peña RC, Smith KP, Capettini F, Muehlbauer GJ, Gallo-Meagher M, *et al*. (1999) Quantitative trait loci associated with resistance to Fusarium head blight and kernel

discoloration in barley. Theor Appl Genet 99:561–69

Desjardins AE, Proctor R H, Bai GH, McCormick SP, Shaner GE, Buechley G, Hohn T M (1996) Reduced virulence of trichothecene antibiotic-nonproducing mutants of *Gibberella zeae* in wheat field tests. Mol Plant-Microbe Interact 9:775–781

Devos KM, Millan T, and Gale MD (1993) Comparative RFLP maps of the homoeologous group-2 chromosomes of wheat, rye and barley. Theor Appl Genet 85:784–792

Devos KM, Dubcovsky J, Dvořák J, Chinoy CN, and Gale MD (1995) Structural evolution of wheat chromosomes 4A, 5A, and 7B and its impact on recombination. Theor Appl Genet 91:282–288

Devos KM, Gale MD (1997) Comparative genetics in the grasses. Plant Mol Biol 35: 3-15

Dickson, JG, Mains EB (1929) Scab of wheat and barley and its control. USDA Farmers' Bulletin 1599. p. 1–18

Dvorak J, McGuire PE and Cassidy B (1988) Apparent sources of the A genomes of wheat inferred from polymorphism in abundance and restriction fragment length of repeated nucleotide sequences. Genome 30:680–689.

Dvorak J and Zhang HB (1990) Variation in repeated nucleotide sequences sheds light on the phylogeny of the wheat B and G genomes. Proc Natl Acad Sci (USA) 87:9640–9644.

Dvorak J, di Terlizzi P, Zhang HB and Resta P (1993) The evolution of polyploid wheats: identification of the A genome donor species. Genome 36:21–31.

Dubin H J, Gilchrist L, Reeves J, McNab A (eds) (1997) Fusarium head scab: Global status and prospects. CIMMYT, Mexico, DF, Mexico. 130 p

Dubcovsky J, Luo MC, Zhong GY, Bransteitter R, Desai A, Kilian A, Kleinhofs A, and Dvorak J (1996) Genetic map of diploid wheat, *Triticum monococcum* L., and its comparison with maps of *Hordeum vulgare* L. Genetics 143:983–999

Dubcovsky J, Lijavetzky D, Appendino L, Tranquilli G (1998) Comparative RFLP mapping of *Triticum monococcum* genes controlling vernalization requirement. Theor Appl Genet 97:968–975

Dubcovsky J, Ramakrishna W, SanMiguel PJ, Busso CS, Yan L, Shiloff BA, and Bennetzen JL (2001) Comparative sequence analysis of colinear barley and rice bacterial artificial chromosomes. Plant Physiol 125:1342–53

Endo TR and Gill BS (1996) The deletion stocks of common wheat. J Hered 87:295–307.

Faris JD, Friebe B, and Gill BS (2002) Wheat genomics: exploring the polyploidy model.

Current Genomics 3:577–591

Faris JD, Simons KJ, Zhang ZC, Gill BS (2005) The wheat super domestication gene Q. Frontiers of Wheat Bioscience 100:129–148

Fedak G (2000) Sources of resistance to Fusarium head blight. p. 4. *In*: Raupp WJ, Ma Z, Chen PD, and Liu DJ (eds), Proceeding of International Symposium on Wheat Improvement for Scab Resistance. May 5–10, 2000 Suzhou and Nanjing, China

Fedak G, Cao W, Han F, Savard M, Gilbert J, and Xue A (2004) Germplasm enhancement for FHB resistance in spring wheat through alien introgression. p. 56–57. *In*: Canty SM, Boring T, Versdahl K, Wardwell J, and Ward RW (eds), Proc. 2nd Int Symp on Fusarium Head Blight, Orlando, Michigan State University, East Lansing, MI.

Felsenstein, J (2005) PHYLIP (Phylogeny Inference Package) V 3.6. Department of Genome Sciences, University of Washington, Seattle, U.S.A. http://evolution.gs.washington.edu/phylip.html

Flood RG, Halloran GM (1986) Genetics and physiology of vernalisation response in wheat. Adv Agron 39:87–125

Frary A, Nesbitt TC, Frary A, Grandillo S, van der Knaap E, Cong B, Liu J, Meller J, Elber R, Alpert K, and Tanksley S (2000). fw2.2: a quantitative trait locus key to the evolution of tomato fruit size. Science 289:85–87

Friebe B and Gill BS (1996) Chromosome banding and genome analysis in diploid and cultivated polyploid wheats. p. 36–60. *In*: Jauhar PP (ed) Methods of genome analysis in plants. Boca Raton, FL, USA, CRC Press.

Gagkaeva TY (2003) Importance of Fusarium head blight in Russia and the search for new sources of genetic resistance in wheat and barley. p. 219–222. *In*: Canty SM, Lewis J, and Ward RW (eds), Proceedings of the 2003 National Fusarium Head Blight Forum. Michigan State University, East Lansing, MI.

Gale MD, Atkinson MD, Chinoy CN, Harcourt RL, Jai J, Li QY, Devos KM (1995) Genetic maps of hexaploid wheat. p. 29–40. *In*: Chen S (ed), Proc. 8th International Wheat Genetics Symposium. China Agricultural Scientech Press, Beijing

Gervais L, Dedryver F, Morlais JY, Bodusseau V, Negre S, Bilous M, Groos C, Trottet M (2003) Mapping of quantitative trait loci for field resistance to *Fusarium* head blight in an European winter wheat. Theor Appl Genet 106:961–970

Gilbert J, Procunier JD, Aung T (2000) Influence of the D genome in conferring resistance to Fusarium head blight in spring wheat. Euphytica 114:181–186

Gilchrist L, Rajaram S, Mujeeb-Kazi A, van Ginkel M, Vivar H, and Pfeiffer W (1997) *In*:

Dubin HJ, Gilchrist L, Reeves J, and McNab A (eds), Fusarium head scab: global status and future prospects. Mexico, DF, CIMMYT.

Gill KS, Gill BS, and Endo TR (1993) A chromosome region-specific mapping strategy reveals gene-rice telomeric ends in wheat. Chromosoma 102:374–381

Gilsinger J, Kong L, Shen X, Ohm HW (2005) DNA markers associated with low Fusarium head blight incidence and narrow flower opening in wheat. Theor Appl Genet 110:1218–1225

Giroux MJ and Morris CF (1998) Wheat grain hardness results from highly conserved mutations in the friabilin components puroindoline a and b. PNAS USA 95:6262–6266

Gooding RW, Lafever HN, Campbell KG, and Herald LD (1997) Registration of 'Freedom' wheat. Crop Sci. 37:1007

Grausgruber H, Lemmens M, Bürstmayr H, Ruckenbauer P (1998) Chromosomal location of Fusarium head blight resistance and in vitro toxin tolerance in wheat using the 'Hobbit sib' (*Triticum macha*) chromosome substitution lines. J Genet Breed 53:173–180

Griffey CA, Chen JL, Pridgen T, Chappell M, and Stromberg EL (1998) Assessment and selection for scab resistance in soft red winter wheat. p. 73–75. *In*: Hart P *et al*. (eds) 1998 National Fusarium Head Blight Forum Proceedings. Michigan State University, East Lansing, MI.

Griffey CA, Chen JL, Pridgen T, Chappell M, and Stromberg EL (1999) Advances in research on Fusarium head blight in the virginia wheat breeding program. p. 165–172. *In*: Wagester JA *et al*. (eds), 1999 National Fusarium Head Blight Forum Proceedings. Michigan State University, East Lansing, MI.

Griffey CA (2005) Identification and incorporation of FHB resistance in winter wheat: an overview of progress and strategies. p. 31-34. *In*: Canty SM, Lewis J, Siler L, and Ward RW (eds), Proceedings of the 2005 National Fusarium Head Blight Forum; 2005 Dec 11–13; Milwaukee, Wisconsin. East Lansing: Michigan State University, MI.

Grodzicker T, Williams J, Sharp P, and Sambrook J (1974) Physical mapping of temperature-sensitive mutations of adenovirus. Cold Spring Harbor Symp. Quant Biol 39:439–446

Guo PG, Bai GH, Li RH, Shaner G, and Baum M (2006) Resistance gene analogs associated with Fusarium head blight resistance in wheat. Euphytica 151:251–261

Gupta M, Balyan HS, Sharma PC, Ramesh B (1998) Genetics and molecular biology of seed storage proteins in wheat. p. 126–157. *In*: Gupta PK (eds) Genetics and Biotechnology in Crop Improvement. Rastogi Publications, Meerut

Gupta PK, Varshney RK, Sharma PC, Ramesh B (1999) Molecular markers and their

applications in wheat breeding. Plant Breed 118:369–390

Gupta A, Lipps PE, and Campbell KG (2000) Finding quantitative trait locus associated with Fusarium head blight of wheat using simple sequence repeat markers. p. 28–32 *In*: Ward RW *et al*. (eds) 2000 National Fusarium Head Blight Forum Proceedings, Erlanger, KY. 10–12 Dec. 2000 Michigan State University, East Lansing, MI.

Gupta PK, Balyan H, Edwards K, Isaac P, Korzun V, Röder MS, Gautier MF, Joudrier P, Schlatter A, Dubcovsky J, de la Peña R, Khairallah M, Penner G, Hayden M, Sharp P, Keller B, Wang R, Hardouin J, Jack P, Leroy P (2002) Genetic mapping of 66 new microsatellite (SSR) loci in bread wheat. Theor Appl Genet 105: 413–422

Guyomarc'h, H, Sourdille P, Charmet G, Edwards K, Bernard M 2002 Characterization of polymorphic microsatellite markers from *Aegilops tauschii* and transferability to the D-genome of bread wheat. Theor Appl Genet 104:1164–1172

Harris LJ, Desjardins AE, Plattner RD, Nicholson P, Butler G, Young JC, Weston G, Proctor R H, Hohn TM (1999) Possible role of trichothecene mycotoxins in virulence of *Fusarium graminearum* on maize. Plant Dis 83:954–960

Heun M, Schafer-Pregl R, Klawan D, Castagna R, Accerbi M, Borghi B and Salamini F (1997) Site of Einkorn wheat domestication identified by DNA fingerprinting. Science 278: 1312–1314.

Hill-Ambroz K, Webb CA, Matthews AR, Li WL, Gill BS, and Fellers JP (2006) Expression analysis and physical mapping of a cDNA library of Fusarium head blight infected wheat spikes. Crop Sci 46:15–26

Holland JB (2004) Implementation of molecular markers for quantitative traits in breeding programs - challenges and opportunities. *In*: Fischer T *et al*. (eds) New directions for a diverse planet: Proceedings for the 4th International Crop Science Congress, Brisbane, Australia, 26 September -1 October 2004

Huang S, Sirikhachornkit A, Faris JD, Su X, Gill BS *et al*. (2002) Phylogenetic analysis of the acetyl-CoA carboxylase and 3-phosphoglycerate kinase loci in wheat and other grasses. Plant Mol Biol 48:805–820

Ittu M, Saulescu NN, and Ittu G (1994) Criteria for estimation for Fusarium scab resistance in wheat. Roman Agric Res 1:19–21

Ittu M, Saulescu NN, and Ittu G (1997) Resistance to Fusarium head blight (scab) in recombinant inbred lines derived from a *Triticum aestivum* cross. Cereal Res Commun 25:757–760

Jauhar PP and Peterson TS (1998) Wild relatives of wheat as sources of Fusarium head blight resistance. p. 3-6. *In*: Hart P *et al*. (eds), 1998 National Fusarium Head Blight Forum

Proceedings. Michigan State University, East Lansing, MI.

Jansen C, von Wettstein D, Schäfer W, Kogel K-H, Felk A, Maier FJ (2005) Infection patterns in barley and wheat spikes inoculated with wild-type and trichodiene synthase gene disrupted *Fusarium graminearum*. Proc Natl Acad Sci (USA) 102:16892–16897

Jansen RC and Stam P (1994) High resolution of quantitative traits into multiple loci via interval mapping. *Genetics* 136:1447–1455

Jia GF, Chen PD, Qin GJ, Bai GH, Wang X, Wang SL, Zhou B, Zhang SH, and Liu DJ (2005) QTLs for Fusarium head blight response in a wheat DH population of Wangshuibai /Alondra's'. Euphytica 146:183–191

Jiang G, Wu Z, Huang D (1994) Effects of recurrent selection for resistance to scab (*Gibberella zeae*) in wheat. Euphytica 72:107–113

Jiang GL, Chen ZX, and Wu ZS (1997) Studies on the development of scab resistance gene pool in wheat. V. Analysis and comparison of recurrently selected lines and their parents. Acta Agronomica Sin 23:34–38

Jiang GL, Shi JR, and Ward R (2005) QTL mapping of Fusarium head blight resistance in novel wheat germplasm CJ 9306. p. 43. *In*: Canty SM *et al*. (eds), Proceedings of the 2005 National Fusarium Head Blight Forum. Michigan State University, East Lansing, MI.

Jiang GL and Ward RW (2006) Inheritance of resistance to Fusarium head blight in the wheat lines 'CJ 9306' and 'CJ 9403'. Plant Breeding 125:417–423

Johnson BL and Dhaliwal HS (1976) Reproductive isolation of *Triticum boeoticum* and *Triticum urartu* and the origin of the tetraploid wheat. Am J Bot 63:1088–1094.

Johnson DD, Flaskerud GK, Taylor RD, Satyanarayana V (2003) Quantifying economic impacts of Fusarium head blight in wheat. p. 461–483. *In*: Leonard KJ and Bushnell WR (eds) *Fusarium* Head Blight of Wheat and Barley. St Paul, MN: APS Press

Johnson VA, Briggle LW, Axtel JD, Bauman LF, Leng ER, Johnston TH (1978) Grain crops. p. 239–255. *In*: Milner M, Scrimshaw NS, and Wang DIC (eds), Protein Resources and Technology. Westport, CT, USA, AVI Publishing

Johnson AR and Wichern DW (1992) Applied multivariate statistical analysis. 3rd edition, Prentice-Hall, Englewood Cliffs, NJ.

Jolliffe IT (1986) Principal Component Analysis. Springer-Verlag, Berlin.

Kam-Morgan LNW, Gill BS, and Muthukrishnan S (1989) DNA restriction fragment length polymorphisms: a strategy for genetic mapping of D genome of wheat. Genome 32:724–732

Kang Z, Buchenauer H (1999) Immunocytochemical localization of fusarium toxins in infected wheat spikes by *Fusarium culmorum*. Physiol Mol Plant Pathol 55:275–288

Keby K and Kuspira J (1988) Cytological evidence bearing on the origin of the B genome in polyploidy wheats. Genome 30:36–43

Kihara H (1919) Ueber cytologische Studien bei einigen Getreidearten. 1. Spezies-Bastarde des Weizens und Weizenroggen-Bastard. Bot Mag 32:17–38.

Kihara H (1944) Discovery of the DD-analyser, one of the ancestors of *Triticum vulgare* (Japanese). Agric Hort 19:13–14

Klahr A, Zimmermann G, Gerhard Wenzel G, and Mohler V (2006) Effects of environment, disease progress, plant height and heading date on the detection of QTLs for resistance to Fusarium head blight in an European winter wheat cross. Euphytica DOI 10.1007/s10681-006-9264-7

Knapp SJ, Stroup WW, Ross WM (1985) Exact confidence intervals for heritability on a progeny mean basis. Crop Sci 25:192–194

Kolb FL, Bai GH, Muehlbauer GJ, Anderson JA, Smith KP, Fedak G (2001) Host plant resistance genes for Fusarium head blight: Mapping and manipulation with molecular markers. Crop Sci 41: 611–619

Kosambi DD (1944) The estimation of map distances from recombination values. Ann Eugen 12:172–175

Kuspira J and Unrau J (1957) genetic analysis of certain character in common wheat using whole chromosome substitution lines. Can J Plant Sci 37:300–326

Lamb KE, Green MJ, Horsley RD, and Zhang BX (2001) Mapping genes conferring Fusarium head blight resistance in a Midwest barley accession Hietpas 5. p. 19–20. *In* Canty SM *et al*. (eds), 2001 National Fusarium Head Blight Forum Proceedings. Kinko's, Okemos, MI.

Lander ES and Botstein D (1989) Mapping Mendelian factors underlying quantitative traits using RFLP linkage maps. Genetics 121:185–199.

Lagercrantz U, Ellegren H, Anderson L (1993) The abundance of various polymorphic microsatellite motifs differs between plants and vertebrates. Nuc Acids Res 21:1111–1115

Law CN (1966) The location of genetic factors affecting a quantitative character in wheat. Genetics 53:487-498

Lemmens M, Reisinger A, Buerstmayr H, Ruckenbauer P (1994) Breeding for head blight (*Fusarium* spp.) resistance in wheat: Development of a mycotoxin-based selection method of seedlings. Acta Hortic 355:223–232

Lemmens M, Scholz U, Berthiller F, Dall'Asta C, Koutnik A, Schuhmacher R, Adam G, Buerstmayr H, Mesterházy A, Krska R, Ruckenbauer P (2005) The ability to detoxify the mycotoxin deoxynivalenol colocalizes with a major quantitative trait locus for Fusarium head blight resistance in wheat. Molecular Plant-Microbe Interactions 18:1318–1324

Leonard KJ, Bushnell WR (eds) (2003) *Fusarium* Head Blight of Wheat and Barley. St Paul, MN: APS Press. 512 p

Lewis JM, Suenaga K, Van Ginkel M *et al*. (2004) Identification and mapping of a QTL for type II resistance to Fusarium head blight on chromosome arm 2DL of wheat. p.89–92. *In*: Canty SM, Boring T, Versdahl K, Wardwell J, and Ward RW (eds), Proc. 2nd Int Symp on Fusarium Head Blight, Orlando, Michigan State University, East Lansing, MI.

Lijavetzky D, Muzzi G, Wicker T, Keller B, and Wing R *et al*. (1999) Construction and characterization of a bacterial artificial chromosome (BAC) library for the A genome of wheat. Genome 42:1176–1182

Lima MIPM, de Sousa CAN *et al*. (2004) Resistance reaction of brazilian wheat cultivars to scab. p. 93. *In*: Canty SM, Boring T, Versdahl K, Wardwell J, and Ward RW (eds), Proc. 2nd Int Symp on Fusarium Head Blight, Orlando, Michigan State University, East Lansing, MI.

Lin F, Kong ZX, Zhu HL, Xue SL, Wu JZ, Tian DG, Wei JB, Zhang CQ, Ma ZQ (2004) Mapping QTL associated with resistance to Fusarium head blight in the Nanda2419 × Wangshuibai population II: Type II resistance. Theor Appl Genet 109:1504–1511

Lin F, Xue SL, Zhang ZZ, Zhang CQ, Kong ZX, Yao GQ, Tian DG, Zhu HL, Li CJ, Cao Y, Wei JB, Luo QY, Ma ZQ (2006) Mapping QTL associated with resistance to Fusarium head blight in the Nanda 2419 × Wangshuibai population II: Type I resistance. Theor Appl Genet 112:528–535

Liu, Z.Z., 1984. Recent advances in research on wheat scab in China. p. 174–181. *In*: Wheats for more tropical environments. CIMMYT, Mexico, D.F., Mexico.

Liu S, Anderson JA (2003) Marker-assisted evaluation of Fusarium head blight-resistant wheat germplasm. Crop Sci 43:760–766

Liu SX, Zhang XL, Pumphrey MO, Stack RW, Gill BS, Anderson JA (2006) Complex microcolinearity among wheat, rice, and barley revealed by fine mapping of the genomic region harboring a major QTL for resistance to Fusarium head blight in wheat. Funct Integr Genomics 6:83–89

Liu ZZ, Wang ZY (1990) Improved scab resistance in China: sources of resistance and problems. p. 178–187. *In*: Saunders DA (ed) Wheat for the nontraditional warm areas. A proceeding of the international conferences. Foz do Iguacu, Brazil. International Mazie and Wheat Improvement Center (CIMMYT), Mexico

Lu WZ, Chen SH, Wang YZ (eds) (2001) Research on Wheat Scab. Beijing, China: Sci. Publ. House

Luo Y, Yoshizawa T, Katayama T (1990) Comparative study on the natural occurrence of Fusarium mycotoxins (trichothecenes and zearalenone) in corn and wheat from high- and low-risk areas for human esophageal cancer in China. Appl Environ Microbiol 56:3723–3726

Ma HX, Zhang KM, Gao L, Bai GH, Chen HG, Cai ZX, Lu WZ (2006a) Quantitative trait loci for resistance to Fusarium head blight and deoxynivalenol accumulation in Wangshuibai wheat under field conditions. Plant Pathology 55:739–745

Ma HX, Bai GH, Zhang X, and Lu WZ (2006b) Main effects, epistasis, and environmental interactions of quantitative trait loci for Fusarium head blight resistance in a recombinant inbred population. Phytopathology 96:534–541

Mujeeb-Kazi A, Bernard M, Bekele GT, and Mirand JL (1983) Incorporation of alien genetic information from *Elymus giganteus* into *Triticum aestivum*. p. 223–231. *In*: Sakamoto S (ed), Proc 6th Int Wheat Genet Symp Maruzen, Kyoto, Japan.

Mardi M, Pazouki L, Delavar H, Kazemi MB, Ghareyazie B, Steiner B, Nolz R, Lemmens M, and Buerstmayr H (2006) QTL analysis of resistance to Fusarium head blight in wheat using a 'Frontana'-derived population. Plant Breeding 125:313-317

Manly KF, Cudmore RH Jr, Meer JM (2001) Map Manager QTX, cross-platform software for genetic mapping. Mamm Genome 12:930–932

Marasas WFO, Nelson PE, Toussoun TA (1984) Toxigenic *Fusarium* Species: Identifcation and Mycotoxicology. p. 155 –211. Pennsylvania State Univ. Press, University Park.

Mardi M, Buerstmayr H, Ghareyazie B, Lemmens M, Mohammadi SA, Nolz R, and Ruckenbauer P (2005) QTL analysis of resistance to Fusarium head blight in wheat using a 'Wangshuibai'-derived population. Plant Breeding 124:329 – 333

Marino CL, Nelson JC, Lu YH, Sorrells ME, Leroy P, Tuleen NA, Lopes CR, and Hart GE (1996) Molecular genetic maps of the group 6 chromosomes of hexaploid wheat (*Triticum aestivum* L. em. Thell). Genome 39:359–366

Mayer M (2005). A comparison of regression interval mapping and multiple interval mapping for linked QTL. Heredity 94:599-605

McKendry, A.L., J.E. Berg, D.N. Tague, and K.D. Kephart. 1995. Registration of 'Ernie' soft red winter wheat. Crop Sci 35:1513

McKendry AL (2000a) Broadening the Genetic Base for Scab Resistance through a CIMMYT /National Scab Initiative Partnership. p. 209–214. *In*: Ward RW *et al*. (eds), 2000 National Fusarium Head Blight Forum. Kinko's, Okemos, MI.

McKendry AL, Murphy JP, Bestgen K, and Navarro R (2000b) Evaluation of Yugoslavian winter wheat germplasm for resistance to Fusarium head blight. p. 215–219. *In*: Ward RW *et al*. (eds), 2000 National Fusarium Head Blight Forum. Kinko's, Okemos, MI.

McKendry AL, Bestgen KS, and Tague DN (2002) Types I, II and field resistance to Fusarium head blight in winter and spring wheat germplasm. p. 204–207. *In*: Canty SM *et al*. (eds), 2002 National Fusarium Head Blight Forum Proceedings. Michigan State University, East Lansing, MI.

McKendry AL, Shaw LJ, and Tremain JA (2004) Evaluation of Fusarium resistant germplasm introduced through the USWBSI/CIMMYT collaboration. p. 103–106. *In*: Canty SM *et al*. (eds), Proc. 2nd Int Symp on Fusarium Head Blight, Orlando, Michigan State University, East Lansing, MI.

McCartney CA, Somers DJ, Fedak G, Cao W (2004) Haplotype diversity at *Fusarium* head blight resistance QTLs in wheat. Theor Appl Genet 109:261–271

McFadden ES and Sears ER (1946) The origin of Triticum spelta and its free-threshing hexaploid relatives. J Hered 37:81–89, 107–116

McMullen M, Jones R, Gallenberg D (1997) Scab of wheat and barley: a re-emerging disease of devastating impact. Plant Dis 81:1340–1348

Mentewab A, Rezanoor HN, Gosman N, Worland AJ and Nicholson P (2000) Chromosomoal location of Fusarium head blight resistance genes and analysis of the relationship between resistance to head blight and brown foot rot. Plant Breed 119:15–20.

Mesfina A, Smith KP, Dill-Macky R, Evansb CK, Waugh R, Gustusa CD, and Muehlbauer GJ (2003) Quantitative trait loci for Fusarium head blight resistance in barley detected in a two-Rowed by six-Rowed population. Crop Sci 43:307–318

Mesterházy Á (1981) The role of aggressiveness of *Fusarium graminearum* isolates in the inoculation tests on wheat in seedling state. Acta Phytopathol Acad Sci Hung 16:281–292

Mesterházy Á (1995) Types and components of resistance to Fusarium head blight of wheat. Plant Breed 114:377–386

Mesterházy Á, Bartók T, Mirocha CG, Komoróczky R (1999) Nature of resistance to Fusarium

head blight and the role of deoxynivalenol for breeding. Plant Breed 118:97–110

Mesterházy Á (2003a) Control of Fusarium head blight of wheat by fungicides. p. 363–380. *In*: Leonard KJ and Bushnell WR (eds) Fusarium Head Blight of Wheat and Barley. St Paul, MN: APS Press

Mesterházy Á (2003b) Breeding wheat for Fusarium head blight resistance in Europe. p. 211–240. *In*: Leonard KJ and Bushnell WR (eds) Fusarium Head Blight of Wheat and Barley. St Paul, MN: APS Press

Miedaner L (1997) Breeding wheat and rye for resistance to Fusarium diseases. Plant Breeding 116:201–220

Miedaner T, Moldovan M, and Ittu M (2003) Comparison of spray and point inoculation to access resistance to Fusarium head blight in a multienvironment wheat trial. Phytopathology 93:1068-1072

Miller JD, Young JC, Sampson RD (1985) Deoxynivalenol and Fusarium head blight resistance in spring cereals. Phytopathol Z 113:359–367

Milus EA, Parsons CE (1994) Evaluation of foliar fungicides for controlling Fusarium head blight of wheat. Plant Dis 78:697–699

Mirocha CJ, Kolaczkowski E, Xie W, Yu H, Jelen H (1998) Analysis of deoxynivalenol and its derivatives (batch and single kernel) using gas chromatography/mass spectrometry. J Agric Food Chem 46:1414–1418

Miller JD, Stack RW, and Joppa LR (1998) Evaluation of *Triticum turgidum* L. var. *dicoccoides* for resistance to Fusarium head blight and stem rust. p. 292-293. *In*: Slinkard AE (eds), Proc. 9th Int Wheat Genet Symp. Vol. 3, University Extension Press, University of Saskatchewan

Mergoum M, Frohberg RC, J. D. Miller JD, and Stack RW (2005) Registration of 'Steele-ND' wheat. Crop Sci 45:1163–1164

Murphy JP, Navarro R A, and Leath S (1999) Evaluation of diploid, tetraploid, hexaploid, and synthetic wheats for type ii resistance to Fusarium head blight. p. 133–136. *In*: Wagester JA *et al*. (eds), 1999 National Fusarium Head Blight Forum. Michigan State University, East Lansing, MI.

Nakagawa M (1995) Study on the resistance of wheat varieties to *Gibberella saubinetti*. II Genetic factors affecting resistance to *Gibberella saubinetti*. Jpn J Breed 5:15–22

Naranjo T, Roca A, Goicoechea PG, and Giraldez R (1987) Arm homoeology of wheat and rye chromosomes. Genome 29:873–882

Nelson JC, Sorrells ME, Van Deynze AE, Lu YH, Atkinson M, Bernard M, Leroy P, Faris JD, Anderson JA. (1995a) Molecular mapping of wheat: major genes and rearrangements in homoeologous groups 4, 5, and 7. Genetics 141:721–31

Nelson JC, Van Deynze AE, Autrique E, Sorrells ME, Lu YH, Merlino M, Atkinson M, and Leroy P (1995b) Molecular mapping of wheat: homoeologous group 2. Genome 38:516–524

Nelson JC, Van Deynze AE, Autrique E, Sorrells ME, Lu YH, Negre S, Bernard M, and Leroy P (1995c) Molecular mapping of wheat: homoeologous group 3. Genome 38:525–533

Nelson JC (1997) QGENE: software for maker-based genomics analysis and breeding. Mol Breeding 3:239–245

Nganje WE, Kaitibie S, Wilson WW, Leistritz FL, Bangsund DA (2004) Economic impacts of Fusarium head blight in wheat and barley: 1993–2001. NDSU Agribusiness and Applied Economics Report No. 538

Okubara PA, Blechl AE, McCormick SP, Alexander NJ, Dill-Macky R, *et al*. (2002) Engineering deoxynivalenol metabolism in wheat through the expression of a fungal trichothecene acetyltransferase gene. Theor Appl Genet 106:74–83

Oliver RE, Cai X, Stack RW, Friesen TL, Halley S, and Xu SS (2005) Fusarium head blight resistance in tetraploid wheat. p. 79. *In*: Canty SM, Lewis J, Siler L, and Ward RW (eds), Proceedings of the National Fusarium Head Blight Forum; 2005 Dec 11-13; Milwaukee, Wisconsin. East Lansing: Michigan State University, MI.

Olivera P, Steffenson B, and Anikster Y, (2003) Reaction of Aegilops sharonesis to Fusarium head blight. p.226. *In*: Canty SM *et al*. (eds), Proceedings of the 2003 National Fusarium Head Blight Forum; 2003 Dec 13-15; Bloomington, MN. East Lansing: Michigan State University, MI.

Otto CD, Kianian SF, Elias EM, Stack RW, Joppa LR (2002) Genetic dissection of a major Fusarium head blight QTL in tetraploid wheat. Plant Mol Biol 48:625–32

Paillard S, Schnurbusch T, Tiwari T, Messmer M, Winzeler M, Keller B, Schachermayr G (2004) QTL analysis of resistance to Fusarium head blight in Swiss winter wheat (*Triticum aestivum* L.) Theor Appl Genet 109:323–332

Paran I and Michelmore RW (1993) Development of reliable PCR-based markers linked to downy mildew resistance genes in lettuce. Theor Appl Genet 85:985–993

Parry DW, Jenkinson P, McLeod L (1995) Fusarium ear blight (scab) in small grain cereals - a review. Plant Pathol 44:207–238

Parry, DW, Pettitt TR, Jenkinson P, Lees AK (1984) The cereal Fusarium complex. p. 301–320.

In: Blakeman JP and Williamson B (eds). Ecology of Plant Pathogens. CAB International, Wallingford OX, UK.

Pavlová A and Šrobárová A (2004) Wheat resistance to seedling and head blight in slovakia. p. 142–147. *In*: Canty SM *et al*. (eds), Proc. 2nd Int Symp on Fusarium Head Blight, Orlando, Michigan State University, East Lansing, MI.

Pestsova E, Ganal MW, Röder MS (2000) Isolation and mapping of microsatellite markers specific for the D genome of bread wheat. Genetics 43:689–697

Qi LL, Echalier B, and Chao S *et al*. (2004) A Chromosome bin map of 16,000 expressed sequence tag loci and distribution of genes among the three genomes of polyploid wheat. Genetics 168:701–712

Reis EM (1990) Integrated disease management. The changing concepts of controlling head blight and spot blotch. p. 165–177 *In*: Saunders DA and Hettel G (eds) Wheat for Nnontraditional Warm Areas. Proceeding of International Conferences, Foz do Iguacu, Brazil. International Maize and Wheat Improvement Center (CIMMYT), Mexico

Riley R and Chapman V (1958) Genetic control of the cytologically diploid behavior of hexaploid wheat. Nature 182:713–715.

Röder MS, Plaschke J, Wendehake K, Plaschke J, Tixier MH, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007–2023

Rohlf FJ (1998) Numerical taxonomy and multivariate analysis system. Version 2.0. Exeter Software, N.Y.

Ruckenbauer P, Buerstmayr H, Lemmens M (2001) Present strategies in resistance breeding against scab (*Fusarium* spp.). Euphytica 119:121–127

Rudd JC, Horsley RD, McKendry AL, Elias EM (2001) Host plant resistance genes for Fusarium head blight: sources, mechanisms, and utility in conventional breeding systems. Crop Sci 41:620–27

Sax K (1922) Sterility in wheat hybrids. II. Chromosome behavior in partially sterile hybrids. Genetics 7:513–550.

Saghai Maroof MA, Soliman KM, Jorgensen RA, Allard RW (1984) Ribosomal DNA spacer length polymorphism in barley: Mendelian inheritance, chromosomal location, and population dynamics. Proc Natl Acad Sci (USA) 81:8014–8018

Schmolke M, Zimmermann G, Buerstmayr H, Schweizer G, Miedaner T, Korzun V, Ebmeyer E, and L. Hart (2005) Molecular mapping of Fusarium head blight resistance in the winter wheat population Dream/Lynx. Theor Appl Genet 111:747–756

Schroeder HW, Christensen JJ (1963) Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831–838

Sears ER (1954) The aneuploids of common wheat. Mo Agric Exp Sta Res Bull 572:1–58.

Sears ER (1966) Nullisomic-tetrasomic combinations in hexaploid wheat. p. 29–45. *In*: Riley R and Lewis KR (eds) Chromosome manipulations and plant genetics. London, Oliver and Boyd.

Sears ER and Sears LMS (1978) The telocentric chromosomes of common wheat. p. 389–407. *In*: Ramanujams S (ed) Proc. 5th Int. Wheat Genetics Symp. New Delhi, Indian Agricultural Research Institute.

Shaner GE, Buechley G (2001) New sources of resistance to Fusarium head blight of wheat. p. 203. *In*: Canty SM *et al*. (eds), 2001 National Fusarium Head Blight Forum Proceedings. Elanger, KY, East Lansing: MI State Univ. Press

Shaner GE (2002) Resistance in hexaploid wheat to Fusarium head blight. p. 208–211. *In*: Canty SM *et al*. (eds), 2002 National Fusarium Head Blight Forum Proceedings. Elanger, KY, East Lansing: MI State Univ. Press

Shaner GE, Buechley G (2003) Relation between head blight and grain quality in the Indiana FHB epidemic of 2003. p. 178–181. *In*: Canty SM *et al*. (eds), 2003 National Fusarium Head Blight Forum Proceedings. Bloomington, MN, Okemos, MI: Kinko's

Shaner GE. 2003. Epidemiology of Fusarium head blight of small grain cereals in North America. p. 84 –119. *In*: Leonard KJ and Bushnell WR (eds), Fusarium Head Blight of Wheat and Barley. St Paul, MN: APS Press

Shen X, Ittub M, Ohm HW (2003a) Quantitative trait loci conditioning resistance to Fusarium head blight in wheat line F201R. Crop Sci 43:850–857

Shen X, Zhou M, Lu W, Ohm H (2003b) Detection of *Fusarium* head blight resistance QTL in a wheat population using bulked segregant analysis. Theor Appl Genet 106:1041–1047

Shen XR, Kong LR, and Herbert Ohm H (2004) Fusarium head blight resistance in hexaploid wheat (*Triticum aestivum*)- *Lophopyrum* genetic lines and tagging of the alien chromatin by PCR markers. Theor Appl Genet 108:808–813

Simons KJ (2005) Cloning and Characterization of the Wheat Domestication Gene, Q. PhD thesis, Kansas State University. 181 pp

Simons KJ, Fellers JP, Trick HN, Zhang ZC, Tai YS, Gill BS and Faris JD (2006) Molecular characterization of the major wheat domestication gene *Q*. Genetics 172:547–555

Simpson DR, Weston GE, Turner JA, Jennings P and Nicholson P (2001) Differential control of

head blight pathogens of wheat by fungicides and consequences for mycotoxin contamination of grain. European Journal of Plant Pathology 107:421–431

Singh RP, Ma H, Rajaram S (1995) Genetic analysis of resistance to scab in spring wheat cultivar Frontana. Plant Dis 79:238–240

Singh RP, van Ginkel M (1997) Breeding strategies for introgressing diverse scab resistance into adapted wheat. p. 86–92. *In*: Dubin HJ, Gilchrist L, Reeves J, and McNab A (eds), Fusarium Head Scab: Global Status and Future Perspects. Mexico, DF: CIMMYT.

Snijders CHA (1990a) Genetic variation for resistance to *Fusarium* head blight in bread wheat. Euphytica 50:171–179

Snijders CHA (1990b) The inheritance of resistance to head blight caused by *Fusarium culmorum* in winter wheat. Euphytica 50:11–18

Snijders CHA (1990c) Fusarium head blight and mycotoxins contamination of wheat, a review. Netherland J Plant Pathol 96:187–198

Snijders CHA, Perkowski J (1990) Effects of head blight caused by *Fusarium culmorum* on toxin content and weight of wheat kernels. Phytopathology 80:566–570

Snijders CHA, van Eeuwijk FA (1991) Genotype × strain interactions for resistance to Fusarium head blight caused by *Fusarium culmorum* in winter wheat. Theor Appl Genet 81:239–244

Somers DJ, Fadak G, Savard M (2003) Molecular mapping of novel genes controlling Fusarium head blight resistance and deoxynivalenol accumulation in spring wheat. Genome 46:555–564

Somers DJ, Isaac P, Edwards K (2004) A high-density microsatellite consensus map for bread wheat (*Triticum aestivum* L.). Theor Appl Genet 109:1105–1114

Song QJ, Shi JR, Singh S, Fickus EW, Costa JM, Lewis J, Gill BS, Ward R, Cregan PB (2005) Development and mapping of microsatellite (SSR) markers in wheat. Theor Appl Genet 110:550–560

Sorrells ME, La Rota M, Bermudez-Kandianis CE *et al*. (2003) Comparative DNA sequence analysis of wheat and rice genomes. Genome Research 13:1818–1827

Sourdille P, Cadalen T, Guyomarc'h H, Snape JW, Perretant MR, Charmet G, Boeuf C, Bernard S, Bernard M (2003) An update of the Courtot × Chinese Spring intervarietal molecular marker linkage map for the QTL detection of agronomic traits in wheat. Theor Appl Genet 106:530–538

Sourdille P, Singh S, and Cadalen T *et al*. (2004) Microsatellite-based deletion bin system for the

establishment of genetic-physical map relationships in wheat (*Triticum aestivum* L.). Funct Integr Genomics 4:12–25

Steiner B, Lemmens M, Griesser M, Scholz U, Schondelmaier J, Buerstmayr H (2004) Molecular mapping of resistance to Fusarium head blight in the spring wheat cultivar Frontana. Theor Appl Genet 109:215–224

Strange RN, Smith H (1987) Specificity of choline and betaine as stimulants of *Fusarium graminearum*. Trans Br Mycol Soc 70:187–192

Stuchlikova E and Sip V (1996) Resistance of Czech and Slovak winter wheat varieties to Fusarium head blight. Genetika Slechteni 32:79–94

Sun G, Bond M, Nass H, Martin R, Dong Z (2003) RAPD polymorphisms in spring wheat cultivars and lines with different level of Fusarium resistance. Theor Appl Genet 106:1059–1067

Sutton JC (1982) Epidemiology of wheat head blight and maize ear rot caused by *Fusarium graminearum*. Can J Plant Pathol 4:195–209

Talbert LE, Smith LY, and Blake NK (1998) More than one origin of hexaploid wheat is indicated by sequence comparison of low-copy DNA. Genome 41:402–407.

Tanksley S (1983) Molecular markers in plant breeding. Plant Mol Biol Rep 1:3–8

Tautz D and Renz M (1984) Simple sequences are ubiquitous repetitive components of eukaryotic genomes. Nucleic Acids Res 12:4127–4138

Teich AH, Hamilton JR (1985) Effect of culture practices, soil phosphorous, potassium, and pH on the incidence of *Fusarium* head blight and deoxynivalenol levels in wheat. Appl Environ Microbiol 49:1429–1431

Tuite J, Shaner GE, Everson RJ (1990) Wheat scab in soft red winter wheat in Indiana in 1986 and ist relation to some quality measurements. Plant Dis 74:959–962

Van Deynze AE, Dubcovsky J, Gill KS, Nelson JC, Sorrells ME, Dvorak J, Gill BS, Lagudah ES, McCouch SR, and Appels R (1995a) Molecular genetic maps for group 1 chromosomes of Triticeae species and their relation to chromosomes in rice and oat. Genome 38:45–59

Van Deynze AE, Nelson JC, Yglesias ES *et al*. (1995b) Comparative mapping in grasses – wheat relationships. Molecular and General Genetics 248:744–754

Van Deynze AE, Sorrells ME, Park WD *et al*. (1998) Anchor probes for comparative mapping of grass genera. Theor Appl Genet 97:356–369

van Eeuwijk FA, Mesterházy A, Kling CHI, Ruckenbauer P, Saur L *et al*. (1995) Assessing non-specificity of resistance in wheat to head blight caused by inoculation with European strains of *Fusarium culmorum*, *F. graminearum* and *F. nivale* using a multiplicative model for interaction. Theor Appl Genet 90:221–228

van Ginkel M, van Der Schaar W, Zhuping Y, Rajaram S (1996) Inheritance of resistance to scab in two wheat cultivars from Brazil and China. Plant Dis 80:863–867

van Ooijen JW, Voorrips RE (2001) JoinMap 3.0, Software for the calculation of genetic linkage maps. Plant Research International, Wageningen, the Netherlands

van Slageren MW (1994) Wild wheats: a monograph of *Aegilops* L. and *Amblyopyrum* (Jaub. & Spach) Eig (Poaceae). Wageningen, Netherlands, Agricultural University. 512 pp.

Vos P, Hogers R, Bleeker M, Reijans M, van de Lee T, Hornes M, Fritjers A, Pot J, Peleman J, Kuiper M and Zabeau M (1995) AFLP: a new concept for DNA fingerprinting. Nucleic Acids Res 23:4407–4414

Waldron BL, Moreno-Sevilla B, Anderson JA, Stack RW, and Frohberg RC (1999) RFLP mapping of QTL for Fusarium head blight in wheat. Crop Sci 39:805–811

Wan, YF, Yen C, Yang JL (1997) The diversity of head-scab resistance in Triticeae and their relation to ecological conditions. Euphytica 97:277–281

Wang S, Basten CJ, Zeng ZB (2006). Windows QTL Cartographer 2.5. Department of Statistics, North Carolina State University, Raleigh, NC

Wang YZ, Miller JD (1988) Effects of *Fusarium graminearum* metabolites on wheat tissue in relation to FHB resistance. J Phytopath 122:118–125

Wang YZ, Miller JD (1988) Screening techniques and sources of resistance to Fusarium head blight. p. 239–250. *In*: Klatt AR (eds), Wheat Production Constraints in Tropical Environments. CIMMYT, Mexico

Weber JL and May PE (1989) Abundant class of human DNA polymorphism which can be typed using the polymerase chain reaction. Am J Hum Genet 44:388–396

Wei YM, Hou YC, Yan ZH, Wu W, Zhang ZQ, Liu DC, and Zheng YL (2005) Microsatellite DNA polymorphism divergence in Chinese wheat (*Triticum aestivum* L.) landraces highly resistant to Fusarium head blight. J Appl Genet 46:3–9

Weng YQ and Liu DJ (1991) Morphological and cytological investigation of interspecific hybrids between *Roegneria ciliaris*, *R. japonensis*, and *R. kamoji*. J Nanjing Agri Univ 14:1–5 (in Chinese with English abstract).

Werner JE, Endo TR, and Gill BS (1992) Toward a cytogenetically based physical map of the

wheat genome. Proc Natl Acad Sci 89:11307–11311

Wiese MV (1987) Scab (head blight) p. 16–18. *In*: Compendium of Wheat Diseases. 2nd ed. American Phytopathological Socciety, St Paul, MN

Williams JGK, Kubelik AR, Livak KJ, Rafalski JA, and Tingey SV (1990) DNA polymorphisms amplified by arbitrary primers are useful as genetic markers. Nucleic Acids Res 18:6531–6535

Windels CE (2000) Economic and social impacts of Fusarium head blight: Changing farms and rural communities in the northern Great Plains. Phytopathology 90:17–21

Wiśniewska H, Perkowski J, Kaczmarek Z (2004) Scab response and deoxynivalenol accumulation in spring wheat kernels of different geographical origins following inoculation with *Fusarium culmorum*. J Phytopath 152:613–621

Witcombe JR (1983) A guide to the species of *Aegilops* L. I. B. P. G. R. Secretariat, Rome.

Xu DH, Juan HF, Nohda M, Ban T (2001) QTLs mapping of type I and type II resistance to FHB in wheat. p. 40–42. *In*: Canty SM *et al*. (eds), 2001 National Fusarium Head Blight Forum Proceedings, Erlanger, KY, 8–10 Dec. 2001. Michigan State University, East Lansing, MI.

Yang ZP, Gilbert J, Somers DJ, Fedak G, Procunier JD, McKenzie IH (2003) Marker assisted selection of *Fusarium* head blight resistance genes in two doubled haploid populations of wheat. Molecular Breeding 12:309–317

Yang J, Bai GH, and Shaner GE (2005a) Novel quantitative trait loci (QTL) for Fusarium head blight resistance in wheat cultivar Chokwang. Theor Appl Genet 111:1571–1579

Yang ZP, Gilbert J, Fedak G, and Somers DJ (2005b) Genetic characterization of QTL associated with resistance to Fusarium head blight in a doubled-haploid spring wheat population. Genome 48:1–10

Yang ZP, Gilbert J and Procunier JD (2006) Genetic diversity of resistance genes controlling Fusarium head blight with simple sequence repeat markers in thirty-six wheat accessions from east asian origin. Euphytica 148:345–352

Yao JB, Ge YF, Wang SW, Yao GC, Zhou CF, Qian CM (1997) Chromosomal location of genes for scab resistance in wheat cultivar Sumai 3. Acta Agron Sin 23:450–453

Yu YJ (1991). Genetic analysis for scab resistance in five wheat varieties, PingHuJianZiMai, HongHuDaTaiBao, ChongYangHongMai, YanGangFangZhu, and Wannian 2. Acta Agron Sinica 17:248–254.

Yu JB, Bai GH, Cai SB, Ban T (2006) Marker-assisted characterization of Asian wheat lines for

resistance to Fusarium head blight. Theor Appl Genet 113:308–320

Zeng ZB (1994) Precision mapping of quantitative trait loci. Genetics 136:1457–468

Zhang, X., Y. Jin YR. Rudd, T. Hall, J. Rudd, and H. Bockelman (2000) Fusarium head blight resistance sources of spring wheat identified from the USDA collection. p. 228–233. *In*: Ward RW *et al*. (eds), 2000 National Fusarium Head Blight Forum Proceedings. Kinko's, Okemos, MI.

Zhang X, Jin Y, Rudd J, and Bockelman H (2001) Evaluation of USDA spring wheat germplasm for Fusarium head blight resistance. p. 220–224. *In*: Canty SM *et al*. (eds), 2001 National Fusarium Head Blight Forum Proceedings. Kinko's, Okemos, MI.

Zhang X, Zhou MP, Ren LJ, Bai GH, Ma HX, Scholten OE, Guo PG, Lu WZ (2004) Molecular characterization of *Fusarium* haed blight resistance from wheat variety Wangshuibai. Euphytica 139:59–64

Zhou WC, Kolb FL, Bai GH, Shaner G, and Domier LL (2000) SSR mapping and sub-arm physical location of a major scab resistance QTL in wheat. p. 69–73. *In*: Ward RW *et al*. (eds), 2000 National Fusarium Head Blight Forum Proceedings, Erlanger, KY. 10–12 Dec. 2000. Michigan State University, East Lansing, MI.

Zhou WC, Kolb FL, Bai GH, Domier LL, and Yao JB (2002) Effect of individual Sumai 3 chromosomes on resistance to scab spread within spikes and deoxynivalenol accumulation within kernels in wheat. Hereditas 137:81–89

Zhou WC, Kolb FL, Yu JB, Bai GH, Boze LK, Domier LL (2004) Molecular characterization of *Fusarium* head blight resistance in Wangshuibai with simple sequence repeat and amplified fragment length polymorphism markers Genome 47:1137–1143

Zhou WC, Kolb FL. Bai GH, Shaner GE, Domier LL (2002) Genetic analysis of scab resistance QTL in wheat with microsatellite and AFLP markers. Genome 45:719–727

Zhu H, Fan Y (1989) Wheat scab epidemics and efficacy of testing for resistance in field in northern Fujiang province. Jiangsu Agric Sci 1:75–78.

Zohary D and Feldman M (1962) Hybridization between amphidiploids and the evolution of polyploids in the wheat (*Aegilops-Triticum*) group. Evolution 16:44-61