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MANAGEMENT AND RESISTANCE IN WHEAT AND BARLEY TO FUSARIUM HEAD BLIGHT[1]

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■ **Abstract** Fusarium head blight (FHB) is a devastating disease of wheat and barley worldwide. Resistant cultivars could reduce damage from FHB. Chinese wheat cultivar Sumai 3 and its derivatives represent the greatest degree of resistance to FHB known. A major quantitative trait locus (QTL) on chromosome 3BS and other minor QTL for FHB resistance have been identified in these cultivars and used in wheat-breeding programs worldwide. Many breeding lines with the 3BS resistance QTL and improved agronomic traits have been developed. In barley, only limited sources of FHB resistance are available, especially in six-rowed barley, and none of them contains a DON level low enough to meet the safety requirement of the brewing industry. Several QTL have been identified for lower FHB severity, DON content, and kernel discoloration and used to enhance FHB resistance in barley. Marker-assisted selection for FHB resistance QTL on 3BS of wheat and on 2H of barley is in progress.

INTRODUCTION

Fusarium head blight (FHB), also called scab, is a devastating and insidious disease of wheat and barley in humid and semihumid areas worldwide (91, 108). Although many *Fusarium* species can cause the FHB, *F. graminearum* Schwabe [teleomorph = *Gibberella zeae* (Schw.) Petch] is the principal pathogen in many countries (13, 69, 91, 93, 112, 120). In China, FHB has affected more than 7 million hectares of wheat and has caused yield losses of more than 1 million tons in severe

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epidemics (9, 62, 69, 120). In the United States, several severe FHB outbreaks on wheat and barley from 1991–1997 resulted in about $1.3 billion of total direct losses and $4.8 billion of losses for the accumulative economic impact of FHB (56). The estimated yield loses of barley due to FHB was 70 million tons, with a raw commodity value of $122 million in the 1993 epidemic alone (107). FHB has also been a threat to wheat and barley production in many other countries (13, 23, 52, 75, 112).

FHB can also cause indirect loss because the fungus contaminates grain with potent mycotoxins, especially deoxynivalenol (DON) (19, 42). DON contamination has raised serious food safety concerns. High levels of DON have been reported in the harvested grain of *Fusarium*-infected wheat and barley (19, 42, 79, 85, 108). The maximum acceptable DON levels for human consumption in wheat grain have been set from 0.5 to 2 ppm in the United States, Canada, and some European countries (42, 93). The acceptable DON level is even lower in malting barley (<0.5 ppm) (108). Grain with a low level of this toxin may command a lower price or be rejected entirely in commerce (13).

Fusarium graminearum survives saprophytically in residue of small grains and maize. Various cultural practices have been proposed to eliminate sources of primary inoculum (13). The adoption of minimum tillage for soil conservation reduces options for this disease-management technique. Seed treatment and a foliar application of fungicide at anthesis might provide some protection (76). Several fungicides are registered for application on wheat and barley late in the season (57, 95, 108). The cost of treatment, the difficulty of determining the optimum time of application, and the lack of any registered fungicides that are highly effective have limited the use of chemical protection against FHB. Even if a fungicide reduces direct yield loss, it may not reduce mycotoxin contamination to a level tolerable for human consumption (71, 108).

Genetic resistance has the potential to provide economical and effective control of this disease. Considerable progress in the search for host resistance has been made in China, Japan, and some other countries in past two decades (9, 20, 22, 69, 75). Improvement of cultivar resistance has become a major breeding objective worldwide. Recent developments in genomic research and biotechnology hold promise for understanding the genetic mechanisms of FHB resistance and allow more effective utilization of FHB resistance genes to develop new resistant wheat and barley cultivars.

SOURCE OF INOCULUM AND DEVELOPMENT OF FHB

F. graminearum survives on a wide range of hosts including not only living plants such as wheat, corn, barley, soybean, and rice, but also on dead tissue of many plant species (93, 124). Crop residues on the soil surface are the major reservoir of pathogens of FHB (93). Ascospores, macroconidia, chlamydospores, and hyphal fragments all can serve as inoculum (13), but ascospores released from soil surface debris are the principal inoculum that initiates epidemics (13, 93, 124). Wheat

planted after corn or wheat often has significantly more FHB than when it is planted after other crops (93). Reduced tillage for soil conservation increases the amount of inoculum that can infect wheat (13).

Airborne spores released from crop residue are deposited on or inside wheat florets where they germinate and initiate infection. The fungus rapidly infects the extruded anthers and then ramifies throughout the developing caryopsis, floral bracts, and rachis (13, 32). The fungus may also infect by direct penetration of glumes, palea, or rachilla (32). Soon after infection, dark-brown, water-soaked spots appear on the glumes of infected florets. Later, entire florets become blighted. The fungus infects other spikelets internally through vascular bundles of the rachilla and rachis in susceptible wheat (32). Blight becomes more severe as the fungus spreads within a spike, and eventually the entire spike will become blighted. Infected florets often fail to produce grain, or the grain they produce is poorly filled. In barley, although the fungus is known to spread on the exterior of the spike under wet conditions, internal spread in the rachis is more limited (32).

When temperature and moisture are favorable, infection can occur any time after commencement of flowering in wheat, but anthesis is the growth stage most vulnerable to infection (1, 6, 110). Because of this brief period of vulnerability, the fungus is generally limited to one infection cycle per season (13). Primary infection can occur on several florets of a spike in field conditions. The dark-brown symptoms usually extend into the rachis, even down into the stem tissue as the fungus spreads within a spike. 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 (7, 91). If heads are extensively invaded at a very early stage, kernels may fail to develop entirely, which significantly reduces grain yield and quality. The abundance of primary inoculum and weather conditions, mainly moisture and temperature, during and after anthesis determine the severity of *Fusarium* head blight.

THE PATHOGENS

More than 17 *Fusarium* species have been isolated from naturally infected wheat or barley spikes (45, 69, 93, 121). All of these species can infect wheat and barley when spikes are inoculated, but with various levels of virulence (45, 75, 121). *F. graminearum* is the most frequently encountered pathogen and the most virulent species worldwide, although *F. culmorum* and *F. poae* are reported to be prevalent in some European countries (9, 45, 75). For natural infection, ascospores of *Gibberella zeae* released from perithecia usually are the major primary inoculum to initiate disease epidemics (93). Macroconidia are equally infective and commonly used for inoculation of wheat and barley (13).

Pathogen isolates may vary in cultural characters, virulence, toxigenicity, aggressiveness, and vegetative compatibility groups (13, 14, 26, 43, 75, 120). Isolates of *F. graminearum* from regions where FHB epidemics are severe and frequent may be more virulent than those from regions where disease pressure is less (14,

121). Highly virulent isolates may cause more severe symptoms in moderately resistant cultivars than do less virulent isolates (14). However, isolate-specific resistance has not been detected.

TYPES OF RESISTANCE

Type I and Type II Resistance to FHB

Schroeder & Christensen (91) proposed two types of resistance in wheat: resistance to initial infection (now referred to as type I) and resistance to spread of blight symptoms within a spike (now referred to as type II). Type II resistance has been extensively studied in wheat and appears to be more stable and less affected by nongenetic factors than type I resistance (13). Type II resistance has been found in a number of wheat cultivars. In contrast, FHB symptoms in barley usually do not spread internally from initially infected spikelets to adjacent spikelets (32). Therefore, type II resistance, although reported for barley (132), has little meaning. Type I resistance is more important for barley (108).

To distinguish the two types of resistance, different inoculation methods are used in wheat. Type II resistance is estimated by delivering conidia into a single floret of a spike and counting the blighted spikelets after some period of time. In a susceptible cultivar, all of the spikelets will become blighted from an initial inoculation of a single floret in as few as 10 days. Type I resistance is estimated by spraying a spore suspension over flowering spikes and counting the diseased spikelets. Both of these procedures are typically done in a greenhouse, where the conditions for infection can be carefully controlled. For both techniques, visual estimation of the proportion of spikelets blighted may be used in lieu of counts, especially when many plants must be evaluated. Whether as counts of blighted spikelets or visual estimates of percentage of blighted spikelets, data can be recorded at intervals (3 to 5 days) after inoculation, to discern the pattern of FHB progress. Such repetitive data can be summarized as the area under the disease progress curve (AUDPC) (97). In this case, the increase in severity of blight is not the result of secondary infection, but rather the invasion of the spike internally following initial infection of one (point inoculation) or more (spray inoculation) florets (13, 43, 75). The assumption for assessment of type I resistance by use of spray inoculation is that all spikes are exposed to inoculum, and the degree of type I resistance will determine what proportion of spikes is infected. Timing of inoculation may be critical for evaluation of type I resistance. Inoculation should be performed when anthers are extruded from all florets. If a plant is inoculated earlier than that, what might appear to be type I resistance could simply be disease escape.

Type I resistance can only be detected under low disease pressure. When a large number of spores are sprayed onto a head followed by a long incubation in moist conditions, differences in degree of type I resistance may not be distinguishable among cultivars. Bai (7) compared the two types of resistance in 5 cultivars ranging from highly resistant (Ning 7840) to highly susceptible (Clark) and using inoculum

concentrations of 2 to 11,000 conidia per spike. Great differences were found in type II resistance among cultivars when 200 or more spores per spikelet were point-inoculated. At this concentration of inoculum, the symptoms in Ning 7840 did not spread from inoculated spikelet (AUDPC of 0.5), whereas Clark was completely blighted (AUDPC of 7.5). However, when about 10,000 spores were sprayed over a spike, all inoculated spikes were infected, and there was no significant difference in incidence among cultivars. Following spray inoculation, many spikes of both resistant and susceptible cultivars had multiple infection sites. Because type II resistance seems less influenced by inoculum dose and moist period duration, it has been widely used to measure FHB resistance of germplasm and breeding materials.

In the field, nurseries may be planted on land with abundant residue of a host crop (wheat, barley, corn), or grain on which the fungus has been cultured may be scattered over the soil surface. With either of these inoculation methods, infections may occur whenever weather conditions are favorable and the host is at a vulnerable stage of growth. Wheat plants may also be sprayed with spore suspensions during the period of anthesis. Because of variation in flowering time among test lines in a nursery, several inoculations may be required, at two- to three-days intervals (43).

It may be difficult to distinguish type II from type I resistance in the field under conditions favorable for infection throughout the flowering period. Plants with only type I or type II resistance may appear to be susceptible. A plant with type I resistance but little type II resistance may appear to be susceptible when inoculum is abundant. In this case, at least one spikelet will likely become infected and blight symptoms will subsequently spread throughout the spike from the infected spikelet (14). If a plant has type II resistance but no type I resistance, infection will be limited to initially infected spikelets. However, if a high proportion of spikelets are infected directly, because of heavy inoculum pressure and favorable environment, severe blight will result regardless of type II resistance. In barley, since type II resistance is not the major type of resistance, disease index (the product of the proportion of spikes showing blight and the proportion of spikelets on affected spikes that are blighted) may be a good measurement of type I resistance.

Wheat cultivar Sumai 3 and its derivatives have excellent type II resistance, and FHB symptoms usually do not spread to uninoculated spiklets after point inoculation in the greenhouse. Sumai 3 represents near the highest degree of type II resistance in wheat. However, several infected spikelets can also be observed in a single spike of Sumai 3 under heavy disease pressure in the field. This probably results from multiple primary infections, not from disease spread within the spike. Adding additional type II resistance genes into Sumai 3 may not increase its resistance in the field. Adding type I resistance to Sumai 3 may improve its overall resistance performance under field conditions.

Other Types of Resistance

Three other types of resistance have been proposed (73, 77): resistance to kernel infection, to DON accumulation, and tolerance. Resistance to kernel infection can

be measured as the percentage of infected kernels. However, the presence of type I or type II resistance would presumably also reduce the degree of kernel infection, so this must be taken into account when attempting to quantify resistance to kernel infection per se (92). Tolerance can be measured by relative yield reduction when diseased and healthy plants of the same cultivar are compared in a statistically sound experimental design. These two resistance types have not been widely accepted because of some conceptual or operational weaknesses (92). Because DON in the grain reduces starch and protein quality and is toxigenic to humans and other animals, it adds additional economic losses to wheat and barley producers and processors. This is especially important to malting barley because even trace levels of DON may significantly reduce beer quality. Wheat and barley cultivars with low or no DON would be very desirable for reducing damage caused by this disease.

Infected grain will usually contain DON regardless of the level of resistance of a cultivar to head blight. However, DON contents differ among cultivars (19). Low DON accumulation in some wheat cultivars compared to other cultivars in the same environment has been named type III resistance (78). Low DON content in a kernel that has been infected could result from three possible causes: (*a*) a low level of DON produced by the fungus, (*b*) a degradation of DON by plant enzymes during kernel development, or (*c*) a high level of DON in spike tissue other than kernels, but failure of DON to move into kernels during their development. Resistant cultivar Frontana produces enzymes that degrade DON (78). Low DON content in a bulk sample of harvested grain may result from fewer infected kernels as well as from the mechanisms described above. Fewer infected kernels may be due to a high level of type I or type II resistance, or due to loss of the most severely affected kernels, with the highest content of DON, during combine harvesting. In general, a significant positive correlation between FHB severity and DON content in harvested grain can be observed when cultivars from full ranges of FHB resistance are analyzed (19, 96). However, when only susceptible and moderately susceptible cultivars are included in an experiment, this correlation may not be obvious (19). In addition, the amount of DON in harvested grain of a susceptible cultivar may vary significantly with methods used for DON analysis, growing conditions, weather after flowering, and harvesting method (19, 79).

Accurate measurement of resistance to DON accumulation still poses problems that may confound data interpretation. In general, DON is measured from harvested grains, which serves the purpose of quantifying DON content in the product that will be processed into food or feed. In this case, DON content is usually highly correlated with fungal biomass (104). If infection occurs during flowering, the infected ovary may never develop into a mature kernel, or the kernel may be so small and light that it will be blown away during threshing (13). These kernels may have the highest levels of DON, and thus the DON content in the harvested grain may be lower than might be expected based on intensity of head blight symptoms. For some moderately resistant cultivars, although infection occurs early, infected kernels may grow to normal size because of the faster grain-filling rate of these

cultivars or the slower rate of fungal invasion of the spike. These cultivars can have an unacceptably high level of DON in harvested grain. On the other hand, if infection occurs later in kernel development, and weather favors fungal growth after infection, DON may also accumulate to a high level in harvested grain. In both cases, a high DON content in harvested grain results because these infected kernels are not light enough to be blown out of the combine with the chaff during harvesting. Whether resistance to DON accumulation is independent of type I or type II is still equivocal. Some molecular mapping studies suggest that major resistance QTL for low FHB severity was also associated with low DON content in wheat and barley (8, 40). Low FHB severity due to type I and type II resistance usually coincides with low DON because of fewer infected kernels (19). More research is needed to determine whether the same or tightly linked QTL controls both traits. To determine if there is resistance to DON accumulation that is independent of type I or II resistance or resistance to kernel invasion poses methodological difficulties. Also methodology for DON measurement still needs to be improved and standardized.

Molecular and Biochemical Mechanisms of Resistance

There have been many attempts to elucidate the mechanisms of wheat resistance to FHB (10, 37, 41, 73, 77, 86, 87), but the biochemical and molecular basis of resistance is largely unknown. The expression of defense-related proteins PR-1, PR-2 (glucanases), PR-3 (chitinase), PR-4 (thaumatin-like proteins), PR-5, and peroxidase was induced in both resistant and susceptible cultivars after point inoculation (87). These proteins were detected as early as 6 to 12 h after inoculation and reached the peak after 36 to 48 h (86). PR-4 and PR-5 transcripts expressed earlier and greater in Sumai 3 than in susceptible cultivar Wheaton (86). Chitinases and β-1,3-glucanases also accumulated faster in Sumai 3 than in its susceptible mutant (63). Expression of a rice thaumatin-like protein gene in wheat delayed FHB symptoms in wheat spikes inoculated with *Fusarium* (37). This suggests that defense-related genes in wheat are activated after fungal infection and defense-related proteins may play some roles in general defense against *Fusarium* infection, but they may not be the key genes responsible for resistance.

Several other enzymes, such as superoxide dismutase, catalase, phenylalanine ammonia lyase, ascorbic acid peroxidase, and ascorbic acid oxidase, have also been related to FHB resistance in wheat. Several groups in China conducted extensive experiments to compare the activities of these enzymes between resistant and susceptible cultivars (35, 69). For example, the activity of superoxide dismutase in the spikes of a resistant cultivar was significantly higher (600 to 700 U/gfw) than that in susceptible cultivars (300 to 500 U/gfw) (34). Opposite results were observed for superoxide dismutase. The specific activity of peroxidase in infected spikes of the resistant cultivars increased until 16 days after infection, but the activity of this enzyme started to decrease in the susceptible cultivars 8 days after infection (123).

Differences in preformed chemical compounds between resistant cultivars and susceptible cultivars have been related to FHB resistance. Strange et al. (110, 111) identified two major components in anthers and wheat germ that could stimulate *Fusarium* growth in vitro. Choline content in susceptible spikes was twice that in a resistant cultivar during anthesis (64). A higher content of chlorogenic acid (a phenolic compound) was detected in susceptible cultivar Nannong 824 than in resistant cultivar Sumai 3 (126).

DON produced by the fungus during fungal infection has been proposed as a virulence factor. Disruption of the gene encoding trichodiene synthase (*Tri*5) in *Fusarium graminearum*, the enzyme catalyzing the first step in the DON biosynthetic pathway, reduced disease severity (41). Restoration of the *Tri5* gene results in increased FHB severity and DON production (41). The DON-nonproducing strain of *F. graminearum* still could infect an inoculated spikelet of wheat in both greenhouse and field (10). DON may not be essential for primary infection by the fungus, but may enhance symptom development and spread of the fungus within a spike. If this is true, low DON content in an infected kernel or expression of a DON detoxification gene from the fungus in wheat may improve wheat resistance. More recently, the *Tri101* gene has been successfully transformed into wheat (83). *Tri101* is a gene from *F. sporotrichioides* encoding an enzyme that transfers an acetyl group to the C_3 hydroxyl group of trichothecenes, including DON, and its expression may limit the accumulation of DON in wheat. Some transgenic plants demonstrated significantly reduced FHB severity in greenhouse experiments.

Resistance in wheat to FHB is a complex, quantitative trait. Resistance probably involves a complex and interacting network of signaling pathways. Application of the new technology for large-scale gene analysis may facilitate discovery of critical pathways and key genes in these pathways. Microarray analysis may allow monitoring the genome-wide gene expression in a single experiment (58, 90). Global monitoring of expression of defense-related genes may lead to a better understanding of the molecular basis of wheat defense against infection by *F. graminearum*, provide more insight into defense-related signal pathways, and facilitate identification of key genes involved in the pathways. Recently, Bernardo et al. (24) isolated about 2300 differentially expressed sequence tags (ESTs) from three suppression subtractive hybridization (SSH) libraries generated from wheat spikes that had been infected with *F. graminearum* for 6, 36, or 72 h previously. The wheat materials consisted of bulked recombinant inbred lines (RILs) from a cross between resistant cultivar Ning 7840 and susceptible cultivar Clark. Complementary DNA microarrays with the ESTs were analyzed to determine *Fusarium*-induced and differentially expressed cDNAs in Ning 7840 and Clark. Preliminary results suggest that more genes are upregulated than are downregulated during the first 24 h after inoculation, but the opposite is true from 36 h after inoculation and onward. Further microarray analysis with near-isogenic lines from Ning7840/Clark is under way. In barley, the Affymetrix GeneChip with over 22,800 unique barley transcripts has been generated and used to examine transcript profiles in barley in response to inoculation with *F. graminearum* (60).

SOURCES OF FHB RESISTANCE

Arthur (6) was the first to note differences in susceptibility to scab (FHB) among wheat cultivars. Considerable attention since then has been devoted to finding sources of resistance that can be used in breeding programs (17, 23, 44, 55, 69; also see http://www.scabusa.org/forum.html). The search for FHB-resistance sources has shown progress in China, Japan, and some other countries in the past three decades (9, 38, 69). In China, 32 wheat accessions were identified to have a high level of resistance after more than 17,000 wheat accessions were screened nationwide (38). Unfortunately, most of the resistant accessions were tall landraces that had small heads, late maturity, and other undesirable agronomic traits. Some of these landraces were used as parents in breeding programs, but the resistance was difficult to incorporate into elite lines. For example, Chinese landrace Wangshuibai has a high level of FHB resistance and has been used as a parent in many breeding programs, but incorporation of resistance genes from Wangshuibai into elite breeding lines has been unsuccessful to date because of their association with undesired agronomic traits. Sumai 3 is an improved cultivar with good combining ability for both FHB resistance and yield traits, and has been successfully used as a resistant parent in wheat-breeding programs worldwide (9, 22, 51, 69).

Wheat accessions with FHB resistance were also identified in Japan (21, 22). Shinchunaga, Nobeokabouzu and Nyu Bai were reported to have a high level of resistance. Among them, Shinchunaga has been successfully used in improving FHB resistance in breeding programs in Japan (22). These resistance materials were also used as parents in the United States and many other countries (39, 49, 74, 89). Similar to Chinese FHB resistant landraces, they all are inferior to Sumai 3 for various agronomic traits (23).

Two Brazilian cultivars, Frontana and Encruzilhada, were also reported to have FHB resistance and were used as parents in some breeding programs (23, 49, 74, 101). Frontana did not show resistance to spread of the fungus when a single floret was inoculated in greenhouse experiments (G. Bai, unpublished data), but it was reported to have low disease incidence in the field (100). In the United States, cultivars Ernie and Freedom were also reported to have a low disease incidence and severity in the field, and have been used as parents in some U.S. breeding programs (89). More recently, several research groups, with support from the U.S. Wheat and Barley Scab Initiative, have reevaluated thousands of wheat accessions from the USDA germplasm collection, CIMMYT, and several other countries (http://www.scabusa.org/forum.html). Most highly resistant wheat accessions have Chinese resistant sources, mainly Sumai 3 or its derivatives, in their pedigrees. Only a few accessions appear to have different sources of resistance, with no known relationship with Chinese sources, such as Chokwang from Korea (94) and Fundulea 201R from Romania (99). Molecular mapping of QTL from Fundulea 201R further indicates that it does not contain the same major QTL as that from Sumai 3.

Various wheat alien species have been screened to identify FHB resistance genes. About 6000 accessions of alien species were tested in China in the early

1980s, but none showed high FHB resistance (38, 122). More recent work has confirmed that diploid and tetraploid wheat species are highly susceptible (118). *Aegilops squarrosa*, a diploid species, was reported to be the source of resistance to FHB when it was used as a D genome donor in synthetic wheat (52). *Roegneria ciliaris*, *R. kamoji*, and *Elymus giganteus* (*Leymus racemosus*) have FHB resistance (67, 119). The FHB resistance in *E. giganteus* was associated with three chromosomes (36). In another study, four other *Elymus* species from Japan were evaluated and some of them had resistance similar to that of Sumai 3 (20). However, the resistance found in alien species is usually associated with undesired spike types, a trait that is difficult to remove from the progenies of these wide crosses. In addition, these materials usually do not surpass the resistance available in *Triticum aestivum*. Utilization of genes from alien species may require significant effort and time for pre-breeding to remove these "wild" characters.

So far, few sources of FHB resistance have been found in barley and the level of their resistance is modest. Although FHB in barley usually does not spread from spikelet to spikelet within a spike, barley seems to be very susceptible to initial infection. Severe disease usually results from multiple initial infections in the spike. In the United States, extensive FHB screening programs were established in barley in the upper-midwestern states in the 1990s to evaluate barley germplasm for FHB resistance and low DON content (107). Through collaboration with workers in China, several resistant cultivars with low DON content were identified, including CI 4196, Zhedar 2, Svanhals, and Imperial. CI 4196 is one of the best sources of FHB resistance identified to date. All these resistant cultivars are two-rowed barley. Six-rowed types are preferred for malting, but they are generally more susceptible to FHB than are two-rowed barley. Chevron, an old cultivar from Switzerland, is a six-rowed malting barley and a popular parent in barley breeding programs. It has high resistance to kernel discoloration, which is a disease complex caused by several different fungi including *Fusarium*. It is the best source of FHB-resistance yet identified from six-rowed barley. At North Dakota State University, over 8200 accessions of six-rowed barley were screened for FHB resistance in the field, and only 13 showed resistance similar to that of Chevron (108). These accessions were from Canada, China, Ethiopia, Romania, and the United States.

In Japan and China, over 10,000 barley accessions from different countries have been screened for FHB resistance, but only several dozen accessions had a low level of FHB (108, 113, 131). As with results from the United States and Europe, two-rowed lines were more resistant than were six-rowed lines, and hulled types were more resistant than were hull-less types. Although these resistant accessions had less severe FHB, they were still infected by *Fusarium* (<20% infection) under favorable weather conditions and accumulated DON at concentions greater than 0.5 ppm (61). To date, no wild species of *Hordeum* have shown greater resistance than that of two-rowed barley (108). DON content in even the best sources of resistance are still well above the specification for the brewing industry (<0.5 ppm), but much lower than that of current commercial malting barley cultivars (108).

STABILITY OF HOST RESISTANCE

Resistant wheat cultivars show consistent resistance to almost all isolates of *F. graminearum* worldwide. For example, resistant cultivar Sumai 3 was released in the 1970s in China. It has been used in Chinese breeding programs where FHB epidemics frequently occur. Since its release, 30 years ago, Sumai 3 and its derivatives are still the best resistance source in China (9, 69). These sources of resistance are the best available for wheat-breeding programs in many other countries. In the CIMMYT program, Sumai 3 and its derivatives, such as the Shanghai and Wuhan series, have been the major source of resistance for wheat FHB for over 20 years. These resistance materials have also been extensively tested for FHB resistance in Japan, United States, and many European counties with a worldwide collection of isolates of *F. graminearum* (7, 9, 23, 59, 75). Failure of resistance in Sumai 3 source has not been reported, and it is still the best source worldwide for resistance to spread of symptoms in the spike.

Although there is significant interaction between wheat cultivars and isolates of the pathogen, there is no evidence for stable pathogen races (14, 75, 104, 120), such as are found in cereal rust fungi, powdery mildew fungi, and some other specialized pathogens. Burgess et al. (31) placed isolates of *Gibberella zeae* (*F. graminearum*) into two groups based on whether they were heterothallic or homothallic. Subsequently, Aoki & O'Donnell (4, 5) transferred group 1 to a new species, *Gibberella coronicola* Aoki & O'Donnell. Based on the test of cultivar resistance to different species of *Fusarium*, Mesterhazy (72) concluded that resistance to certain strains of *F. graminearum* as well as to other species of *Fusarium* was not strain- or species-specific in wheat cultivars. The species of *Fusarium* that cause head blight can infect many other cereals and maize without showing specialization for any one host, and a host-specific, blight-causing *Fusarium* species has not been documented to date (114). Therefore, the resistance genes in Sumai 3 and other sources currently used in breeding programs are not expected to succumb to new races in the near future.

INHERITANCE

Inheritance of type II resistance in wheat has been extensively studied (15, 16, 18, 23, 27, 68, 81). Classic genetic research indicates that a few major genes (QTL) accompanied by some minor genes control type II resistance with a relatively high heritability (13, 16, 66, 81, 115). Additive gene effects play a major role but nonadditive gene effects might also be significant in most cases (15, 16, 102, 103). Dominance appears to be the most important nonadditive component (18, 102), although epistatic effects were also detected in some studies (15).

Estimates of the number and location of FHB resistance genes in wheat and barley vary with resistant lines studied, research methods, and experimental conditions. Analyses of monosomic or chromosome substitution indicate that resistance

genes from different Chinese and Japanese wheat cultivars are distributed over the entire wheat genome except for chromosome 1A (69). The estimated number of chromosomes for FHB resistance is one for wheat cultivar YGFZ (127), two for Wanning 2 (127) and WZHHS (65), four for Sumai 3 (125), and five for Wangshuibai (66) and PHJZM (127). Classic genetics research identified two resistance genes in Frontana (115), WZHHS, Sumai 3, and Ning 7840 (18), and three genes in WSB and YGFZ (18). Different numbers of genes for the same resistant cultivars have been proposed in different studies (69). Kolb et al. (59) pointed out several possible reasons for inconsistent results from different investigations including polygenic control of FHB resistance in wheat, effect of different genetic backgrounds, different types of resistance evaluated, genotype and environment interactions, heterogeneous sources of a resistant parent, or inoculation techniques used in different studies. Molecular marker technology may be able to provide more precise information on the number and location of QTL for FHB resistance.

MOLECULAR MAPPING OF QTL FOR FHB RESISTANCE

Assessment of resistance to FHB must be done when the main culm of a plant reaches the anthesis stage. This often prevents making crosses with a tested plant until its progeny can be grown. Environment significantly affects phenotypic assessment of the disease. Molecular markers may resolve the problems associated with phenotypic evaluation of the disease and make it possible to combine resistance genes from different sources through marker-assisted selection.

QTL Mapping in Wheat

Several types of markers have been used to identify QTL for FHB resistance. Random amplified polymophic DNA (RAPD) markers were used to identify two QTL from resistant cultivar Ning 7840 (7) and two QTL from a moderately resistant cultivar Fukuhokomugi (20). Using restriction fragment length polymorphism (RFLP) markers, two resistance QTL from Sumai 3 and two from Stoa were detected in a population of RILs derived from Sumai 3/Stao (117). A QTL on 3BS from Sumai 3 and another on 2AL from Stoa had a major effect on FHB resistance that explained 15.4% and 14.3% of phenotypic variation, respectively. A QTL on 2AL (Stoa) and on 6BS (Sumai 3) were minor; each explained less than 8% of phenotypic variation.

Simple sequence repeats (SSR) and amplified fragment length polymorphism (AFLP) are now used more frequently in QTL mapping of FHB resistance, due to their stability (relative to RAPD) and simplicity (relative to RFLP). Bai et al. (12) identified 11 AFLP markers tightly linked to a major QTL for FHB resistance in a population of RILs derived from Ning 7840/Clark. This population was evaluated for type II resistance in five greenhouse experiments. One major QTL explained up to 53% of phenotypic variation. This QTL was also associated with low DON accumulation in infected kernels (8). This major QTL is located on 3BS (129).

One of AFLP markers for the QTL was converted into a sequence tag site marker for marker-assisted selection (54).

More than 1000 SSR primers for wheat have been released so far (http://wheat.pw.usda.gov/ggpages/whatsnew/2003.shtml). The *Gwm* primers (88) and *Barc* primers (http://www.graingenes.org/cgi-bin/ace/query/graingenes?query=find+probe+barc*+AND+pcr_primers) have been extensively used for mapping FHB resistance QTL from Sumai 3 and other sources. Gilbert et al. (50) used SSRs to analyze the F_1 pentaploid from a cross of Sumai 3/DT 486 (a susceptible tetraploid), and concluded that there were no resistance genes in Sumai 3 on the D genome. Anderson et al. (2) re-analyzed the RILs from Sumai 3/Stoa (117) with SSR markers and reported that the 3BS major QTL explained 41.6% phenotypic variation. Two new QTL were detected, on 6AS and 3AL, in the RIL population derived from the cross between Butte 66 and ND2603 (a resistant line from North Dakota derived from Sumai 3). Zhou et al. (129) confirmed that the major QTL for FHB resistance in Ning 7840 (a derivative of Sumai 3) is the same 3BS QTL found in Sumai 3 and further pointed out that two modifying QTL on 2BL and 2AS can enhance the resistance conferred by the major QTL on 3BS. In another study, Zhou et al. (129) evaluated Sumai 3 substitution lines for type II resistance and DON content and found that QTL on chromosomes 7A, 3B, 2B, and 6B from Sumai 3 significantly reduce FHB symptoms. QTL on chromosomes 7A and 3B reduce both head blight severity FHB and DON accumulation in infected kernels. QTL on chromosomes 1B, 2D, and 4D appeared to increase DON accumulation. Buerstmayr et al. (29, 30) used SSR and AFLP markers to map QTL for type I and type II FHB resistance in a DH population derived from CM-82036/Remus and confirmed the major effect of the 3BS QTL on FHB resistance in this population. This QTL explained 60% of phenotypic variation in single floret inoculation, and 29% of phenotypic variation when heads were inoculated by spraying them with a spore suspension (a method for assessing type I resistance). Two additional QTL were identified, on chromosomes 5A and 1B. Based on their results from experiments using different inoculation methods, they concluded that a QTL on 3BS was mainly responsible for FHB restriction of symptom spread within a spike and another QTL on 5A was mainly responsible for resistance to initial infection (30). The QTL on 3B and 5A were further validated in five different breeding populations with CM-82036 as a resistant parent (3).

Bourdoncle & Ohm (25) mapped QTL for type II resistance in Huapei 57-2, which is an FHB-resistant wheat cultivar from China, and detected a major QTL on 3BS at the same location as the QTL in Sumai 3. Somers et al. (106) screened 328 polymorphic SSR loci in a population derived from Wuhan-1/Maringa. Wuhan-1 is a Sumai 3-derived, FHB-resistant wheat line selected in the wheat-breeding program of CIMMYT. Four QTL for FHB resistance were detected on 2DL, 3BS, and 4B, respectively, with 2 QTL on 3BS. QTL on 2DL and 3BS reduced disease severity by 32% after single-floret inoculation, and QTL on 3BS and 4B conferred a 27% decrease in FHB in the field. QTL on 3BS and 5AS significantly reduced DON accumulation in harvested grain.

Mapping of QTL from sources other than Sumai 3 has also been reported. SSR marker *Xgwm 2* on 3A is tightly linked to a QTL from *T. dicoccoides* (84). This marker explains 38% of phenotypic variation. Based on the fact that this QTL expressed in other genetic backgrounds but not in *T. dicoccoides*, a gene on chromosome 2A from wild Emmer line "Israel A" was proposed to suppress the FHB resistance of the 3A QTL (47). The same QTL was detected on 3A in a recombinant inbred population derived from cross Fundulea 201R/Peterson (99). Fundulea F201R is a Hungarian cultivar unrelated to Sumai 3. Another major QTL on 5A and two minor QTL on 1B and 3D were also reported. Four QTL together explained 32.7% of phenotypic variation for FHB resistance, which is smaller than that of the QTL in Sumai 3 and Ning 7840 (2, 12). Nine QTL explained 30% to 45% of total phenotypic variation in RILs derived from Renan (a resistant European winter wheat cultivar)/Recital (susceptible) (48). 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 a minor effect. QTL on 3A and 5A in cultivar Frontana explain 26% of type I resistance (109).

Wangshuibai is a Chinese landrace and Ning 894037 is a breeding line selected by somaclonal variation from Yangmai 3, a line selected from Funo. Sumai 3 does not appear in the pedigree of Wangshuibai or Ning 894037. However, a major QTL on 3BS in Ning 894037 explains 42.5% of phenotypic variation in resistance (98). Additional QTL on 2B and 6D have a minor effect. Although the banding pattern of marker alleles in Wangshuibai differs from those linked to the QTL in Sumai 3 (11), the 3BS major QTL was nevertheless detected in the same region from three populations: Wangshuibai/ND571 (53), Wangshuibai/Wheaton (130), and Wangshuibai/Alondra's (G. Bai, unpublished data). However, this QTL makes a smaller contribution to FHB resistance than the QTL on 3BS in Sumai 3, and significant epistatic interaction is detected between the QTL on 3BS and other QTL from Wangshuibai (53).

In summary, the major QTL on 3BS is found in most Chinese resistance sources and has the largest effect on type II resistance identified so far. Other sources of FHB resistance were also identified, but with smaller effect on FHB resistance than that of the 3BS QTL. The QTL on 5A is frequently detected in both Chinese and European resistant wheat cultivars. In addition, QTL on 2A, 3A, 6B and 7A may also contribute to FHB resistance in different genetic backgrounds.

QTL in Barley

Since there are few sources of FHB resistance in barley, only a few studies report molecular mapping of resistance QTL in barley. Molecular mapping of a population of RILs derived from the cross between the six-rowed barley cultivars Chevron and M69 identified that 10, 11, and 4 QTL were associated with FHB severity, kernel discoloration (KD) score, and low DON content, respectively (40). These QTL are distributed over all seven barley chromosomes. Three QTL on 2H and 7H for low DON are associated with low FHB severity. Several QTL for low KD

score mapped near QTL for low severity or DON. Two QTL on chromosome 2 (2H) and one on 6 (6H) might have a large effect on low KD and could be used for marker-assisted selection (33, 82). Near-isogenic lines with the low KD QTL on 2H had 40% less head blight than lines that lacked this QTL (82). In another study, Chevron did not show any QTL with major effect on FHB resistance, but markers linked to QTL on chromosomes 1, 2, and 4 were proposed for marker-assisted selection (70).

In the two-rowed cultivar, a QTL from CMB 643 (a parent with moderate FHB resistance), on chromosome 2, explains 33% of type II resistance (132). Two QTL from Gobernadora (an FHB-resistant parent), on chromosome 4 and 5, each explain 7% and 10% of phenotypic variation, respectively. QTL for type I resistance are on chromosome 1, 3, and 4. The QTL on chromosome 3 from CMB 643 appears to be more stable and has a larger effect on FHB. It explains 3% to 16% of phenotypic variation in different experiments. The other two QTL are most probably from Gobernadora and explain 4% to 10% of phenotypic variation in different locations. QTL for low DON were detected on five chromosomes [1, 2, 3, 4, and 5], but only the QTL on chromosome 4 was detected in more than one experiment. This QTL explained 7% and 13% of phenotypic variation in two experiments. The other QTL were detected only at one location and r^2 values were low (4% to 8%). The QTL for low DON on chromosomes 2 and 4 were at the same position as QTL for type II resistance, suggesting that the same QTL may control both traits.

In all these studies, most of FHB resistance QTL have been mapped to the same locations as QTL for other traits such as heading date, plant height, lateral floret size, spike angle, and kernel plumpness, etc. It is unknown if these QTL for these various traits are linked or whether the same QTL show pleiotropic effects that are due to close linkage or pleiotropy.

BREEDING FOR FHB RESISTANCE

Conventional Breeding

Though progress has been made in breeding for resistance to FHB during the past three decades, breeding commercial wheat cultivars that combine desired agronomic traits and a high level of FHB resistance remains a big challenge. This is because of the polygenic control of disease resistance, the association of so many undesired agronomic traits in the available FHB resistance sources, the complicated disease evaluation procedures, and the effect of environment on resistance phenotype. Moreover, resistance is not a single trait. It involves resistance to primary infection of the spike (type I resistance), subsequent spread of the pathogen within the spike (type II resistance), to kernel invasion, and to DON accumulation. It is not yet known to what extent these traits are under the control of common QTL. Most resistance breeding effort has focused on three aspects: improving agronomic traits of highly resistant materials available in wheat, improving resistance level of

currently grown commercial cultivars, and introducing new resistance genes from other gene pools.

In China, breeding wheat lines for a high level of FHB resistance started in the early 1960s. Selection of plants with superior resistance from commercially grown cultivars led to several moderately resistant cultivars, including Wanning 2, Wumai 1, Yangmai 1, and Yangmai 2 (9, 69). These cultivars provided some protection during moderate epidemics and were widely grown for many years in a large wheat production area along the Yantze River Valley. Since its development in the 1970s, the resistant cultivar Sumai 3 has been extensively used as a resistant parent in Chinese breeding programs nationwide. In the 1980s, many Sumai 3-derived resistant lines were developed through cross breeding. These resistant lines usually had stable type II resistance, similar to that of Sumai 3, and agronomic traits superior to those of Sumai 3, although they were still not acceptable for commercial production. These lines include the Ning series, Fu 5125, Fu 5114, and Fan 60096, and they played a significant role in the improvement of resistance to FHB in many breeding programs. Among them, Ning 7840 (Avrora/Anhui11//Sumai 3) has been a popular parent for FHB improvement. Its resistance is as good as that of Sumai 3, but it has better yield potential and resistance to leaf rust, stripe rust, stem rust, and powdery mildew (120, 128). It is a good parent for integration of resistance to multiple diseases. Many breeding lines derived from Ning 7840 possess moderate resistance to FHB and higher yield potential, shorter stature, higher test weight, and better processing quality than Sumai 3 (9).

Chinese wheat cultivars resistant to FHB have been widely used as parents in breeding programs in the United States and elsewhere. For example, ND2603 (Sumai 3/Wheaton), developed at North Dakota State University, has a high level of resistance (2) and is used as a resistant parent in breeding programs in the Upper Midwest. Wuhan 3 is a breeding line with FHB resistance derived from Sumai 3. It has been extensively used as a resistant parent in CIMMYT and several US wheat-breeding programs (101). In addition, several moderately resistant cultivars have been developed through the CIMMYT-China shuttle-breeding program and used in commercial production in regions of China where FHB is a threat (101). In Europe, breeding lines CM82038 and Sgv-NB/MM-Sumai 3, with a high level of FHB resistance, were also developed from Sumai 3 (29, 75).

Recently, many U.S. wheat-breeding programs have made FHB resistance one of their major objectives. Resistance genes from Sumai 3 have been transferred into elite breeding lines. From 1995 to 2002, about 60% of advanced resistant lines tested in the U.S. Uniform Regional Scab Nursery had Sumai 3 in their pedigrees (46). This nursery includes the best FHB resistance lines from major U.S. FHB breeding programs. Although these lines are still not suitable for large-scale commercial production, some are good parents for further cultivar development.

Although Sumai 3 has been the major source of resistance used in wheat breeding programs worldwide, other sources of resistance have also drawn breeders' attention. In the United States, winter wheat cultivars Ernie and Freedom

show good FHB resistance in the field and have been used as parents in several wheat-breeding programs (89). Molecular marker analysis indicates they do not have the 3BS QTL for FHB resistance (11). More recently, cultivar Truman was developed at the University of Missouri. It has better FHB resistance and overall performance on other traits than Ernie or Freedom (http://www.scabusa.org/pdfs_dbupload/08-04_McKendry_Truman-Release.pdf).

In Japan, Shinchunaga is an old cultivar selected from a natural mutation of landrace Nakanaga (22). It has been extensively used as a resistant parent in FHB breeding programs in Japan (22). Moderately resistant cultivars Saikai 165, Tokai 63, Tokai 66, and several Norin series cultivars were all derived from Shinchunaga. To improve the level of FHB resistance, another highly FHB-resistant landrace, Nobeokabouzu komugi, has recently been used as a parent in Japanese breeding programs (22). Some Japanese resistance sources have also been used in U.S. breeding programs (89). Bacup was a FHB resistant cultivar developed by scientists at USDA-ARS and the University of Minnesota for commercial production in years of severe FHB epidemics to replace highly susceptible cultivars. FHB resistance of this cultivar was derived from Nuy Bay, another Japanese FHB-resistant landrace. Bacup has good resistance with slightly improved agronomic traits compared to its resistant parent and can reduce yield losses in severe FHB epidemics. However, yield potential is significantly lower than other commercial cultivars, which limits its value. Nobeokabouzu komugi has also been used as a resistant parent in Europe and by CIMMYT (28, 74, 75). Although several Japanese landraces demonstrated a high level of resistance to FHB, their agronomic traits are worse than those of Sumai 3 (22). Sumai 3 is still the major source of FHB resistance in these breeding programs. Whether resistance QTL from Japanese sources are different from Chinese sources remains to be determined.

Breeding Using New Technology

Besides conventional plant breeding, various other breeding strategies have been used to improve resistance in wheat to FHB (9, 69). In China several FHB resistant lines were derived from susceptible commercial cultivars by exploitation of somaclonal variation (69). One of these, Shenka 1, was grown commercially over a large area for five years. It yields as well as the widely grown Yangmai 5 and had better resistance to FHB. Two lines, also developed from somaclonal variation, Ning 894013 and Ning 894037, have FHB resistance similar to Sumai 3, but perform better agronomically. Molecular mapping indicates that Ning 894037 carries the same major QTL as that in Sumai 3 (98).

To combine quantitative resistance and superior agronomic characters, a modified recurrent selection method has been used to maintain genetic diversity and break unfavorable linkages (9). With the aid of a dominant male-sterile gene, *Ta*1, several gene pools have been developed by first crossing several parents and then practicing phenotypic recurrent selection. After eight cycles of selection, several resistant lines were released. Some, such as W14, have a high level of resistance and

some improved agronomic traits. Others have high yield potential and improved FHB resistance, such as several lines in the CJ series.

Several techniques have been used to introduce alien resistance genes into wheat, such as including synthesizing new hexaploid wheat through combining durum wheat (AABB) with *Triticum taushii* (DD), or introducing single genes, alien chromosomes, or DNA fragments from wild relatives into wheat. By combining genomes of durum wheat (AABB) and *T. tauschii* (DD) and using the doubled haploid technique, several synthetic wheat lines with some resistance to FHB were developed at CIMMYT (52, 89). Because these synthetic hexaploids contained the entire D genome from *T. taushii*, they had many undesirable traits from the *T. tauschii*. They were tall, difficult to thresh, and had low yield. It can be difficult to correct these deficiencies through conventional breeding (89).

To avoid introduction of the entire genome from a wild species, individual chromosomes can be introduced into wheat. Some wheat addition lines with chromosomes from *Roegneria ciliaris*, *R. kamoji*, or *Elymus giganteus* have been produced in China (36). These alien addition wheat lines were more resistant than their wheat parents. Several resistant lines selected from the crosses between *E. giganteus* addition lines and Sumai 3 or Xiangmai 1 showed good resistance and better agronomic traits than Sumai 3. The disadvantage of this technique is that introduction of a single alien chromosome still may introduce many undesired agronomic traits. The materials created from wide crosses require much work over a long period to remove unwanted alien genes. Direct transfer of a target gene through a transgenic approach might circumvent this problem.

With transgenic techniques, a gene can be transferred into wheat from any organism. This ability significantly broadens the number and diversity of genes that can be used for enhancing FHB resistance in wheat. The U.S. Wheat and Barley Scab Initiative has supported several university research groups in the United States to work on transfer of FHB resistance into wheat and barley cultivars. Progress has been made in optimizing transformation technologies and in production of transgenic plants (80). Several genes for antifungal proteins (AFP) or with a DON-reducing function have been used as transgenes. Chen et al. (37) reported that transgenic wheat plants carrying a rice thaumatin-like protein gene exhibited delayed expression of FHB symptoms. To enhance FHB resistance by reducing DON accumulation, the gene (*TRI101*) for trichothecene acetylransferase from *Fusarium sporotrichioides* was successfully transferred into the wheat cultivar Bobwhite. One line had significantly less disease than its nontransformed parent in the greenhouse (83). So far, no transgenic plant surpasses the resistance level in resistant wheat cultivars already identified within *T. aestivum*. Our limited understanding of the basis of resistance to *F. graminearum* makes identification of candidate transgenes difficult. In addition, technical and social problems associated with transgenic plants limit the utility of this approach for development of wheat resistant to FHB (80).

SUMMARY

Control of FHB will require application of several different disease management strategies. Resistant cultivars should be a major part of an integrated approach to reducing the damage from FHB. Fungicides may be useful when weather or other cultural conditions are particularly favorable for disease development. At present, however, there are no highly effective fungicides. The Chinese wheat cultivar Sumai 3 and its derivatives are currently the best sources of resistance to FHB, and may provide the maximum degree of type II resistance. Wheat breeders throughout the world are using these cultivars as resistant parents. Many improved lines or cultivars with FHB resistance, mainly from Sumai 3, have been developed in breeding programs worldwide in the past two decades, but these lines either do not provide adequate protection to FHB in environments especially favorable for the disease, or they lack acceptable yield potential to meet the needs of commercial wheat production. Therefore, most cultivars in current production are still susceptible. Combining all the desired agronomic characters, plus resistance to other important diseases and insects, with a high degree of resistance to FHB is still a major challenge. To date it has been difficult to combine the high degree of type II resistance of Sumai 3 with these other necessary traits. A rapid, efficient, and accurate technique for identifying resistance in segregating materials needs to be developed so that large populations can be screened. Marker-assisted selection may provide such a technique for dissecting and stacking different resistant QTL for FHB resistance. The application of high-throughput markers for FHB resistance QTL may significantly improve selection efficiency. USDA Regional Genotyping Centers may provide the ideal facility for breeders to access this technology (116).

Fortunately, there is as yet no evidence of races of *F. graminearum* that specifically interact with the type II resistance of Sumai 3. However, if a cultivar with only type II resistance were exposed to prolonged conditions favorable for infection, it could sustain unacceptable damage. Type II resistance needs to be combined with type I resistance to provide effective control in the field. Moreover, among currently grown cultivars (none of which has a high degree of resistance), there is only a weak correlation between head blight intensity in the field, visible damage to kernels, or DON content in grain (97). It remains to be seen how well a high degree of resistance to head blight will protect grain quality in the field. It may be necessary for breeders to find genes that directly affect the ability of the fungus to grow in grain and produce DON.

Of the several types of resistance that have been reported or hypothesized (73), type II resistance is the most stable and well-studied. It is a quantitative trait controlled by one or a few major QTL and probably some minor QTL. The major QTL on chromosome 3BS is found in most resistant cultivars from China. Several QTL on other chromosomes have also been reported, but their expression is not stable over different environments or in all genetic backgrounds. Some of them may modify the expression of the 3BS QTL or may be responsible for different types of resistance, and therefore these QTL merit further investigation.

There are very few FHB resistance sources in barley. Only a few barley cultivars have a relatively higher level of FHB resistance. Most of these resistant cultivars are two-rowed barley. Within six-rowed barley, which is preferred for malting, cultivar Chevron has the best degree of resistance, but its DON level is still too high and far from meeting the safety requirements of the brewing industry. In contrast to wheat, type I resistance is the major resistance type in barley. Molecular mapping indicates that many QTL, spread over many chromosomes and with minor effects, control this resistance. One QTL on chromosome 2 seems to be stably expressed and has been suggested for application in marker-assisted selection (70, 82).

Several genes are induced during infection or reduce disease severity after transformation, but the biochemical and molecular mechanisms of FHB resistance are still poorly understood. Several recent developments in genomics and biotechnology hold promise for understanding the genetic mechanism of FHB resistance and for more effective development of resistant wheat and barley cultivars. Transformation technology has been successfully used to transfer antifungal protein genes into wheat and barley. But key FHB resistance genes from wheat and barley need to be identified. Functional genomics tools such as microarray analysis and ESTs open a new era for genome-wide gene expression profiling, which may lead to discovery of new resistance genes and understanding of resistance mechanisms. High-resolution mapping and molecular cloning of the 3BS QTL may provide useful genetic materials for further study of gene function.

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Annual Review of Phytopathology
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CONTENTS