HOST PLANT RESISTANCE AND VARIETY DEVELOPMENT

Chairperson: Paul Murphy

QUANTITATIVE TRAIT LOCI ASSOCIATED WITH REDUCED DEOXYNIVALENOL IN THE SOFT RED WINTER WHEAT 'ERNIE'

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OBJECTIVES

The objectives of this research were to (1) identify QTL associated with low DON in the Missouri Fusarium head blight (FHB) resistant cultivar, Ernie, and (2) determine whether or not they differed from those QTL identified for type II FHB resistance in this cultivar.

INTRODUCTION

Fusarium head blight (FHB), caused by *Fusarium graminearum* Schwabe [telomorph: *Gibberella zeae* Schw. (Petch)] reduces grain yield in wheat (*Triticum aestivum* L.) in many regions of the world. Losses in grain quality can also occur due to contamination with the mycotoxin deoxynivalenol (DON) produced in susceptible wheat varieties. Deoxynivalenol is linked to feed refusal in livestock (Meronuck and Xie, 2000) and causes depression of the immune system, nausea, and vomiting in humans (Prelusky et al., 1992). Genetic resistance is the most cost effective method to reduce quality losses associated with DON contamination in wheat. Breeders believe that selection of lines for low FHB may result in a corresponding reduction of DON in those lines. This association however, has not been well established.

Ernie, a soft red winter wheat developed and released by the University of Missouri, has a moderately high level of type II FHB resistance. In inoculated trials, it also has low DON. Four quantitative trait loci (QTL) associated with type II FHB resistance were identified on chromosomes 2B, 3B, 4B, and 5A using a population of recombinant inbred lines (RIL) derived from cross of Ernie/MO 94-317 (Liu et al., 2005). It is not known, however, whether selection for these QTL in populations derived from Ernie will also result in lower DON levels in resulting genotypes.

MATERIALS AND METHODS

A set of 243 F8 and F9 recombinant inbred lines (RILs) developed from the cross Ernie/MO 94-317 was used for QTL mapping. The experiment was arranged as a randomized complete block design with three replications and grown in the greenhouse in 2002 and 2003. Eight plants/RIL/replication were point inoculated with *F. graminearum*, harvested at maturity, and hand threshed to ensure all diseased kernels were collected. Seed within each replication of each RIL were bulked, ground and analyzed for DON content at Michigan State University. The concentration of DON was quantified using the mycotoxin extraction kit Veratoxin for DON 5/5 (Veratox®). Broad-sense heritability was determined from the analysis of variance as, $h2BS = [ó^2G/(ó^2G + ó^2GxY /Y + ó^2E/RY)]$, where $ó^2G$ is the genetic variance among RILs, $ó^2GxY$ is the variance due to genotype-by-year interaction, $ó^2E$ is the variance due to error, and R and Y are the number of replications, and years, respectively (Nyquist 1991). The minimum number of genes was estimated using Cocherham's (1983) modification of Wright's (1968) formula.

Polymorphisms between Ernie and MO 94-317 were assessed using 64 *Eco*RI/*Mse*I AFLP primer pairs and 420 SSR markers. Both AFLP and SSR polymorphic makers were used to construct the linkage map using Mapmaker, Version 3.0 (Lander et al., 1987) using the Kosambi mapping function. Markers were grouped with a LOD value of 3.0 and distance less than 37 cM. Composite interval mapping was used for the QTL analysis using WINQTLCART (Version 2.0). Permutation tests (Doerge and Churchill, 1996) were used

to determine critical thresholds for significance of each potential QTL at each location. Significance at á=0.05 was determined from the 950th of 1,000 permutations of the data.

RESULTS AND DISCUSSION

Deoxynivalenol data for 2002 and 2003 were continuously distributed but not normally distributed therefore data were log transformed and reanalyzed. Transformed data approximated a normal distribution. Using transformed data, Bartlett's test indicated that the error variances within each year were homogenous (P= 0.05) therefore, data were combined over years and reanalyzed. RILs were considered fixed while years and replications were considered random effects.

Results of the analysis of variance indicated that both genotypic effects (RILs) and genotypic X environment interactions were highly significant (P = 0.0001). Mean DON levels for Ernie (resistant) and MO 94-317 (susceptible) were 4.5 and 89.3 ppm, respectively. Among RILs mean DON levels ranged from 1.5 to 100 ppm.

Broad-sense heritability (68%) indicated that DON levels were genetically controlled and moderately heritable which suggested that lines with low DON levels could be developed in breeding programs. Five genes were determined to condition DON levels in this cross.

A threshold LOD value of 3.5 was used to declare significant QTL. Three QTL were detected which accounted for 32% of the phenotypic variation in DON levels in RILs of this cross. These QTL were located on chromosomes 3B, 4B, and 5A and accounted for 13.2, 6.9, and 11.6% of the phenotypic variation, respectively (Table 1). A fourth QTL on 2B was significant in 2002 (LOD=3.8) but was not significant in 2003 (LOD=1.0) and therefore was not significant (LOD=2.9) in the combined analysis. Based on the sign of additive values, all QTL originated from the resistant parent.

CONCLUSIONS

QTL regions on 3B, 4B, and 5A were consistently associated with low DON in two years of analysis of this trait in the cross Ernie x MO 94-317. All alleles were from the resistant parent Ernie. These QTL were identified in the same regions as those previously identified by Liu et al. (2005) for type II resistance in Ernie. Both the 4B and 5A markers were identical while that on 3B was closely linked to the 3B marker for type II resistance. These data suggest that in Ernie, DON level and type II FHB resistance are not independent, therefore, selection for type II resistance should result in low DON.

ACKNOWLEDGEMENTS

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Table 1. Quantitative trait loci (QTL) associated with reduced DON in recombinant inbred lines of the soft red winter wheat cross Ernie/MO 94-317. Lines were phenotyped for DON levels from experiments conducted in 2002 and 2003. All alleles were from Ernie.

Chromosome location	Marker	QTL peak position	LOD	R^2 (%)	Additive effect
2B	Xgwm276b	120.2	2.9	4.0	-0.088
3B	E8M4_6	123.7	5.3	13.2	-0.142
4B	Xgwm495	0.0	4.7	6.9	-0.102
5A	Xbarc056	44.7	5.0	11.6	-0.133

CIMMYT'S CHALLENGES FOR GLOBAL COMMUNICATION AND GERMPLASM ENHANCEMENT FOR FHB RESISTANCE IN DURUM AND BREAD WHEAT

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OBJECTIVES

CIMMYT's challenge is to identify and validate novel FHB resistance for durum/bread wheat and barley, pyramid the complementary sources of resistance, diversify the resistance gene pool currently utilized by the USWBSI community, and to facilitate the utilization of resistant germplasm through global communication.

INTRODUCTION

Many institutions around the world have devoted substantial resources to combat FHB, and have met a measure of success. However, the global community is facing the threat of imminent epidemics. Unless steps are taken to defeat the disease, this threat will materialize into a much greater problem and as such requires a global response. CIMMYT has adopted a holistic approach to enhance novel FHB resistance among genebank accessions and synthetic wheat derivatives and combine their resistance using systematic screening in multiple environments and genetic characterization by DNA markers. CIMMYT's role in the Global Fusarium Initiative is to provide a platform for international collaboration on Fusarium research, and facilitate information exchange, germplasm enhancement and the development of breeding methods and materials globally. This Global Fusarium Initiative would encourage communication and cooperation among individuals, institutions and governments focusing on this disease. Specific activities will be linked using a web site and on-site forums (http://www.fusarium-net.org). Global G x E meta-data compilation, updated global information, and a global crop information system on FHB data will be a feature of this web site. The Global Fusarium Initiative provides the platform to fight this grave threat which will require all our wisdom and expertise to overcome.

CIMMYT LAUNCHES A NEW CHALLENGE ON FHB RESISTANCE IMPROVEMENT

Evaluation of wheat germplasm for FHB resistance to detect useful genes for various cultivation environments - We aim to acquire potentially novel sources of resistance from global hotspots through our widespread contacts. This will allow us to identify globally stable resistance and will contribute to our understanding of the underlying mechanisms of resistance. Expectations are high that useful resistance genes may be identified during the screening of germplasm accessions and that the effects of Genotype x Environment x Management interactions and the distribution of Fusarium isolates will be better understood. Germplasm will be evaluated first at several FHB hot spot locations in Mexico, and then globally through CIMMYT's International Wheat Improvement Network (including CIMMYT-Turkey). This will ensure that materials are exposed to a range of Fusarium isolates. Validation of newly acquired resistant sources using DNA markers and haplotype evaluation will help identify new resistance genes.

A total of 7,197 spring bread & durum wheat and 2,703 barley entries were evaluated for FHB in the field under natural rainfall and supplementary sprinkler irrigation at CIMMYT's highland research station in Toluca in 2005. In addition, part of the bread wheat material was planted at Patzcuaro in Mexico to

obtain multi environment data. Our FHB shuttle breeding through the exchange of segregating populations with China and Uruguay was also resumed in 2005. A number of F3 segregating bulks have been sent to Wuhan and Nanjing in China for planting in November 2005. These populations combine various scab resistance sources. The same materials have been increased for Uruguay and will be sent in early 2006 for planting in May 2006. Materials selected from these bulks will be selected locally by CIMMYT and local scientists. In some cases, the local/national programs may select a different plant ideotype than CIMMYT scientists for resistance to FHB, levels of the mycotoxin DON, and end use quality attributes. After two or three cycles of selection locally under scab pressure, these materials will be sent to CIMMYT for further development and eventual distribution globally.

We aim to increase FHB resistance using 3 approaches: 1) Acquisition of novel resistance 2) Screening transgressive segregates combining different resistance genes, and 3) Evaluation of advanced, adapted materials with multiple-disease resistance. Figure 1 shows a potential strategy for introgression and pyramiding of genes to enrich FHB resistance in wheat breeding programs. Currently, the effort to combine FHB and Fusarium crown rot (FCR) resistance involves the use of molecular markers to combine the FHB resistance of Sumai 3 with the FCR resistance of a bred wheat line 2.49. A number of derivative materials positive for both markers are currently undergoing field evaluation to test the effectiveness of these combined resistances. In addition, a number of complementary sources of FHB resistance are being combined in crosses. These materials will enter the international shuttle breeding program linking China, Uruguay and Mexico.

CIMMYT efforts and perspective on germplasm enhancement FHB resistance in durum wheat - The most serious challenge in the development of FHB resistant durum wheat is that there is as yet no known effective durum source for resistance. Therefore, it is essential to find novel source of FHB resistance in durum and/or other tetraploid wheat. The resistance sources of these lines would be readily incorporated into elite durum lines in the course of breeding. CIMMYT has adopted a systematic search of the primary gene pool of durum wheat in CIMMYT gene bank containing the largest global collection of wheat and wheat relatives. We are also encouraging global communication to understand the genetic diversity of FHB resistance among primary durum wheat sources with their limit or potential. Establishment of a global platform to facilitate germplasm exchanges and introduction of highly resistant durum wheat germplasm from international programs via CIMMYT's international network will be required. Similarly, we will promote the collaboration with NDSU for diversifying the resistance tetraploid gene pool currently utilized by the USWBSI community to facilitate the utilization of resistant germplasm through the global communication.

An alternative way to diversify FHB resistance of durum wheat is the introduction of resistance factors or QTLs on Dgenome chromosomes. This approach may be more promising than seeking novel sources of resistance, especially if one prefers to avoid the possibility that there are no sources resistance in durum and wild tetraploid wheat because they have been not exposed to FHB in their history. There are several hexaploid and synthetic hexaploid derivatives developed from durum wheat /*Ae. tauschii* crosses which show high FHB resistance. The most resistant hexaploid wheat known and also studied at this moment is Sumai 3 which has the strong resistance QTL on chromosome arm 3BS. Trials to introgress FHB resistance from eight hexaploid wheats crossed with 14 elite durum wheat lines were begun at CIMMYT in the 2004 summer/fall cycle in El Batan. Eight F1 plants produced were top-crossed to six durum elite lines, which were resistant to leaf rust and had good to acceptable quality attributes in the 2005 Obregon nursery, and five of the crosses produced TC1F1 seed. Sixty-five plants of the TC1F1 generation were planted in El Batan and molecularly screened with genotype of DNA markers for the Sumai 3 FHB-QTL region. Twelve plants, representing three crosses, were identified harboring the Sumai 3 FHB-QTL, and back-crossed to the durum elite line used as the recurrent parent in the TC1F1. These lines will be promoted to improve agronomic traits, quality and rust disease resistance in the CIMMYT durum wheat breeding program. Elite candidates of them will be screened for FHB evaluation

after the TC5F1 or BC5F1 generations. Several synthetic wheat of the D genome (genome constitution=AABBDD) showed resistance equal to or higher than that of Sumai 3. These novel sources of resistances were most likely were derived from the D genome. Attempts have been initiated to induce homeologous recombinations between the D genome with A or B genomes using the *ph1* pairing homolog mutant (Figure 2 and 3).

There are two logical reasons for the extreme susceptibility of durum wheat to FHB. One is that durum wheat is inherently lacking in genes conferring resistance. The other is that durum wheat has strong susceptibility factors for FHB (Ban and Watanabe, 2001). Besides systematical screening primary source of durum wheat for resistance, we propose two research strategies for FHB resistance improvement of durum to avoid the risk of only confirming the lack of resistance in the primary gene pool. The first strategy is to find and utilize resistance source in alternative sources in CIMMYT's genebank accessions including durum and other cultivated tetraploid wheats, wild relatives and ancestral species. We have observed that the Type II resistance of *T. monococcum* ranges from 9.4% to 45.7%, and is higher than that of durum wheat. CIMMYT has produced more than 200 lines of synthetic wheat of the A and B genomes (genome constitution=AAAABB and AABBBB), and there are several resistance candidate where the Type II resistance scores are as minimal as 9.5%.

The second strategy is to find and remove of the strong risk factors in durum wheat. The identification of susceptibility factors is important as identification a source of resistance. It is especially true in the case of durum wheat which is quite susceptible to FHB. When the durum-shaped plants in the progenies of Sumai 3/ durum wheat lines are selected, much of the susceptible ideotypes are transmited as well. It may be difficult to produce resistant varieties, even by adding resistance factors, without first eliminating the susceptibility factors. We are making wide crosses to identify FHB risk factors in durum with cytogenetic markers to enhance transgressive segregation for FHB resistance in durum wheat.

Development of Global Fusarium Initiative for collaborative research and FHB holistic operation in CIMMYT – CIMMYT has been conducting a holistic operation to enhance FHB resistance in wheat germplasm through systematic screening in multiple environments. Novel genetic variation is found among CIMMYT's genebank accessions and synthetic wheat derivatives. The FHB research in wheat has been systemized in a simple workflow on four levels: evaluation of resistance in the field (phenotyping); genetic characterization by DNA markers (genotyping); gene discovery; and development of DNA marker assisted selection (MAS) for use in breeding.

FHB is a grave threat that requires an integrated research approach to overcome. CIMMYT intends to develop a global platform for international collaboration on FHB, thereby acting as a facilitator for global information exchange and germplasm enhancement and distribution. We recognize the need to enhance international relationships as a part of each National/International Project/Consortium. CIMMYT will take a more proactive stance to elevate the work of FHB resistance breeding and to raise the profile of this global challenge. New projects recently initiated at CIMMYT could be combined with research efforts elsewhere to focus and organize a worldwide effort to combat the disease.

For this reason, we have developed a Global Fusarium Initiative at CIMMYT which has been supported by the Japanese government since 2004. The challenges and specific activities are based on the new paradigm which arose from the JIRCAS Workshop held in February 2004 in Tsukuba (Ban, 2004). The concept of a Global Fusarium Initiative was proposed and accepted at the 2nd International Symposium on Fusarium Head Blight, incorporating the 8th European Fusarium Seminar, 11-15 December 2004, Florida USA (Van Ginkel and Ban, 2004). A new global collaboration for consensus QTL mapping of FHB resistance in wheat, involving the world's most advanced FHB researchers, will be one of the activities. *Fusarium* fungi, the pathogen of FHB, also causes Fusarium crown rot (FCR) in Australia, Turkey and other places, and is another constraint to global wheat production. We have inte-

grated research and germplasm enhancement for both FHB and FCR under the Global Fusarium Initiative. This initiative will encourage communication and co-operation among individuals, institutions and governments focusing on this disease. We have developed a Global Fusarium Initiative web site with on-site forums (http://www.fusarium-net.org) to facilitate and coordinate our activities in this fight against the dangers of FHB.

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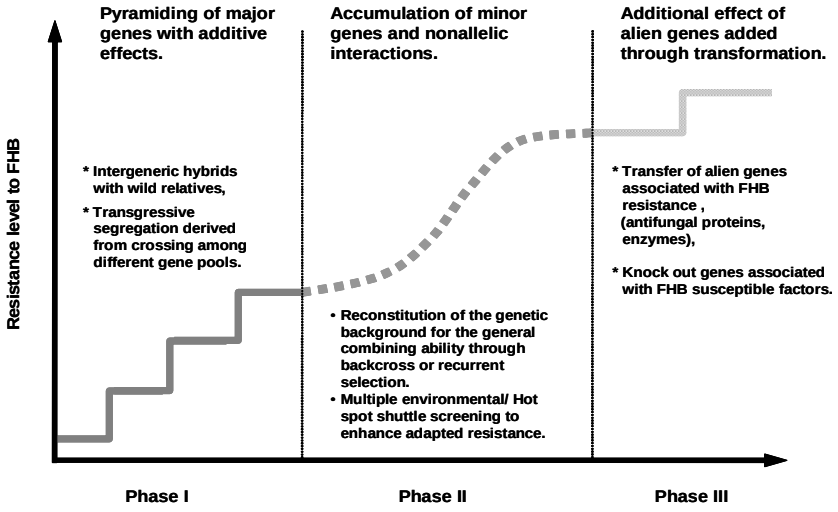


Figure 1. Potential strategy for introgression and pyramiding of genes to enhance FHB resistance in wheat breeding programs.

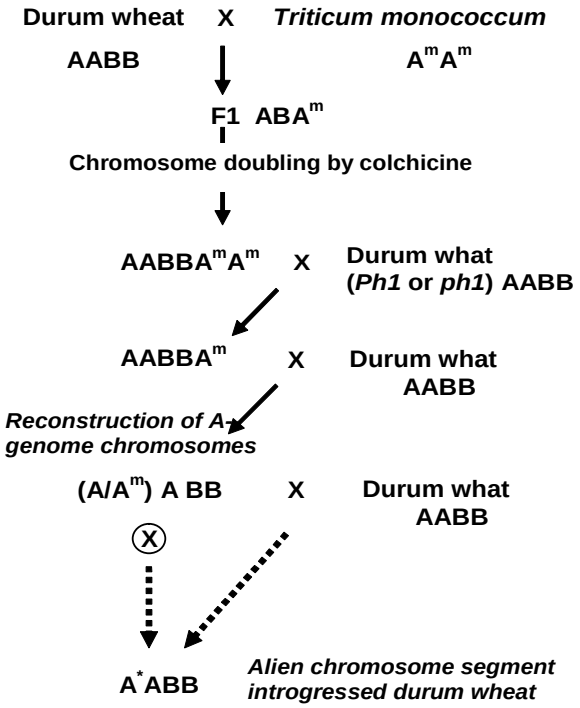
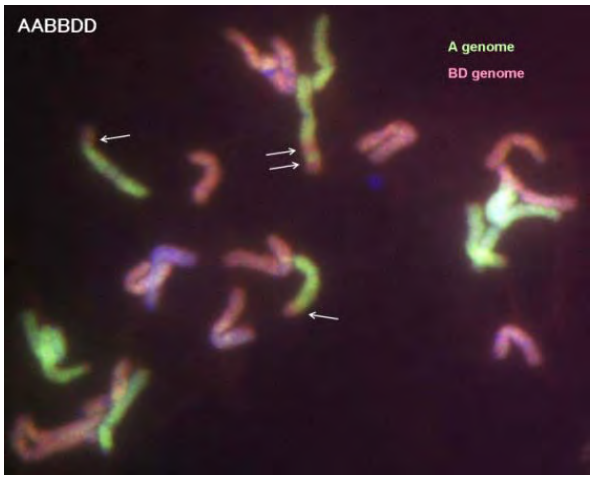


Figure 2. Utilization of A genome ancestor for reconstruction of durum wheat A, B genomes toward FHB improvement.



Superior resistance of FHB can be expected in the D genome of Ae. tauschii. Several synthetic wheat of D genome (durum x Ae. tauschii) in CIMMYT showed resistance equal or higher than that of Sumai 3 (Mujeeb-Kazi and Delgado, 2002). These resistances supposedly reside in D genome and can be transferred into durum by the use of ph1 mutant system.

Figure 3. Translocation of the B and D genome chromosomes into the A genome. The arrows indicate translocation of B or D genome into A genome.

GLOBAL COLLABORATION OF GENETIC STUDIES AND BREEDING FOR FUSARIUM HEAD BLIGHT RESISTANCE IN WHEAT

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ABSTRACT

Fusarium head blight (FHB) is a grave threat that we must integrate all our wisdom and expertise to overcome. CIMMYT's role in the **Global Fusarium Initiative** is to provide a platform for international collaboration on *Fusarium* research, and facilitate information exchange, germplasm enhancement and the development of breeding methods and materials globally. This **Global Fusarium Initiative** will encourage communication and cooperation among individuals, institutions and governments focusing on this disease.

The challenges of this initiative are:

- Identification of new sources of resistant germplasm and pre-breeding.
- Delineation of the nature of wheat resistance to FHB and host-pathogen interaction.
- Development of effective cropping systems adjusting pathogen cycle and wheat growth.
- Germplasm sharing and intellectual property rights (IPRs) management.
- Knowledge sharing among the global community .

The specific activities of this initiative are:

- Linking with relevant *Fusarium* initiatives.
- Website and e-News, http://www.fusarium-net.org.
- Global compilation of Genotype x Environment x Management meta-data through new international interactive screening nursery system.
- Up to date global information on FHB epidemics, toxins and resistant breeding.
- Biennial meetings for information sharing and focused discussion.
- Global Crop Information System on FHB.

We aim to acquire potentially novel sources of resistance from global hotspots through our wide-spread contacts. Expectations are high that useful resistance genes may be identified during the screening of germplasm accessions and that the effects of Genotype x Environment x Management interactions and the distribution of *Fusarium* isolates will be better understood. In addition, we are working to develop a compilation/monitoring system for *Fusarium* genetic diversity, pathogenicity, and toxigenicity to further our abilities to control FHB.

IDENTIFYING MARKER-TRAIT ASSOCIATIONS FOR FUSARIUM HEAD BLIGHT USING BREEDING GERMPLASM

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OBJECTIVE

To test the utility of a variance component approach to association mapping in breeding germplasm to identify new and validate existing QTL for Fusarium head blight in barley.

INTRODUCTION

Breeding and genetic research are generally conducted as two separate enterprises. Under the best circumstances they are done in parallel and progress in one informs the other. Integrating gene discovery with breeding has several advantages including using: germplasm most relevant to breeding, large populations over multiple years and environments, and large amounts of breeding data that is not typically used for genetic studies. However, the conflicting procedures used for these two enterprises makes their coordination a challenge. Genetic studies rely on the intensive study of a single carefully chosen population, while breeding research generally allocates resources to study large numbers of progeny from many different populations with limited replication. Association mapping is a general statistical approach to identify genes and marker-trait associations by measuring linkage disequilibrium in populations with complex structure. Genetic studies in animal systems have utilized statistical methods in this area because they are unable to generate large segregating populations as is possible in plant systems (George et al., 2000). Recently, research in plant genetics have begun to use some of these techniques to increase the power of genetic studies (Jannink, 2001).

The USWBSI has provided resources to establish large multi-location Fusarium head blight (FHB) screening nurseries to generate large volumes of FHB disease data. In this study, we use selective genotyping, as described by Wingbermuehle et al. (2004), to identify QTL for FHB using individual breeding populations referred to as Fusarium early generation (FEG) populations. We then use the association mapping approach of George et al. (2000) to identify marker alleles linked to FHB resistance across multiple FEG populations.

MATERIALS AND METHODS

Field evaluations of the FEG lines - The 2004 FEG populations were evaluated at Crookston and St. Paul, MN. The experimental design at each environment was a randomized complete block design with 2 replications. Entries were planted in 1.8 m long single row plots, spaced 30 cm apart. At St. Paul, a macroconidia inoculation technique was used whereas at Crookston a grain-spawn inoculation technique was used (Mesfin et al., 2003). Nurseries were mist irrigated daily to enhance disease. Entries were scored for percent FHB severity by examining 20 random spikes from each plot at Crookston and 10 random spikes from each plot at St. Paul. The number of infected spikelets from each spike were counted and expressed as a percent of the total spikelets present. Heading date was scored as the number of days after planting to 50% emergence from the boot.

Selective genotyping - FEG populations with clear segregation in FHB severity were chosen for further study. The phenotypic extremes for FHB severity, which we refer to as the resistant and susceptible tails of the population, were selected for genotyping analysis. The 12-23 % of the lines with the lowest FHB severity were chosen for the resistant tail. The 11-12% of the lines with the highest FHB severity were chosen for the susceptible tail.

FEG tails t-tests - Forty-eight SSR markers (Beaubien and Smith, 2004; Ramsay et al., 2000; Thiel et al., 2003) were screened on the parents of the chosen FEG populations to test for polymorphisms. For each polymorphic marker in a population, a two-tailed *t*-test comparing the marker allele distribution between the resistant (p_{Res}) and susceptible (p_{Susc}) tails was conducted by calculating:

$$t = \frac{p_{Res} - p_{Susc}}{[p(1-p)/2n_{Res} + p(1-p)/2n_{Susc}]^{1/2}},$$

where n_{Res} is the number of individuals in the resistant tail and n_{Susc} is the number of individuals in the susceptible tail (Bernardo, 2002, p. 294). We used a significance level of $p<0.05$ for our threshold of detection (Snedecor and Cochran, 1980).

Association mapping and Linkage disequilibrium - Associations were detected using a two-stepped variance approach as proposed by George et al. (2000). Genetic variance was estimated using a BLUP approach (Bernardo, 2002, p. 235) (Proc IML, SAS, 2003). Marker-trait associations were detected using a mixed model that accounted for population structure using a coancestry matrix developed from pedigree data. We considered weak associations those with an observed *p*-value between 0.01 and 0.001 and strong associations those with an observed *p*-value of 0.001 or less. Linkage disequilibrium (LD) was calculated as multi-allelic r between markers on the same chromosome (PowerMarker, Liu and Muse, 2005).

RESULTS AND DISCUSSION

Six FEG populations derived from parents representing six sources of FHB resistance (Table 1) were chosen for selective genotyping. Each of these populations showed significant FHB severity segregation as is clear from the population standard deviation and in the difference in mean between the resistant and susceptible tail (Table 1). In some populations both parents had resistance to FHB, but from different resistant sources.

Of the 39 markers that exhibited polymorphism on at least one set of FEG parents, 22 have been evaluated thus far (Table 2). Preliminary LD analysis shows that at distances less than 20 cM, LD is high but it dropped off considerably at distances greater than 20 cM (Figure 1). This indicates that complete genome coverage with gaps less than 20 cM, should be sufficient to detect most marker-trait associations.

Two markers were associated with FHB severity (Table 2). HVM040 [chr. 4(4H)] had a weak association with FHB severity (*p*-value=0.0014, Table 2). HVM040 was also significant based on selective genotyping *t*-tests in three of the four FEG populations for which it was polymorphic (Table 2.). For each of the significant FEG populations, the A allele confers lower FHB severity (data not shown). This allele is traced back to AC Oxbow in the FEG 103 and FEG 112 populations and Atahualpa in the FEG 107 population. HVM054 [chr. 2(2H)] had a strong association with FHB severity (*p*-value=0.0003, Table 2). HVM054 was also significant based on selective genotyping *t*-tests in in all three FEG populations for which it was polymorphic (Table 2), and was associated with a minor FHB QTL in Mesfin et al. (2003). For each of the significant FEG populations, the B allele confers lower FHB severity (data not shown). This allele is traced back to Zhedar1 in the FEG 104 population. It is less clear where the allele originates from in the other FEG populations because the B allele does not match their resistant parent allele source. One possible explanation is that it is expressing the Chevron allele, which was prevalent in our breeding lines even before the FHB program because it is in the pedigree of Peatland. None of the markers were associated with heading date.

In this preliminary study, we were able to detect two FHB marker-trait associations with a minimal set of markers using phenotypic data from breeding trials. We believe that the power of the study will improve as both the number of breeding lines and markers increase. Therefore this approach may serve as an alternative to traditional QTL mapping for FHB QTL and complement existing studies.

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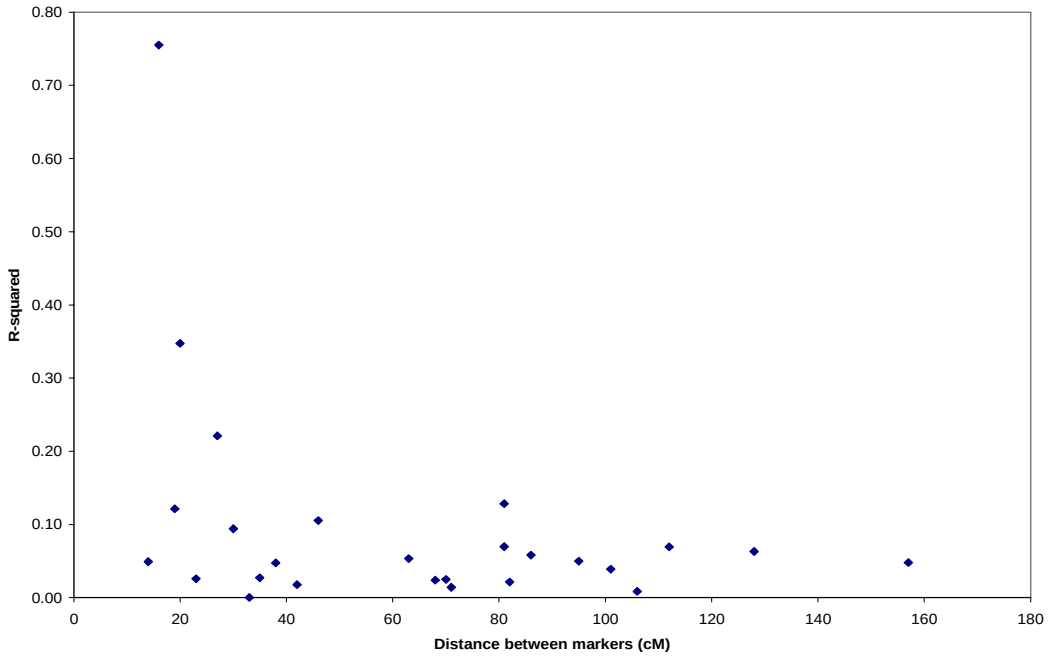

Figure 1. Linkage disequilibrium between markers on the same barley chromosome.

Table 1. FHB severity means for the six selected FEG populations and the resistant and susceptible tails selected from those populations.

FEG Population	Source(s) of resistance	Population Mean (±SD)	Resistant Tail Mean	Susceptible Tail Mean	Parent 1 Mean	Parent 2 Mean
103	AC Oxbow, Atahualpa	9.9±4.6	5.9±2.3	13.0±4.3	5.9	16.3
104	Zhedar1	8.7±4.4	4.4±2.3	15.3±4.5	7.4	16.3
105	Frederickson, PFC88209	7.4±4.5	4.1±2.7	11.8±4.7	5.4	5.6
107	Atahualpa, Frederickson, Harrington	9.9±5.1	6.1±3.2	15.4±4.6	8.2	8.4
112	AC Oxbow, Atahualpa, Zhedar1	10.0±6.4	5.5±4.0	17.0±6.3	6.4	11.0
121	AC Oxbow, Harrington, Zhedar1	5.3±3.4	3.3±1.9	8.5±3.0	3.9	6.1

Table 2. Association analysis and Two-tailed *t*-test results for 22 SSR markers.

		Association analysis *p*-values		Two-tailed *t*-tests by FEG population					
Chr.	Marker	FHB	HD	103	104	105	107	112	121
	EBmac0603	0.5462	0.2312	ns[1]	ns	ns	ns		
1	HVCMA	0.3103	0.8952					ns	
	Bmag0120	0.1868	0.5112			ns	ns	ns	
	Bmac0156	0.6235	0.5637	*p*<0.05		ns	ns	ns	ns
	HVM036	0.1473	0.9225	*p*<0.005				ns	ns
	GBM1052	0.3968	0.91464	*p*<0.025				ns	ns
2	Bmag0140	0.7363	0.0452						ns
	Bmag0125	0.7263	0.8296						ns
	HVM054	**0.0003**	**0.0746**	***p*<0.01**	***p*<0.005**			***p*<0.05**	
	Bmag0749	0.6153	0.0713	ns				ns	
3	Bmag0877	0.1616	0.1185			ns	ns		
	HVM040	**0.0014**	**0.9676**	***p*<0.01**			***p*<0.001**	***p*<0.025**	**ns**
4	EBmac0906	0.683	0.384				ns		
	HVM067	0.0203	0.3791				*p*<0.01		
	HVM043	0.5737	0.634						ns
5	Bmag0718	0.5796	0.4223						ns
	Bmag0579	0.2479	0.9989			ns		ns	*p*<0.05
	Bmag0173	0.2506	0.0239	*p*<0.01	ns	*p*<0.01		ns	
6	Bmac0040	0.112	0.947	ns		ns	ns	*p*<0.05	
	UMB603	0.304	0.6175	ns	ns	ns		ns	ns
7	Bmac0163	0.166	0.7608			ns			
	Bmac0303	0.3464	0.3841			ns	ns		

[1] FEG populations that were polymorphic for a marker but not significant are denoted with "ns." FEG populations that were not polymorphic for a marker have a blank cell.

DETERMINING AND REPORTING THE REACTION OF KANSAS COMMERCIAL WHEAT CULTIVARS TO FUSARIUM HEAD BLIGHT

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ABSTRACT

Fusarium Head Blight (FHB) is a serious disease of wheat. The best control for FHB is sowing resistant cultivars. To help Kansas wheat producers select which cultivars to plant, accurate information about their reaction to diseases must be disseminated to them. There are two main Extension publications that are used in Kansas for this purpose. They are *Kansas Performance Tests with Winter Wheat Varieties* and *Wheat Variety Disease and Insect Ratings*. Both are available as hard copy or online (http://kscroptests.agron.ksu.edu/04/04wheat/4w-Disease_Insects.asp and http://www.oznet.ksu.edu/library/plant2/samplers/mf991.asp). The reactions of 28 or 68 cultivars, respectively, to 12 different diseases (including FHB) are reported using a 1-to-9 scale where 1 = highly resistant and 9 = highly susceptible. Forty-seven Kansas winter wheat cultivars have been tested between one and 20 times each in 20 field experiments over a 6-year period. Experimental design for each location/year was a randomized complete block with four replications and plots were single rows, 2.3 m long. Corn grains colonized by *Fusarium graminearum* were applied to the soil surface in three applications about 2 wk apart beginning 4 wk prior to heading (100 g/m^2 total applied). During flowering, plots were sprinkler irrigated (3 min/hr) from 9:00 p.m. until 6:00 a.m. FHB index (% diseased spikelets) was determined for each cultivar between four and six times and averaged. For each experiment/year, index data were transformed to a 1-to-9 scale using linear regression. During the first two years of testing cultivars, an index value of 0% was assigned a Scale Value of "1" and the index value of the susceptible check cultivar with the highest index value in a location/year was assigned a Scale Value of "9." For years three through six, the known Scale Values for reference cultivars within each experiment/year, and their FHB index values, were used to generate the linear model. The models were then used to transform index values for all cultivars in that experiment to Scale Values. A mean Scale Value was calculated for each cultivar (mean of 1-20 experiment/years), rounded to the nearest whole number, and entered in the KSU Extension publications mentioned above. Because both publications are updated every year, Scale Values may be updated as more data become available. Scale Values obtained from these field experiments may be modified based upon observations of cultivars in KSU Extension demonstration plots or producer's fields where FHB naturally occurs. However, no significant disparities have been noted between ratings produced from inoculated nurseries and reactions seen in commercial fields.

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GENE PROFILING STUDIES IN TRICOTHECENE-INFLUENCED BARLEY - *F. GRAMINEARUM* INTERACTION

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ABSTRACT

Fusarium head blight (FHB) of barley is caused by *Fusarium graminearum* (teleomorph *Gibberella zeae*). Trichothecene mycotoxins, produced by the fungus during infection, play a role in virulence. Loss-of-function mutations in the *Tri5* gene, which encodes the first committed enzymatic step in the trichothecene biosynthetic pathway, results in the loss of trichothecene production and reduced virulence. We used the Barley1 and *Fusarium* Affymetrix GeneChips to examine the genetic mechanisms involved in the host and pathogen during trichothecene accumulation. We isolated RNA from spikes of the barley cultivar Morex inoculated with the *Tri5* mutant (non-trichothecene producing), andwild type (trichothecene producing) *Fusarium graminearum* strains and water, and hybridized the RNA to the Barley1 and *Fusarium* GeneChips. Three hundred and thirty seven barley transcripts were identified that were differentially accumulating in wildtype or *Tri5* inoculated plants versus water control inoculated plants. One hundred and twenty three of these 337 barley transcripts, were differentially accumulating in plants inoculated with the wildtype strain versus the plants inoculated with the *Tri5* mutant strain ($P<0.001$), indicating that there are barley genes that are up-regulated specifically during trichothecene accumulation. In the same set of 337 barley transcripts, we also detected 26 that were differentially accumulating in plants inoculated with the *Tri5* mutant strain compared to plants inoculated with the wildtype strain ($P<0.001$), indicating that these barley genes may be down-regulated during trichothecene accumulation. During the same interaction, 603 transcripts were found to be differentially accumulating between the *Tri5* mutant and wildtype *F. graminearum* strains. Seven transcripts showed exclusive accumulation in the *Tri5* mutant *F. graminearum*. Five hundred and ninety three transcripts were up-regulated in wild type compared to the *Tri5* mutant. Three transcripts differentially accumulated in the *Tri5* mutant strain compared to the wildtype strain. Annotations and the functional significance of these differentially accumulating transcripts will be presented.

A COST-EFFECTIVE HIGH THROUGHPUT GENOTYPING METHOD

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ABSTRACT

Although extensive breeding effort has been directed at improving resistance to Fusarium head blight (FHB) in wheat and barley throughout the region, the rate of releasing resistant cultivars is slow. The main challenges lie in the complex inheritance of FHB resistance, and screening of a large number of breeding lines by conventional phenotypic testing. DNA markers have been identified and tagged to a few major resistance genes in both wheat and barley. But widespread application of marker assisted selection in wheat and barley breeding for FHB resistance has been limited up until now. The recent rapid advancement of high throughput platforms and DNA-based diagnostic assay technologies have enabled the Fargo genotyping lab, along with the wheat and barley breeders from Minnesota, to initiate a pilot study to establish a cost-effective working protocol that is amenable to automation. This protocol has been implemented in the breeding programs to enhance wheat and barley breeding efforts in selecting and releasing lines resistant to FHB. A detailed method from sample preparation by the breeders to genotyping data delivery from the Fargo genotyping lab will be presented.

PHENOTYPIC AND GENOTYPIC ANALYSIS OF SCAB RESISTANCE IN SOFT RED WINTER WHEAT GERMPLASM

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ABSTRACT

Effective MAS for FHB resistance depends on knowledge of the genetic relationship of the germplasm to be improved with identified FHB resistance QTLs. This information is lacking for most moderately resistant soft wheat germplasm. In this study, a large set of soft wheat lines and checks were evaluated in inoculated field nurseries at four locations to determine their relative levels of FHB resistance and agronomic performance at each location. The lines were also genotyped at SSR markers from regions of the genome where QTLs for FHB resistance have been identified. These data are being combined with pedigree information to develop a data base on the putative source of FHB resistance alleles in soft wheat germplasm. The analysis will allow breeders to better select parents for crossing in efforts to develop lines having high levels of resistance to scab.

MATERIALS AND METHODS

Breeders from ten states in the eastern US submitted soft wheat lines with moderate to strong resistance from native and/or exotic sources. The 247 lines, including susceptible checks, were grown in screening nurseries at Wooster, OH, Urbana, IL, Lexington, KY and Blacksburg, VA. Data were collected on severity and incidence of disease and a scab index was calculated. Lines were also evaluated in the greenhouse at Lafayette, IN and percent infected florets were recorded.

Genomic DNA was isolated from five plants of each the lines. Two of the plants from which DNA was isolated are being grown in the greenhouse at Raleigh, NC. Seed of these plants will be provided to breeding programs interested in crossing to genotyped individuals. For marker analyses, DNA was also isolated from exotic sources of FHB resistance that were not suited for field evaluation and from lines provided by the University of Missouri not included in the field study. All lines are being genotyped with simple sequence repeat (SSR) markers from regions of the genome where QTLs for resistance have been identified (for review see McCartney et al. 2004).

RESULTS

The mean scab index of lines across locations ranged from 4.1 to 51.7. A large percentage of lines (78%) were classified as having a low rating (<24.3). Twenty-one lines were classified as susceptible (index >32.0) and 36 lines were intermediate or were inconsistent across locations. To date, marker analysis identified the Sumai 3 haplotype at the 3BS QTL region in a small number of lines. These lines had low levels of disease and exotic sources of resistance in the pedigree. Analysis of haplotypes at other QTL regions will be presented.

COMPARISON OF TWO SCAB INOCULATION METHODS IN WHEAT

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ABSTRACT

Fusarium head blight (FHB), or head scab, is a widespread and destructive disease of wheat and barley. Identifying breeding lines with host plant resistance to FHB is an important breeding objective. Many inoculation and evaluation methods are used to identify breeding lines with resistance to FHB. In many cases phenotypic data collected using different inoculation and evaluation methods are poorly correlated. Our objectives in this study were to determine if FHB resistance ratings resulting from two different inoculation methods were highly correlated, and if the same breeding lines with the highest resistance were selected using the two inoculation methods. The two inoculation methods used were a spray-and-bag method using a macroconidial suspension and enhancement of natural infection using infected grain spawn and mist irrigation. Each method was tested in 2005 using a total of 132 lines in three separate experiments. Scab incidence and severity data were collected for both methods, and a FHB index was calculated. The scab data combined with agronomic data were correlated using the PROC CORR procedure of SAS and a significance threshold of a= 0.05. Both scab incidence (r = 0.22) and the FBH index (r = 0.19) were significantly correlated between methods, but with low linear relationships. Analyzing the data using only a subset of the lines with a FHB index in the top 20% and bottom 20% of the grain spawn infected method increased the correlation for both incidence (r = 0.42) and FHB index (r = 0.39). Breeding lines with highest resistance under grain spawn/mist infection did not agree with lines selected as most resistant with the spray and bag infection. For thirty-two (24%) lines the FHB index differed by more than thirty between the two methods. In this study many of the lines selected with one inoculation method would not have been selected with the second method; however, this is preliminary data. It is disconcerting that some of the lines with the highest resistance using one method were not selected using the second method. These results reemphasize the importance of basing selection for FHB resistance on multiple evaluations.

THE ALIEN GENE COULD BE ONE OF THE 'FIGHTERS' AGAINST FUSARIUM HEAD BLIGHT IN WHEAT

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ABSTRACT

Fusarium head blight (FHB), caused mainly by the fungus *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schw.) Petch], has been an important disease of wheat worldwide. Epidemics of this disease can result in significant economic losses for wheat growers in terms of yield and quality. Extensive efforts have been made to utilize host resistance to manage this devastating disease. The progress, however, has been limited because of the lack of effective resistance sources and the complex inheritance of the currently identified sources of resistance. This study was initiated to discover novel sources of resistance in the relatives of wheat, an invaluable gene pool for wheat improvement. Fusarium head blight resistance has been identified in a number of relatives of wheat. Resistance in some of the relatives has been transferred to wheat via chromosome manipulation. We have evaluated reaction of 293 lines derived from the crosses of wheat with its relatives to FHB over two greenhouse seasons. Of these 293 derivatives, 66 were susceptible, 153 appeared moderately resistant, and 74 lines exhibited a level of resistance comparable to *Triticum aestivum* cv. Sumai 3, the most widely used source of resistance to FHB. Alien species involved in development of these derivatives include *T. tauschii* (Coss.) Schmal., *Roegneria kamoji* C. Koch, *R. ciliaris* (Trin.) Nevski, *Leymus racemosus* Lam., *Thinopyrum ponticum* (Podp.) Barkworth & D.R. Dewey, *Th. elongatum* (Host) D.R. Dewey, *Th. junceum* (L.) Love, *Th. intermedium* (Host) Barkworth & D.R. Dewey, *Elymus rectisetus* (Nees) Love et Connor, *Dasypyrum villosa* L., *Secale cereale* L., and oat *(Avena sativa* L.). The wheat-alien species derivatives identified as resistant to FHB include wheat-alien species amphiploids, synthetic hexaploid wheat lines, and wheat-alien species chromosome substitution and translocation lines. These derivatives could serve as novel sources to enhance resistance of wheat to FHB. However, these lines contain varied amounts of alien chromatin in their genomes and cannot be utilized directly in breeding. We have been characterizing chromosome constitutions of these lines using molecular cytogenetic techniques and molecular markers. Meanwhile, we have been eliminating unwanted alien chromatin from their genomes via chromosome manipulation. This will allow for the development of breeder-friendly germplasm lines resistant to FHB and the involvement of alien resistance genes in fighting this destructive disease in wheat.

SEARCHING FOR NOVEL SOURCES OF RESISTANCE TO FHB IN BARLEY

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ABSTRACT

ICARDA has been producing barley with enhanced resistance to Fusarium head blight (FHB) in its program based in Mexico in cooperation with CIMMYT since the early 80's. Its germplasm bank in Syria offers a diverse reservoir of genes that can be explored as possible new sources of resistance for this devastating disease. Barley and wheat were domesticated in the Near East region some 8-10,000 years ago. They have developed different adaptive mechanisms for stress tolerance under farmers' selection. Crop wild relatives represent even richer reservoirs of genes for stress tolerance and adaptation, as their history in the Central and West Asia and North Africa (CWANA) region is much longer and includes periods of a very harsh climate in the Pleistocene Era. The ICARDA/CIMMYT barley breeding program started to research for resistance to FHB, in response to the need of resistance to this disease in the countries of the Andes. In 1986 a total of 5000 barley accessions were screened in Mexico, from those only 23 were found with some level of resistance, which were intensively introgressed into the main program. Resistant sources were shared with programs worldwide, especially after the epidemic outbreaks of the 90's. Collaboration and cooperative research with research groups of advanced research institutions such as the US Wheat & Barley Scab Initiative (USWBSI) is leading the program to make available germplasm sources with enhanced levels of resistance. The environmental conditions present at the Toluca Experiment Station in Mexico are ideal for FHB development and evaluation. Besides Toluca, data available from the US, Canada, China, Ecuador, Brazil and Uruguay were obtained through collaboration with other programs. In recent years the program started a directed comprehensive screening of the gene bank of ICARDA, searching for unique sources of resistance not yet identified by other programs. Preliminary results indicate that new barley sources might be identified at the ICARDA Gene Bank.

HAPLOTYPE SELECTION OF TWO MAJOR QUANTITATIVE TRAIT LOCI FOR IMPROVED FUSARIUM HEAD BLIGHT RESISTANCE IN ELITE WHEAT BACKGROUNDS

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ABSTRACT

This study was conducted to evaluate and validate the presence of two major Fusarium head blight (FHB) resistance QTL on chromosomes 3BS and 5AS in seventy soft red winter (SRW) wheat elite lines. Five haplotypes were characterized among the elite lines on the basis of allelic differences of four marker loci linked to the 3BS QTL and two marker loci linked to the 5AS QTL. Genetic effects of the loci and QTL haplotypes for FHB resistance were analyzed on the basis of disease data collected in both greenhouse (type II resistance) and field experiments (type I and II resistance or field resistance). This study validated the presence of two major QTL on chromosome 3BS and 5AS, and illustrated the utility of SSRs and STS markers in the two QTL regions in selection of FHB resistance in elite wheat backgrounds. Findings of this study also indicate that the 3BS QTL region may be comprised of multiple loci governing FHB resistance. The 3BS QTL1 region, flanked by markers Xbarc133-XSTS142, has a significant effect towards improving both type II and field resistance. The 3BS QTL1 may be unique to Chinese sources. The 3BS QTL2 region, flanked by markers Xgwm493-Xcfd79, has a significant effect towards improving type II resistance but likely has less effect or even a negative effect on field resistance in adapted wheat backgrounds. The 3BS QTL2 is common in both Chinese and native sources. This study confirmed that the 3BS and 5AS QTL have an additive x additive effect towards improving both type II and field resistance. Simultaneous MAS of ideal haplotypes for both QTL likely will be the most effective strategy for improving FHB resistance. The ideal haplotype was comprised of four favorable marker alleles including two (Xbarc133 and XSTS142) on 3BS and two (Xbarc117 and Xbarc56) on 5AS. Selection of desired marker alleles in coupling at each QTL region may be difficult initially if they are derived from different parents, but once combined subsequent selection should be easy and provide a reliable and effective means for incorporating and improving overall FHB resistance in adapted backgrounds. This study also presents and discusses possible strategies for combining FHB resistance with high yield potential through MAS. Elite lines having desirable haplotypes identified in the current study will provide breeding programs with a source of unique and adapted FHB resistant parents and some of the lines also may have potential for release as cultivars.

SCAB SCREENING OF SOFT RED WINTER WHEAT GENOTYPES IN MARYLAND

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ABSTRACT

The 2004/2005 wheat growing season did not present favorable environmental conditions for the development of a scab (*Fusarium graminearum*) epidemic in Western Maryland as in the previous 2 seasons. Seed quality and test weight of wheat were excellent across the mid-Atlantic region. A nursery of soft red winter wheat advanced lines from the Maryland breeding program was grown under field conditions in Salisbury (MD) with misting and artificial inoculation (corn kernel method). The level of scab incidence, severity, percentage of *Fusarium* damaged kernels (FDK), and Deoxynivalenol (DON) were assessed as well as heading date, height and kernel weight. One-hundred and sixty advanced wheat lines were tested in this replicated nursery. The incidence of the disease was fairly uniform across this nursery with significant differences between the susceptible (Coker 9835) and moderately resistant checks (McCormick). There were significant genotypic differences overall for scab incidence, severity, FDK and DON among these genotypes. A small group of advanced lines that included the moderately resistant genotype McCormick showed moderately high levels of resistance to scab with low scab FDK and DON values. On the other hand, there were a large number of genotypes that were very susceptible although variation in the various measures of scab damage was large. The lowest coefficient of variation was observed for incidence (36%) and the largest was observed for FDK (71%). Several lines with low FDK and DON were derived from the cross PION2643/MASSEY*3/ BALKAN//SALUDA. Two of these lines (MV6-82-8 and MV6-82-10) were entered into the Uniform Northern and Southern Scab Nurseries in 2005/2006. These lines do not have any of the chinese or other exotic sources of resistance to scab in their pedigree. It is important, however, to continue to screen adapted advanced lines of soft red winter wheat for even moderate scab resistance within the native soft red winter wheat germplasm. This can be useful for future breeding in combination with other major sources of resistance to scab to reach the goal of developing disease-resistant varieties in the near future that are adapted to the mid-Atlantic region of the USA.

BREEDING EFFORTS TO DEVELOP FUSARIUM HEAD BLIGHT RESISTANT DURUM WHEAT IN NORTH DAKOTA

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ABSTRACT

Durum wheat is one of the major cereal crops in the world and its production in North Dakota accounts for about 75% of the U.S. production. Fusarium head blight (FHB) caused by the fungus *Fusarium graminearum* Schwabe (telomorph *Gibberella zeae* (Schwein.) Petch. has been seriously attacking durum wheat (*Triticum turgidum* L. var. durum) in North Dakota and the surrounding states. FHB has caused a continuous decline in harvested durum acreage and production in ND. Fungicides may reduce the disease but the most environmentally safe and economical way to control the disease is with genetic resistance. Our objectives are in line with the objectives of the US Wheat and Barley Scab Initiative, which are to identify and characterize FHB resistant durum wheat that can be used to develop FHB resistant cultivars/germplasm with good agronomic and quality traits.

To date, we have evaluated a total of 6,000 durum accessions from the world collection at the Academy of Agricultural Sciences, Plant Protection Institute Shanghai, China. None of these accessions were resistant. We are evaluating 1,500 additional accessions at the Department of Plant Protection, Hangzhou, Zhejiang, China. We also have screened material from CIMMYT and ICARDA and identified five Tunisian lines that have a moderate level of Type II resistance to FHB. We are in the process of characterizing the resistance in these lines using segregating pattern and normal distribution for Type II disease severity. We also will be utilizing simple sequence repeat (SSR) markers to identify the FHB QTL in these lines. Two CIMMYT lines have been identified that have 14% Type II disease severity. We have received germplasm from ICARDA for FHB evaluations starting in 2005-06. Our intent is to screen a wide range of durum germplasm until a good source of resistance to FHB is identified.

In previous studies we found that Langdon Triticum dicoccoides 3A substitution line [LDN(DIC-3A)] had a moderate level of Type II resistance. We have developed doubled haploids lines from crossing durum wheat cultivars to the LDN(DIC-3A) line. We have evaluated these lines for Type II resistance using the injection method and the microsatellite marker Xgwm2 and for agronomic and quality traits in preliminary yield trials grown at Prosper and Langdon, ND. Lines that were selected as resistant to FHB did not have acceptable agronomic and quality traits to be released as cultivars. They are being used as parents for second cycle of breeding. Additional lines have been generated by backcrossing this source of resistance to popular durum cultivars ('Ben', 'Lebsock', 'Maire' and 'Plaza'). These lines are now being increased for further evaluation. LDN(DIC-7A) was identified by Drs. James Miller and Robert Stack to have some level of resistance to FHB. We are developing populations by crossing the LDN(DIC-7A) with durum cultivars/experimental lines for breeding purposes.

We have transferred the resistance from the Chinese hexaploid wheat 'Sumai 3' and 'Wangshuibai' to durum wheat. Several populations have been developed from crossing the FHB resistant durum lines with the Sumai 3 and/or Wangshuibai resistance with new ND generations from these populations are being evaluated for Type II resistance using the injection method and the DNA markers Xgwm533, Xgwm493, STS3B-66,

barc133, and barc180. Several lines from these populations will be evaluated as F5:6 lines and subsequent generations for agronomic traits, quality, and disease resistance. Lines that have good level of resistance and possess good agronomic and quality traits will be released as cultivars to the producers. Some of the identified resistant lines will be used as parents in crosses to generate a second cycle of breeding.

ACKNOWLEGDEMENT

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DIGITAL IMAGE ANALYSIS OF PRIMARY LEAF LESIONS ON WHEAT SEEDLINGS OF FRONTANA AND ALSEN INOCULATED WITH *FUSARIUM GRAMINEARUM*

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ABSTRACT

Digital image analyses were conducted on primary leaf lesions of Frontana and Alsen wheat cultivars either 96 or 120 h post-inoculation with macroconidial inoculum of *F. graminearum*. Seedlings were planted in Conetainers in a split-plot design, with cultivars as main-plots and fungal isolates as subplots, and grown to the two-leaf stage in the greenhouse, exactly 14 days post-planting. Primary leaves of seedlings were inoculated separately using five different isolates of *F. graminearum*, obtained from the University of Minnesota Small Grains Pathology Project, St. Paul. Water inoculated primary leaves of seedlings for each cultivar provided control comparisons. Following inoculation, plants were maintained at nearly one hundred percent relative humidity at 23 °C for either 72 or 96 h. Lighting was provided under a 12 h light:dark period while plants were maintained in the incubation chamber. Following incubation, plants were removed to lab benches beneath artificial lighting at temperatures from 21 to 23 °C for another 24 h. Primary leaves were excised at their base near the ligule and placed on a photographic stage. Leaves with lesions were photographed using a high-resolution digital camera. Images were analyzed using the Assess digital image analysis software obtained from the American Phytopathological Society Press. Threshold levels of lesion area were established by setting the hue, saturation, and intensity indices of the program to discriminate lesions of inoculated leaves relative to control leaves to provide differentiation of symptomatic versus healthy appearing leaf area (chlorotic and necrotic tissue relative to healthy green tissue). In preliminary experiments, mean percent lesion area of inoculated leaves of Frontana was 1.9 % and was significantly lower (P=0.05) than for Alsen, which was 4.5 %. No significant differences were observed among isolates (P=0.05) of *F. graminearum* and no significant cultivar by isolate interactions were observed (P=0.05) for percent lesion area assessment. This technique of digital image leaf lesion assessment is being explored to differentiate among susceptible and resistant leaf reactions of segregating wheat populations in a rapid screening of reaction to early infection of wheat.

A RECIPROCAL BACKCROSS MONOSOMIC ANALYSIS OF THE FHB RESISTANT WHEAT CULTIVAR 'FRONTANA'

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ABSTRACT

Fusarium head blight (FHB) caused by the fungal organism *Fusarium graminearum* Schwabe [teleomorph *Gibberella zeae* (Schwein.)], is a disease of wheat and other small grains which frequently causes a significant reduction in grain yield and quality. Wheat breeders are interested in incorporating alternative sources of resistance into new genotypes as well as identifying genes which function differently from inhibiting fungal spread after infection. The spring wheat cultivar 'Frontana' has been described as exhibiting a mode of FHB resistance which might limit initial infection by the pathogen or degrade the fungal toxin after initial infection. A study was initiated to identify what chromosomes were involved in determining the FHB resistance of Frontana. Frontana was hybridized to a set of 'Chris' spring wheat monosomics, and a backcross reciprocal crossing procedure was followed to produce two sets of disomic lines, one set with critical chromosomes originating from FHB resistant Frontana and the other with critical chromosomes originating from FHB susceptible Chris. The parental genotypes; resistant and susceptible controls; and disomic lines were grown in a RCBD with three replications in two separate greenhouse experiments (GH experiment-1 and GH experiment-2). Disomic lines for Frontana critical chromosomes 3B and 7D were not produced and so were not available for testing. Plants were spray-inoculated with a single *Fusarium* isolate at a concentration of 25,000 spores ml^{-1}. In GH experiment-2, percent disease severity ratings of disomic lines were made one, two, and three weeks after inoculation. After harvesting seed from both experiments, disomic plants were evaluated for resistance by counting tombstone kernels, weighing seed, and analyzing seed samples for deoxynivalinol (DON) content. In GH experiment-1, disomic lines with Frontana chromosomes 2D, 5A, 6A, and 7A had significantly lower percent tombstone kernels, kernels g^{-1} seed, and DON content compared with other disomic Frontana lines; however, all four lines had higher mean values than the Frontana control. In GH experiment-2, disomic lines with Frontana chromosomes 6A and 7A again exhibited significantly lower values for percent tombstone kernels and DON content compared with nearly all of the other disomic Frontana lines as well as the Frontana parent. In GH experiment-2, percent severity ratings for disomic Frontana lines 6A and 7A were similar to the 'Alsen' control at all three weeks and significantly less than the Frontana parent at all three weeks. Results indicate that genes for FHB resistance in Frontana are likely carried on chromosomes 6A and 7A.

EVALUATION OF WHEAT LINES NEAR-ISOGENIC FOR DIVERSE FUSARIUM HEAD BLIGHT RESISTANCE QTLS

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ABSTRACT

The hard red spring wheat (HRSW) production region has suffered large economic losses during the past decade due to Fusarium head blight (FHB) epidemics. To enrich HRSW with novel FHB resistance QTLs, a program to introgress FHB resistance QTL from diverse wheat genotypes and wheat relatives into HRSW in a systematic fashion has been initiated. The project is employing four generations of marker-assisted backcrossing to create HRSW lines near-isogenic for different FHB resistance QTLs (QTL-NILs). These QTL-NILs are being developed in three FHB susceptible HRSW backgrounds (Norm, Wheaton, Apogee). Apogee is a rapid cycling dwarf wheat that allows for the development of unique resources for FHB research. In 2005, FHB resistance evaluations were conducted on the first set of completed QTL-NILs. This included QTL-NIL series in both Norm and Apogee that have either a marker for a FHB resistance QTL on Freedom (a soft red winter wheat) chromosome 2A, or for individual QTLs from Sumai 3 (*Qfhs.ndsu-3BS, Qfhs.ifa-5A*) that are serving as checks. Greenhouse FHB evaluations involved point inoculations to assess the spread of disease symptoms in the spike (type II resistance). Twenty five plants (5 pots with 5 plants per pot) per QTL-NIL were evaluated. Disease spread in Norm was 10.0. In the best Norm-*Qfhs.ndsu-3BS* NIL, disease spread was 4.7, while disease spread was limited to just 4.2 in the best Norm-Freedom 2A NIL. In the Apogee QTL-NIL series, the best *Qfhs.ndsu-3BS* line exhibited a disease spread of 3.4, compared to a disease spread of 9.0 in Apogee. Disease spread in the best Apogee-Freedom 2A NIL was limited to 5.1 spikelets. A replicated FHB field evaluation of the first set of NILs was also conducted in 2005. In the Norm QTL-NIL series, the best *Qfhs.ndsu-3BS* and Freedom 2A NILs exhibited FHB severities of 14.5 and 13.9, respectively. In contrast, Norm exhibited a FHB severity of 27.6. The best Norm-*Qfhs.ifa-5A* NIL exhibited a FHB severity of 18.9. Similarly, Apogee exhibited a FHB severity of 30.2, while the best Apogee-*Qfhs.ndsu-3BS* and Apogee-Freedom 2A NILs exhibited significantly lower FHB severities (10.7 and 11.3, respectively). The best Apogee-*Qfhs.ifa-5A* NIL exhibited a FHB severity of 22.4. These results suggest both that *Qfhs.ndsu-3BS* and the Freedom 2A QTL have each been introgressed into NILs in both Norm and Apogee, and that the Freedom 2A QTL may confer a significant level of FHB resistance to HRSW. Results for *Qfhs-ifa-5A* are more equivocal. Experiments will be repeated to confirm this first round of greenhouse and field evaluations. We are now completing the introgression of two additional QTLs, *Qfhs.ndsu-3AS* from *Triticum dicoccoides*, and a QTL from Frontana located on chromosome 3A. These QTL-NIL series provide prebreeding resources for breeding programs, as well as also genetic stocks for 1) quantifying gene pyramiding, 2) examining the molecular basis of diverse, and 3) exploring biological differences between resistance to initial infection (type I) and type II resistance.

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HIGH RESOLUTION PROFILING OF WHEAT GENES DIFFERENTIALLY EXPRESSED IN RESPONSE TO *FUSARIUM GRAMINEARUM* INFECTION

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ABSTRACT

Fusarium head blight (FHB) of wheat caused by *Fusarium graminearum* (Schw.) affects wheat production worldwide reducing yield and quality. Genome-wide expression profiling of genes altered during host-pathogen interactions may improve our understanding of the mechanism(s) underlying resistance to FHB. A cDNA biochip representing 5664 ESTs, derived from a suppression subtractive hybridization library of wheat-*F. graminearum* interactions, was used for tissue-specific profiling of differentially-expressed genes in response to *F. graminearum* infection. The 93FHB37 wheat line carrying three major resistance QTLs mapped to chromosomes 3BS, 6BS and 5AL was used for the study. Inoculated wheat spikes were dissected into tissues: glume, lemma, palea, ovary, anther and rachis. The monitoring of genes in specific tissues avoided the averaging of expression data that occurs when using an entire spike as a biological sample. Hybridizations were completed using 30 arrays including 5 independent hybridizations for each tissue. Significant analysis of microarrays (SAM) resulted in the identification of transcripts encoding defense and stress related proteins, components of the ethylene and the phenylpropanoid pathways and a member of WRKY transcription factor family. Analysis of variance revealed that about 37% of genes responding to *F. graminearum* showed a significantly different expression pattern among separate floral tissues.

IDENTIFICATION AND INCORPORATION OF FHB RESISTANCE IN WINTER WHEAT: AN OVERVIEW OF PROGRESS AND STRATEGIES

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Since 1990, Fusarium Head Blight (FHB) epidemics have occurred in many of the eastern and Great Plains winter wheat production regions of the U.S. where much of the wheat crop is planted directly into maize residue. Subsequently, severe FHB epidemics resulting in significant losses in grain yield and quality have occurred on an annual basis in one or more of these wheat production regions. Prior to 1990, few winter wheat breeding programs considered it necessary or placed a significant amount of emphasis on the identification and development of cultivars having resistance to FHB. Initially uncertainty prevailed as to whether cultivars having significant resistance to FHB could be derived from existing breeding populations in which parental lines had not been selected on the basis FHB resistance.

PROGRESS AND EFFECTIVENESS OF FHB BREEDING EFFORTS

Much of the information included in this paper and the oral presentation was compiled from responses to a questionnaire sent to colleagues representing 14 public and four private winter wheat breeding programs working on FHB resistance and variety development in the U.S. When asked whether current breeding methods, available technologies, and strategies deployed have been effective in development of FHB resistant cultivars, the responses varied from very effective to not exceedingly effective. A majority of breeding programs indicated that significant progress has been made in development of cultivars having moderate and in a few cases high levels of FHB resistance derived from adapted native sources and/or that FHB resistance has been successfully transferred from exotic sources into more adapted backgrounds. While competitive cultivars having FHB resistance derived from native sources have and will continue to be developed, the FHB resistance level in most but not all of these likely will be inadequate under severe epidemics. To a certain extent the same is true for cultivars having only Type II resistance derived from exotic sources, wherein development of competitive cultivars that are highly resistant to FHB and possess other traits of critical importance has proven more difficult to achieve. However, as breeding programs begin to use more adapted and improved parental lines, having diverse combinations of Type II and other types of resistance derived from exotic sources and adapted lines having native resistance, the number of competitive FHB resistant cultivars will increase dramatically.

PRIMARY FACTORS HINDERING EFFECTIVENESS OF FHB BREEDING

Difficulty in obtaining uniform FHB epidemics of desired intensity and phenotypic data that is consistent and reliable from inoculated and mist-irrigated field nurseries is problematic due to lack of control over environmental conditions, particularly temperature. This is further confounded by the effects of significant genotype by environment interaction and differences in plant structure, height and maturity on FHB phenotypic data, and lack of consistent correlation between FHB incidence, severity and FDK with DON concentration. While significant progress has been made in identifying, mapping, incorporating and enhancing FHB resistance in winter wheat, there has been less success to date in combining high levels of FHB resistance with other traits of critical importance that are ultimately required in successful and competitive cultivars. Deriving lines that are high yielding as well as highly resistant to FHB as well as maintenance of genetic gains in yield that are at least similar to current levels are problematic. However, as one respondent clearly stated "success likely will come through the deployment of a

long term recurrent selection strategy," which is relevant since progress achieved to date in wheat breeding programs has resulted ultimately from long term recurrent selection in which favorable alleles and linkage groups have been established and maintained primarily via phenotypic selection. So the difficulty encountered in initial breeding efforts, which can be considered as pre-breeding, to incorporate high levels of FHB resistance, derived from exotic or non-adapted parental sources, into high yielding wheat backgrounds is not surprising or unexpected. Likewise restoring favorable linkage groups for other quantitative traits, such as adaptation, milling and baking quality and the numerous assortments of major and minor gene loci governing horizontal and general background resistance to a vast array of biotic and abiotic stresses, likely will require several breeding cycles to achieve, which generally is limited to two cycles per year in winter wheat breeding programs. Progress has further been restricted by limited knowledge of the inheritance and diversity of genes conferring FHB resistance among native sources versus known FHB QTL, and lack of molecular markers to apply MAS in populations comprised of native sources of FHB resistance. In addition to the aforementioned problems associated with phenotypic selection, it requires extensive time, resources, and highly-trained personnel to implement. While it is generally agreed upon that high-throughput MAS has the potentially to greatly enhance the effectiveness of breeding for FHB resistance, the capacity to implement this on a broad scale has still not been optimized. There also is need for more PCR-based selectable molecular markers that are reliable, predictive and broadly applicable, such as "gene based" markers. There is need for validation of putative novel FHB resistance QTL in diverse genetic backgrounds and to develop selectable markers for these with emphasis on complementary types of FHB resistance, particularly tolerance to toxin accumulation. There is need to document whether pyramiding diverse QTL conferring primarily Type II resistance will act in a complementary manner and result in an increase in the overall level of FHB resistance as well as providing genetic diversity.

FHB RESISTANCE IDENTIFIED IN NATIVE SOURCES

Upon evaluation of existing adapted winter wheat lines and cultivars in FHB nurseries, several were documented as having moderate levels of FHB resistance, and subsequently referred to as "native" resistance or native sources. The SRW wheat cultivar Freedom, released by Ohio State University in 1991, was among the first released winter wheat cultivars identified with native resistance, and its FHB resistance was subsequently mapped to chromosome 2AS. Shortly thereafter the SRW wheat cultivars COKER 9474 and Ernie, both released in 1994, were identified as having moderately high levels of native FHB resistance. FHB resistance in Ernie was subsequently mapped and reported to be conferred by QTL on chromosomes 2B, 3B, 4B, and 5A. Like many native FHB resistance sources, COKER 9474 has the same allele as Chinese sources for the 3BS QTL marker Xgwm 493. An even higher level of native FHB resistance was identified in the cultivar Truman, released by the University of Missouri in 2003. Since 1994, more than 15 SRW and several HRW wheat cultivars having native FHB resistance have been released and include Foster (1996), Patton, Roane, Hondo and Heyne (1998), Wesley and Goldfield (1999), McCormick and Tribute (2002), Neuse, Truman, INW0304 and IL94-1653 (2003), Cecil and INW0411 (2004), and Bess, NY88046-8138, COKER 9511, and WestBred X00-1079 (2005). Most of these native sources have Type II field resistance, and several also have resistance conferring low FHB incidence (Type I), and reduction in FDK and DON. Winter wheat lines having native FHB resistance and being considered for release during the next three years include eight lines in 2006 (developed by breeding programs in Georgia, Illinois, Indiana, Nebraska, Ohio, and New York), two lines in 2007 (from Ohio and Missouri), and five lines in 2008 (from Kentucky and Ohio). It is apparent that native FHB resistance currently comprises and will continue to provide a base level of FHB resistance in winter wheat cultivars. FHB resistance in only a few native sources has been genetically character-

ized or mapped, and this remains a critical priority if genes in these potentially novel sources of resistance are to be effectively used, selected for and combined with genes from other unique native and exotic sources in cultivar development programs.

INCORPORATION OF FHB QTL FROM ASIAN AND EUROPEAN SOURCES

In an endeavor to incorporate novel FHB resistance and/or to enhance current resistance derived from native sources, many programs initiated efforts using a vast array of breeding methods to incorporate Type II FHB resistance, derived predominantly from a seemingly diverse array of Asian and other sources, into adapted winter wheat backgrounds. Subsequent emphasis has been placed on identifying diverse sources of Type II resistance as well as other unique types of resistance and their incorporation and combination in elite wheat lines. Of the QTL reported for FHB resistance, those located on chromosomes 1B, 2AS, 2B, 2D, 3A, 3BS, 4BL, 5AS, and 7B have been postulated as conferring resistance among current winter wheat cultivars and advanced elite lines. Winter wheat varieties having FHB resistance derived directly from Asian (3BS and 5AS) and/or European (1B and 3A) sources or from diverse combinations of these with native sources includes the cultivars 25R18 (released in 1999), 25R42 (2001), 25R35 and 25R54 (2003), INW0412 (2004) and 25R51 (2005). In addition, six elite wheat lines having FHB resistance derived primarily or partially from exotic sources are being considered for release within the next three years by breeding programs at Purdue, Cornell and Virginia Tech. To date, notably fewer cultivars having FHB resistance derived from exotic sources have been released in comparison to cultivars having native resistance. This is due in part to the time and resources required to incorporate FHB resistance from exotic sources into adapted winter wheat backgrounds, lack of precise and broadly applicable high-throughput PCR-based markers for all known FHB QTL, and difficulties encountered in eliminating undesirable traits and in restoring favorable linkage groups for adaptation, pest resistance, grain yield, and quality.

BACKCROSSING TO INCORPORATE FHB RESISTANCE

Initial backcrossing efforts relying on phenotypic selection for Type II resistance via point inoculation under greenhouse conditions generally were effective in transferring FHB resistance conferred by genes having major effects on reduction of disease spread, and were further expedited upon the availability and use of molecular markers linked to known FHB QTL such as the one on chromosome 3BS. While many of the FHB resistant donor parents were subsequently characterized as having additional QTL (e.g. 5AS, 5DL) conferring significant but lower levels of Type II resistance and/or other types of FHB resistance, many of these QTL were not retained in backcross progeny selected solely on the basis of Type II phenotypic reaction. Subsequent identification and validation of these and other novel QTL and availability of predictive molecular markers to deploy in MAS has and will continue to greatly enhance the ability of breeding programs to further enhance the level of FHB resistance and to reduce linkage drag in future cultivars. In collaboration with the USDA-ARS Genotyping Centers, several programs have developed regional MAS backcrossing populations to rapidly incorporate two or more known FHB resistance QTL into adapted winter wheat backgrounds.

DOUBLE-HAPLOID FHB BREEDING EFFORTS

Doubled haploid breeding was initiated by several programs to accelerate the transfer of FHB resistance into adapted wheat backgrounds. While FHB QTL such as 3BS and 5AS were incorporated into winter wheat backgrounds, most lines lacked other traits of critical importance required for cultivar release, such as resistance to other prevalent diseases and yield potential and, therefore, are best suited for use only as improved FHB resistant parental lines. Most breeding programs have discontinued development of doubled haploid lines as part of their routine cultivar development efforts, as this method requires extensive time and resources to implement and large populations are

required to identify desirable progeny, particularly when non-adapted parents are involved.

BREEDING METHODS AND STRATEGIES DEPLOYED IN DEVELOPING FHB RESISTANT CULTIVARS

In more than half of the winter wheat breeding programs surveyed, parental selection, line screening and selection in all populations includes emphasis on FHB resistance. Breeding programs in regions less prone to FHB epidemics generally develop separate crosses targeting FHB resistance, which are either advanced with their traditional populations to the pure line stage or are advanced separately with selection for FHB resistance. Several programs have independent breeding projects focused specifically on development of FHB resistant cultivars and a few programs also have independent parent building programs. Nearly 85% of the winter wheat breeding programs predominantly use a bulk or modified bulk breeding method with mass selection, while a few of these programs subsequently implement pedigree selection for FHB resistance in target populations. Two breeding programs use a pedigree method predominantly with selection for FHB resistance applied in some or all generations of inbreeding. Most programs begin selection for FHB resistance the first year after deriving pure lines ($F_4 - F_6$) and concurrently with first year yield testing. While most of these programs advance segregating populations to the pure line state without artificially exposing them to FHB epidemics, a few programs advance all or selected target populations in mist-irrigated and/or inoculated (natural or artificial) nurseries and either select and bulk resistant individuals or advance selected progeny using the pedigree method. Several programs using either the pedigree method or applying mass selection for FHB resistance during bulk population advancement select for plump seed via visual selection, sieving or using a gravity table. All programs conduct routine FHB screening of breeding materials, comprised predominantly of pure lines, entries in uniform FHB nurseries and official variety trials, parental lines, and select populations and/or early generation progeny, in 1 to 3 inoculated and mist-irrigated field nurseries. Half of the programs spray FHB field nurseries with conidial spore suspensions and the other half spread *Fusarium* colonized grain, primarily maize, as the primary inoculum source. Programs having FHB nurseries at multiple sites often plant these into maize stubble and rely on natural inoculum and/or epidemics. One program also evaluates Type II resistance in a field nursery using single floret inoculation and another program evaluates FHB resistance of lines via bagging spikes sprayed with a conidial spore suspension. All programs assess incidence, severity, and index in FHB field nurseries, and most assess at least their advanced lines evaluated in these nurseries for percentage of Fusarium Damaged Kernels (FDK), and DON toxin concentration. More than 70% of the programs routinely evaluate FHB resistance of parents, germplasm, advanced lines, and entries in uniform FHB nurseries in greenhouse tests, primarily for Type II resistance using single floret inoculation although a few programs also or only assess Type I resistance using a spike spray-inoculation method. One program also assesses Type II resistance of breeding populations in two greenhouse cycles each year. Several winter wheat breeding programs now conduct routine "in house" MAS or haplotyping of FHB resistance in a diverse assortment of materials and generations, including various stages of backcross progeny development, selection among F_1's derived from 3-way crosses, selection in early generations (F_2 and $F_{3)}$, selection and haplotyping of pure lines (F_4-F_7), and selection throughout the breeding process. A few programs lacking lab support for conducting MAS collaborate with the Genotyping Centers in this endeavor. While most programs applying MAS are routinely using markers for the 3BS QTL and many programs recently have begun using markers for the 5AS and other QTL, availability and use of markers for other validated unique FHB QTL are needed to accelerate progress. Additional information also is needed on haplotypes of FHB resistant parental sources, exotic and native, and for current and new FHB resistant wheat lines to further enhance breeding efforts. Capacity of breeding programs and Genotyping Centers to implement routine MAS on a large scale likely will impact the rate of future success.

STRESS-DIRECTED SELECTION IDENTIFIES LINES OF SPRING WHEAT WITH ENHANCED RESISTANCE TO FUSARIUM HEAD BLIGHT AND OTHER DISEASES

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ABSTRACT

A protocol of 'Stress-Directed Selection' (SDS, an iterative approach in which populations are subjected to repeated rounds of multiple disease pressures and combined resistances selected) identified spring wheat populations with improved FHB resistance. This approach has already succeeded in identifying lines with superior FHB resistance when one parent of the cross was a known FHB-resistance source. This work examines the application of SDS to populations derived from crosses of elite, FHB-susceptible parents. BC_1F_n progeny of the cross 'Superb'*[2]/ 'CO960293' (spring wheat , winter wheat, respectively) were subjected to SDS and selected under FHB pressure in alternating generations after BC_1 F_4. A small proportion of the lines showed unexpectedly good FHB resistance which was confirmed under controlled conditions. The progeny of these lines were subjected to SDS and about half the population was clearly FHB-resistant while the other half was as susceptible as the recurrent parent. Application of SDS to a selfing population of the breeding line c2652 (highly FHB-susceptible, but being examined as a possible new source of wheat streak mosaic virus resistance) identified lines with improved and apparently stably inherited FHB resistance.

MICROARRAY ANALYSIS OF FUSARIUM HEAD BLIGHT TOXIN DEOXYNIVALENOL (DON) REGULATED GENES OF *ARABIDOPSIS THALIANA*

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OBJECTIVES

This study analyzed gene expression in response to DON in Arabidopsis culture cells using cDNA microarray in order to understand the molecular mechanisms of response to DON activity in the plants and the role of DON in fungal pathogenesis.

INTRODUCTION

Gibberella zeae (Schwein.) Petch. (anamorphs, *Fusarium graminearum* and *F.culmorum*) is the primary producer of DON in cereal grains such as wheat, barley, and corn. The U.S Food and Drug Administration regulation requires that DON levels should not exceed 1ppm and 5 ppm in finished wheat products for human and animal consumption respectively (3). Many genes specifically induced in plants by pathogens and their metabolites play an important role in the defense response of plants. Toxins that interfere with the expression of genes (especially defense related genes) in plants may be considered as virulence factors. A strong correlation was found between *Fusarium* resistance and toxin resistance in the field and laboratory tests of a large number of wheat cultivars (4). The evidence from recent studies indicates that trichothecenes, including DON are involved in plant pathogenesis by enhancing the virulence of plant-pathogenic *Fusarium* species on cereal hosts (6). Deoxynivalenol (DON), a low-molecular-weight inhibitor of protein synthesis binds to the ribosomes and interfers with peptidyltransferase activity (7). Most of our knowledge about the effects of DON on the regulation of gene expression comes from the studies on animals or other microorganisms. DON is known to induces mitogen-activated protein kinases (MAPKs) including stress-activated protein kinases (SAPK/JNK1 and 2), extracellular signal regulated protein kinases (ERK (1/2) and p38MAPK in vitro and in vivo (8). However, little information is available about the activity of DON on the regulation of plant gene expression. DON was reported to inhibit ribosomal gene Rp13 expression in Rice (2). Here, we identified DON regulated Arabidopsis genes and analyzed their functions. Results showed that DON affects the expression of a wide spectrum of genes involved in responses of Arabidopsis plants to many environmental stimuli or stresses.

MATERIALS AND METHODS

DON treatment of Arabidopsis Suspension Cell Cultures and RNA Extraction - The Arabidopsis Columbia-0 suspension cells cultures were grown in Erlenmeyer flasks at room temperature, under ambient light, with constant shaking at 115-rpm in a rotating shaker. DON treatments at different time courses were started using suspension cells 3 d after subculturing, at an approximate cell density of 2 x 105 cells mL–1. Cultures were added with filtered and sterilized DON dissolved in ddH_2O in the amount of 5ug DON /mL. The same volume of sterile ddH_2O was added to the control. RNA extracts from suspension cells collected at 6 and 24 hours after addition of DON were used for microarray analysis. Total RNAs from suspension cells of Columbia wild type treated with DON and water were extracted with Promega RNAgents kit (Cat#Z5110) following the instructions by the manufacturer as recommended by Arabidopsis Functional Genomics Consortium. The RNA was further purified according to Qiagen RNAeasy midi kit protocol (75144).

Probe labeling and cDNA microarrays hybridizations - In each experiment, 100 ug of total RNA from two biological replicates from 6 and 24 hour following

DON treatment was labeled using Klenow Labeling as described (1). The labeling reactions were purified using the QiaQuick PCR cleanup kit (Qiagen). The experimental and control tissues labeled with either Cy3 or Cy5 Fluorescent dye were hybridized to microarray slides (MSU-2_03-00) containing 11,521 element prepared by the Arabidopsis Functional Genomics Consortium as described (1). Each pair of samples from the 6 and 24 hour time points were used in two microarray assays (Technical replicates). However the second replicate (same RNA) were reverse labeled relative to the first one. The slides were scanned to measure the fluorescence corresponding to hybridization intensities using the ScanArray 4000 (Packard BioChip technologies, Billerica MA).

Microarray data analysis - The intensities of the spots were measured using the Scananalyze V2.44 software (http:/rana.lbl.gov/EisenSoftware.htm). An initial normalization for standardizing Cy3 and Cy5 intensities on each of the 4 array was performed with Perl script program. The resulting data were transferred to Excel spread sheet files (Microsoft, Redmond, WA) and imported to microarray analysis software (GeneSpring 7.0; Silicon Genetics, Redwood City, CA, USA). The data were further normalized using per spot (signal channel divided by channel) and per chip (value of each spot divided by the 50 th percentile) intensity dependent normalization (Lowess) function of the GeneSpring 7.0 program. Expression ratios from two repeats were averaged, and genes showing a value above 2.0 or below 0.5 was regarded as induced or repressed, respectively. The Arabidopsis information Resource (TAIR) databases and tools were utilized to update and further analyze the microarray data (www.arabidopsis.org).

RESULTS AND DISCUSSION

The transcript levels of 272 and 480 genes were induced and repressed respectively after the exposure of the suspension cells to DON for 6 hours. Of 272 induced genes, 20 genes displayed minimum of 4 and up to a 25 fold increase in transcript levels. These highly induced transcripts include; a putative steroid sulfotransferase, an F-box family protein, four various types of AtPase proteins, an UDP-glucosyl transferase, an ankyrin repeat family protein, two protein kinase family protein, an ABC transporter family protein, a LEA domain-containing protein, an WWE domain-containing protein and a mitochondrial pentatricopeptide (PPR) repeat-containing protein. The Arabidopsis suspension cells quickly and strongly activated the genes that regulate growth, development and as well as defense response against the phytotoxic action of the toxin. Fourteen genes including a dehydrin family protein, a glutathione S-transferase, two tubulin alpha-2/alpha-4 chain (TUA4–TUA2) protein, an aspartyl protease, a sugar transporter family protein, a delta tonoplast integral transport protein were highly repressed following 6 hour toxin treatment (cut off value of less than 0.2). Exposure of the suspension cells to the toxin for 24 hour resulted in induction of 22 and repression of 35 new genes. The number of genes that remained induced and repressed at both time points were only 4 and 12 respectively.

The distribution of putative functions for 294 induced and 515 repressed genes among functional categories of the Arabidopsis Information Resource (TAIR) showed that they are involved in a broad range of biological processes of plants (Figure 1). However, the functions of large percentage of both induced (72.3 %) and repressed (73 %) genes have still unknown. The genes associated with transcription and transportation related processes were more abundant among induced transcripts than repressed whereas genes associated with electron transport/energy pathways, cell organization/biogenesis, protein metabolism, and stress and abiotic and biotic stimuli response functions were represented at higher proportions among repressed transcripts. Currently, only 7 (2.7 %) of DON induced and 50 (10%) of DON repressed genes can be placed into steps in known metabolic pathways. Interestingly, two DON induced genes are involved in sugar (trehalose) and one in amino acid (cysteine) biosynthesis pathways. Repressed genes catalyze diverse and multiple reactions in a broad range of metabolic pathways in the generation of precursor metabolites and energy (24 %), biosynthesize (36 %) and degradation/ assimilation/utilization (40%) processes of Arabidopsis plants.

DON resulted in induction of 15 and repression of 32 ribosomes and translation related genes that play important roles in protein synthesis. None of the 15 induced genes were ribosomal and none coded for ribosomal proteins however; they were all involved in protein synthesis associated ribosomal and translational activities. On the other hand, 22 out of 35 repressed transcripts were ribosomal genes and coded for ribosomal proteins. 50S, 40S and 30S ribosomal proteins were the most common products of toxin-repressed genes. DON strongly repressed the transcription of ribosomal genes that contributes to the structural integrity of ribosome and impeded the translation and protein synthesis mechanisms. The toxin differentially regulated the transcript levels of the genes that involved in the translation mechanism as translation factors at all levels.

DON induced 32 and repressed 19 genes that code for transcription factors in toxin-treated Arabidopsis culture cells. Most of them were affected within 6 hours of toxin treatment, strongly increasing the possibility that they are part of the regulatory circle that governs toxin response. Various types of 9 Zinc finger and 7 WRKY family protein, 2 AP2, 2 bZip and one disease resistance transcription factor genes constituted the majority of induced transcription regulators. Abundance of these defense related transcription factors suggest that regulatory mechanism controlling the toxin response may be similar to that of the defense response to invading pathogens in plants. Ethylene and auxin responsive protein genes were the most abundant transcription factors among the toxin repressed transcripts. The toxin regulated transcription factors is known to play essential roles extensively in defense response and molecular signaling along with growth and development of plants.

DON toxin caused the induction of 24 and suppression of 48 transcripts categorized as stress response genes by TAIR (Table 1). 14/24 induced transcripts were typical biotic stress genes involved in defense responses to various pathogens. Seven other induced genes are known to be reporters of abiotic stress while three genes function in general stress response. 8/14 defense response genes encode putative disease resistance proteins that all, except one, contains NBS-LRR motif and 2/14 unknown proteins that putatively play role in defense against pathogens. These plant resistance genes (R) play a primary role in detection of pathogen and initiation of specific defense response that includes a type of programmed cell death (apoptosis) known as hypersensitive response (HR) in plants (5). These findings suggest that DON triggers pathogen-like plant defense response and therefore must play an important role in pathogenicity of the fungus. The majority of DON-suppressed stress genes are involved in response to various environmental abiotic stress conditions such as drought, heat, cold, light, toxin and oxidation. Six genes including a pathogenesis-related (PR-1-like) transcript were also known to respond to biotic stresses. Analysis of stress genes shows that DON makes significant contribution to virulence and pathogenicity of the Fusarium head blight pathogen. Real-time quantitative PCR was performed to confirm the results of microarray expression assays for 8 induced and 10 repressed genes. The fold changes in transcript levels from Real-time PCR correlated with those from microarray assays for all the genes tested in regression analyses ($R^2 > 90$).

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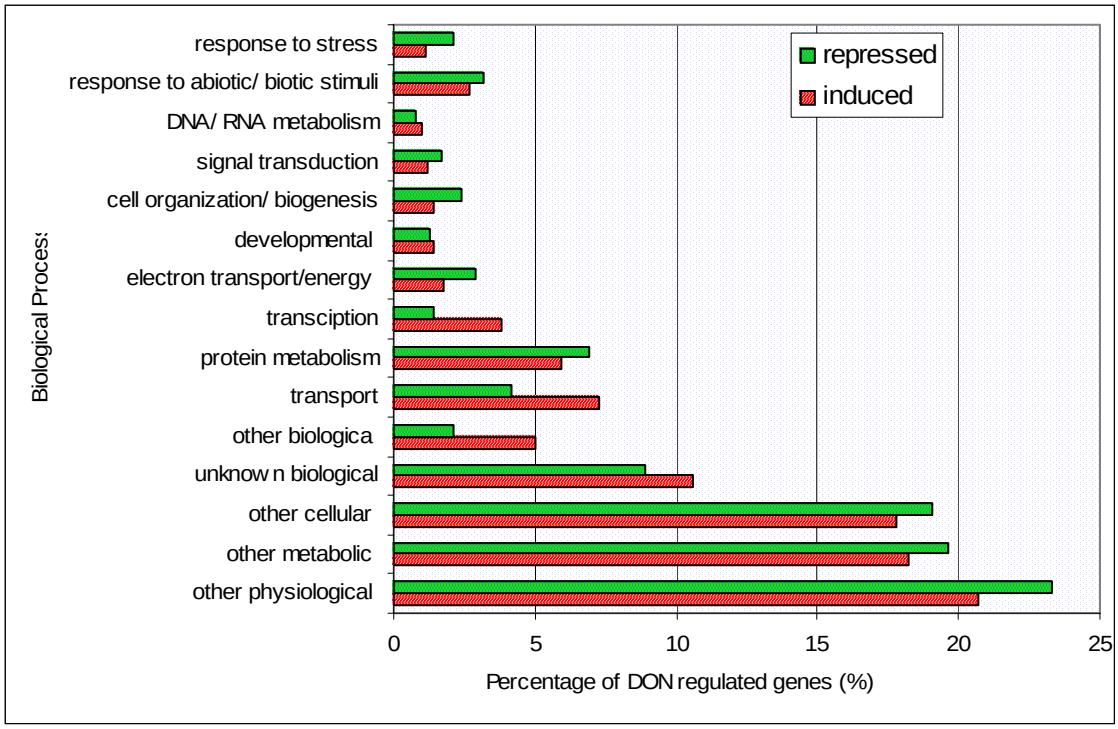

Fig 1. Molecular function and comparative analysis of the DON regulated genes. 294 induced and 515 repressed genes were grouped using the TAIR functional categorization of *Arabidopsis thaliana* genome.

Table 1. Stress (abiotic or biotic) related genes regulated by DON toxin.

Gene (AGI locus)	Description	Expr. [1]	Time (hour)	TAIR [2] Cat.	stress/response [3]
Induced					
At5g44510	disease resistance protein (TIR-NBS-LRR)	2.92	6	S, AB	pathogen/defense-AP
At4g33300	disease resistance protein (CC-NBS-LRR)	2.76	6	S, AB	pathogen/defense-AP
At2g21100	disease resistance-responsive protein	2.71	6	S, AB	pathogen/defense-fungi
At5g46520	disease resistance protein (TIR-NBS-LRR)	2.58	6	S, AB	pathogen/defense-AP
At5g46510	disease resistance protein (TIR-NBS-LRR)	2.55	6	S, AB	pathogen/defense-AP
At5g49140	disease resistance protein (TIR-NBS-LRR)	2.36	6	S, AB	pathogen/defense-AP
At1g72910	disease resistance protein (TIR-NBS-LRR)	2.41	6	AB	pathogen/defense
At3g44630	disease resistance protein (TIR-NBS-LRR)	2.59	6	AB	pathogen/defense
At2g03760*	steroid sulfotransferase-**ST**	25.33	6	AB	pathogen/defense
At3g54420*	class IV chitinase-**ATEP3**	3.03	6	S, AB	pathogen/defense- bacteria
At5g47120*	Bax inhibitor-1 putative-**ATBI-1**	2.01	6	AB	pathogen/defense-AP
At3g13950	expressed protein	3.33	6	S, AB	pathogen/defense
At5g57280	expressed protein	2.73	6	AB	pathogen/defense-bacteria
At1g32230*	WWE domain-containing protein-**RCD-1**	4.11	6	S, AB	pathogen/defense-bacteria-AP
At4g36990*	heat shock factor protein 4-**HSF4**	2.57	6	S, AB	heat
At5g49480*	Na-inducible Ca-binding protein-**ATCP1**	2.40	6	S, AB	salt,
At3g19580*	zinc finger (C2H2 type) protein 2-**AZF2**	2.14	6	S, AB	salt, water deprivation, ABA
At1g66340*	ethylene receptor 1-**EtR1**	2.05	6	S, AB	ethylene
At1g31480*	shoot gravitropism 2-**SgR2**	2.10	6	AB	gravitropism
At3g47340*	asparagine synthetase -**ASN1**	2.78	6	AB	light,sucrose
At5g20250*	raffinose synthase family protein-**DIN10**	3.19	6	AB	light, sucrose
At3g53990	universal stress protein (USP)	2.36	6	S	general stress
At5g01520	zinc finger (C3HC4-type RINg finger)	2.28	6	S	general stress
At1g59870	ABC transporter family protein	2.17	6	S	general stress
repressed					
At4g33680*	aminotransferase class I and II-**AGD2**	0.47	6	S, AB	pathogen/defense-SA
At4g22670	tetratricopeptide repeat(TPR) protein-	0.44	6	S, AB	pathogen/defense
At2g47730*	glutathione S-transferase 6-**ATGSTF8**	0.46	24	AB	pathogen/defense-toxin
At3g16770	AP2 domain-containing protein RAP2.3	0.42	6	S, AB	pathogen/defense, ethylene
At2g46370*	auxin-responsive GH3 family protein-**JAR1**	0.45	6	S, AB	pathogen/defense, auxin, JA
At2g19990*	pathogenesis-related protein-**PR-1-like**	0.25	6	NC	pathogen/defense
At4g15910*	drought-responsive protein-**ATDI21**	0.47	6	S, AB	water deprivation, ABA
At3g63520*	9-cis-epoxycarotenoid dioxygenase-**CCD1**	0.49	6	S, AB	water deprivation
At1g47128*	cysteine proteinase-**RD21A**	0.48	6	S, AB	water deprivation
At1g54410	dehydrin family protein	0.19	6	S, AB	water deprivation-gen.stress
At2g30870*	glutathione S-transferase-**ATGSTF8**	0.28	6	S, AB	water deprivation-toxin
At1g78360*	glutathione S-transferase-**ATGSTF8**	0.31	6	AB	toxin
At2g30860*	glutathione S-transferase-**ATGSTF9**	0.19	6	AB	toxin
At5g64120*	peroxidase	0.41	6	S, AB	oxidative stress
At4g21960	peroxidase 42 (PER42) (P42) (PRXR1)	0.49	24	S,AB	oxidative strees
At5g38000*	NADP-dependent oxidoreductase	0.48	6	S, AB	oxidative stress
At1g08830*	superoxide dismutase-**CSD1**	0.49	6	S, AB	oxidative stress
At1g19570*	dehydroascorbate reductase	0.12	6,24	S, AB	oxidative stress
At2g32120*	heat shock protein 70 family protein	0.47	6	S, AB	heat
At5g59720*	18.1kDa class I heat shock pro-**HSP18.2**	0.46	6	S, AB	heat
At1g54050*	17.4 kDa class III heat shock protein	0.41	6 ,24	S, AB	heat
At5g28540*	luminal binding protein 1 (BiP-1)	0.45	6	S, AB	heat
At5g42020*	luminal binding protein 2 (BiP-2) (BP2)	0.38	6	S, AB	heat
At5g62690*	tubulin beta-2/beta-3 chain-**TUB2**	0.45	6	S, AB	cold
At5g12250*	tubulin beta-6 chain-**TUB6**	0.43	6 ,24	S, AB	cold
At5g23860*	tubulin beta-8 chain-**TUB8**	0.34	6	S	cold
At5g62700*	tubulin beta-2/beta-3 chain-**TUB3**	0.29	6	S	cold
At5g63980*	3'(2'),5'-bisphosphate nucleotidase-**SAL1**	0.43	6	S, AB	cold
At5g09810*	actin 7 / actin 2-**ACT7**	0.44	6	S, AB	ligt, wounding
At4g34190*	stress enhanced protein 1-**SEP1**	0.44	6	AB	light
At1g60950*	ferredoxin, chloroplast (PETF)	0.41	6	AB	light
At3g19820*	cell elongation protein-**DWF-1**	0.36	6	AB	light
At3g07500	far-red impaired responsive protein	0.46	6	AB	light
At3g12610*	DNA-damage-repair/toleration pro-**DRT100**	0.41	6	AB	light, UV, chemical, drugs
At3g43810*	calmodulin-7-**CAM7**	0.34	6	AB	general abiotic-biotic
At5g13740	sugar transporter family protein	0.17	6	AB	general abiotic-biotic
At3g45780*	protein kinase-**PHOT1**	0.46	6	AB	general abiotic-biotic
At1g44575*	photosystem II 22kDa protein-**NPQ4**	0.42	6	AB	general abiotic-biotic
At3g07930	HhH-GPD base excision DNA repair	0.50	24	S	general stress
At2g12730	Mutator-like transposase family	0.49	24	S	general stress
At4g23940	FtsH protease	0.43	6	S	general stress
At3g22880*	meiotic recombination protein-**ATDMC1**	0.42	6	S	general stress
At3g17020	universal stress protein (USP)	0.34	6	S	general stress
At2g47590*	photolyase/blue light photoreceptor-**PHR2**	0.28	6	S	general stress
At1g20340*	plastocyanin (plastocyanin GI)-**DRT112**	0.42	6	AB	chemical
At5g05200	ABC1 family protein	0.46	6	AB	antibiotic
At4g23100*	glutamate-cysteine ligase-**RML1**	0.45	6	AB	cadmium ion
At2g27190*	iron(III)-zinc(II) purple acid phos-**PAP1**	0.41	6	S	phosphate starvation

[1]Fold changes in the transcript levels determined by GeneSpring 7.0 program. The genes with cut off values of above 2.0 and below 0.5 considered induced and repressed, respectively. [2]Functional categories of genes determined by TAIR; S-response to stress, AB-response to abiotic and biotic stimuli. [3]denotes stress that a gene respond. SA: Salycilic acid, JA: jasmonic acid ABA, Abscisic acid. *indicates the genes previously mentioned in the literature as corresponding stress responsive and available at TAIR homepage. Gene models are given in bold uppercase letters.

PROGRESS IN DEVELOPMENT OF RESISTANCE TO FHB IN ROMANIAN WHEAT

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ABSTRACT

Growing wheat cultivars that combine high levels of resistance to FHB (mainly Type II) and low contamination with DON, with other desirable agronomic traits, remains the most reliable strategy for control of scab in both, conventional and organic farming systems in Romania.

The damaging potential of this disease could be considerably high in years and locations characterized by high humidity at anthesis. That is why improvement of resistance to FHB is continuously an objective of major concern in the breeding research of winter wheat carried on in Romania, at ARDI Fundulea and other of its regional breeding centers.

Current goals:

i) Improvement of screening techniques;
ii) Selection of new sources that combine a higher resistance to FHB than that of *Fundulea 201R*, with resistance to other pathogens (e.g.*Tilletia* spp.) and better agronomic traits; and
iii) Introduction of MAS.

Screening - Due to the very complex inheritance of resistance and high genotype x environment interaction, our screening strategy is based on multi environment field experiments (year/location), under artificial inoculation (point/ single head method). For a better characterization of host resistance, at least two selected *Fusarium* isolates and combined pre and post-harvest criteria are used per genotype/year for inoculation and assessment, respectively.

Selection of *Fusarium* isolates according to their high aggressiveness and DON content (if available) is a prerequisite condition for more accuracy of assessment for resistance. A large range of variation for this trait has been reported in local *Fusarium* populations**.**

To avoid the misinterpretation of experimental field data, the classification of entries into groups of genotypes inoculated in the same day is recommended. Between the groups inoculated in different days, the sum of degrees calculated 48 hours before and 20 days post inoculation could be very informative, too.

It is necessary to emphasize also that ring trials for resistance to FHB, based on large national or/and international cooperation like: CIMMYT, USWBSI (Southern and Northern scab nurseries), European Fusarium Ringtest etc, could play a significant role in rationale and accelerated selection for more resistant and adapted winter wheat genotypes.

Selection for resistance. Trials performed in the last years evidentiated that a higher level of resistance to FHB than that identified in Fundulea 201R could be achieved. Contrary to this previous Romanian source of resis-

tance, not related with Chinese FHB resistant lines, the newest advanced lines have a better bread making quality and other desirable traits. Advanced lines as F 00628 G34-1, F 01461G3-2, F 99051G3-3INC2, F 01096G2-2, F01459G4-1 etc are derivatives of crosses bread wheat/ triticale and of bread wheats with complementary levels of resistance to FHB.

Markers assisted selection - The use of microsatellite markers associated with resistance to FHB has to validate in our research, the resistance derived from crosses among Romanian and Asian sources of resistance and to improve the use of MAS in breeding for this trait in winter wheat research. We currently use microsatellite markers associated with QTL's for resistance to FHB, located on chromosomes 3BS (*Xgwm 493, Xgwm 533*), 3A (*Xgwm 674*) and 5A (*Xgwm 304*) are in progress. The microsatellites markers *Xgwm 493* and *Xgwm* 533, linked with the major QTL *Qfhs.ndsu*-3BS from Sumai, have been already identified in some derivatives from crosses Sumai/Romanian lines.

These results represent a good premise for further approaches on resistance to FHB in winter wheat.

QTL MAPPING OF FUSARIUM HEAD BLIGHT RESISTANCE IN NOVEL WHEAT GERMPLASM CJ 9306

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ABSTRACT

Fusarium head blight (FHB or scab) caused by *Fusarium graminearum* is one of the most destructive diseases in wheat and barley. QTL mapping and marker-assisted selection enhance the efficiency of utilizing elite germplasms and breeding resistant cultivars. The objective of this study was to detect the DNA markers associated with the resistance in the novel wheat germplasm CJ 9306, which was developed through multiple-parent crossing and recurrent selection combined with modified pedigree methods with the aid of a dominant male-sterile gene (*Ta1*) at Nanjing Agricultural University, China (Jiang, 1997). A recombinant inbred population with 152 $F_{6:7}$ RILs derived from a cross Veery xCJ 9306 was phenotyped for resistance to fungal spread in greenhouse in 2002 and 2004 by single-floret inoculation. A total of about 680 SSR primer pairs (including *Xbarc, Xgwm* and *Xwmc* primers) were screened for polymorphism between the two parental lines. Polymorphic markers (about 170) were used to genotype the mapping population, and the segregating data were applied to construct a genetic linkage map using JoinMap version 3.0 and referring to a high-density linkage map (Shi and Ward, 2004). Preliminary results suggested three chromosome regions carrying QTLs associated with the resistance to fungal spread. A major QTL on 3BS (*Xgwm493-Xgwm533-2*) explained 40.3% of the phenotypic variation. Two additional QTLs on 5BL (*Xbarc74-Xbarc408*) and 2DL (*Xgwm157-Xgwm539*) explained separately more than 9% and 8% of the phenotypic variation. In total, these three QTLs could explain approximately 58% of the phenotypic variation. The genotyping data are in progress of further analysis.

RESISTANCE TO FUNGAL SPREAD AND DON ACCUMULATION OF *FUSARIUM GRAMINEARUM* IN WHEAT

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OBJECTIVES

1) To characterize the genetic variability of Type II resistance and DON contents in a RIL population derived from Veery/CJ 9306;

2) To estimate the association between the re sistance to fungal spread and DON accumu lation.

INTRODUCTION

Fusarium head blight caused by *Fusarium graminearum* is a world-wide important disease in wheat and barley. It caused devastating losses of crop production and severe toxin contamination of the grains in the North America in the last decade. The production and accumulation of the mycotoxin, deoxynivalenol (DON) in the infected kernels by the fungus is detrimental to the health of livestock and human beings. Characterization of the genetic variability of the resistance and DON accumulation is informative and helpful for development of resistant cultivars.

MATERIALS AND METHODS

A set of 152 recombinant inbred lines (RILs) derived from a cross Veery/CJ 9306 and two parents were evaluated for FHB resistance (Type II) in greenhouse in 2002 and 2004. The RILs were grown in a randomized design with two replications, and for each replication, six plants were planted in two pots. Two parents Veery and CJ 9306 were planted as controls many times at an interval of a week, in order that they could be included in each inoculation to estimate the differences among inoculation dates.

Single-floret inoculation was conducted immediately prior to or after initial anthesis (Jiang et al., 2001). The inoculumn was *F. graminearum* isolate PH-1 (NRRL 31084) for 2002 and a mixture of two isolates PH-1 and WF-1 for 2004. Twelve to fifteen microliters of conidospore suspension (5×10^4 spores/ml, produced by CMC liquid culture) was injected into a central basal floret of the spike with a self-refilling syringe. Six to eight spikes were inoculated per replication. The inoculated spikes were tagged to indicate the date of inoculation and record the symptoms of disease. The inoculated pots were placed in a misting chamber with an auto-mist-irrigation system programmed to deliver 20-second mist at intervals of 6 minutes and a temperature controlling system set at 22-26ºC. After mist-irrigation, pots were transferred to another greenhouse compartment. The number of scabby spikelets (NSS) on the inoculated spikes was visually counted at 5, 9, 13, 17, 21 and 25 days post inoculation (dpi), respectively. On the 25th day, the number of total spikelets and number of infected rachis sections (NIRS) were also estimated, and the percentage of scabby spikelets (PSS) was calculated for each observation. Using PSS data, the area under disease progress curve (AUDPC) was computed. Due to extremely high correlations between NSS or PSS and AUDPC or NIRS based on 2002 data, only NSS and PSS were determined at 21 and 25 dpi in 2004.

After all the plants were ripe, inoculated and non-inoculated spikes for each replication were harvested separately and threshed carefully with a head thresher at lower speed to avoid blowing the scabby or shriveled kernels away. Ten to twelve scabby kernels were randomly taken from the inoculated spikes to serve as a sample for Deoxynivalenol (DON) test. DON extraction and analysis were based on a modified

method of Mirocha et al. (1998). Briefly, seeds were weighed and placed into a 1-dram glass vial capped with a screw cap and extracted by soaking and shaking with 2 ml of acetonitrile/water (84/16 v/v) for 24 hr. The extract was passed through a minicolumn packed with C_{18} and aluminum oxide. One and a half milliliters of the filtrate were placed into a ½-dram glass vial and evaporated to dryness under nitrogen. Twenty-five microliters of TMS reagent (TMSI/TMCS 100:1) were added, and the vial was rotated so that the reagent contacted with all residue in the vial. The vial was placed on a shaker for 10 min, and then 200µl of isooctane were added followed by 200 µl of HPLC water to quench the reaction. After vortex, the upper layer was transferred to a GC vial. Selected ion monitoring (SIM) was used for GC/MS analysis (Shimadzu GCMS-QP2010, Shimadzu Corporation, Kyoto, Japan), with fragment ion (m/z value) of 235.10 as target ion and 259.10 and 422.10 as reference ions.

Statistical analysis was based on replication means for all the inoculated spikes within a replication. One-way ANOVA was computed first for single year data, and then two-way ANOVA based on two-year combined data was conducted to estimate the inter-year effect and genotype × year interaction. For PSS, due to a high consistency between the results of original observed values and arc-sin transformed values, the results based on original data were presented here. Broad-sense heritability was estimated on the basis of ANOVA results. Simple correlation was analyzed among the traits or indications of the resistance.

RESULTS AND DISCUSSION

All the indications of resistance to fugal spread (NSS, PSS, AUDP and NIRS) for the controls/parents were not significantly different for the date of inoculation for each year (data not shown), suggesting a high consistency among the different inoculations due to well-controlled environmental conditions.

One-year ANOVA showed that the differences among RILs were highly significant for all the indications of resistance and DON contents (Table 1). Two-year ANOVA also suggested a significant inter-year difference (F=6.21–13.52, P<0.001) and a significant genotype × year interaction (F=2.78–7.12, P<0.01) besides significant RILs difference (Table 2). In most cases, the averages of RIL population, with large variability, were around the mid-parent values. Frequency distributions were continuous and exhibited two or three peaks, except for NIRS (Figure 1). The results further supported our previous postulation that the resistance was inherited as a qualitative-quantitative trait (data to be published). Transgressive segregation was evident for all the indications, especially in the susceptible direction. Some lines were superior to CJ 9306 for NIRS and DON content.

The estimates of heritability suggested a higher broad-sense heritability for the resistance to FHB spread within the spikes (Tables 1 and 2). Comparatively speaking, the estimates of broad-sense heritability and coefficients of variation for 2004 were larger than those of 2002. Heritabilities based on two-year combined analysis were reduced due to removing genotype × year variance. Among different measures, DON content and NIRS had lower heritabilities and coefficients of variation, indicating that these two measures were more variable than the others.

Correlation analysis showed that there were extremely high correlations between different indications of the resistance to fungal spread (r=0.90-0.98, P<0.001) (Table 3). However, the correlation between DON content and spread resistance was moderate to higher (r=0.66-0.76, P<0.001). In addition, the correlation between years for DON content was obviously lower than for NSS and/or PSS, further indicating that DON accumulation was more easily affected by the environments. Different degrees of relationship between DON accumulation and resistance were addressed in previous studies: lower (Somers et al., 2003; Zhou et al., 2002), moderate (Bai et al., 2001; Mesterházy et al., 1999) and higher (Mesterházy et al., 1999). Clearly, the inconsistencies were attributed to DON sampling and analysis methods as well as the types and numbers of experimental materials.

ACKNOWLEDGEMENT

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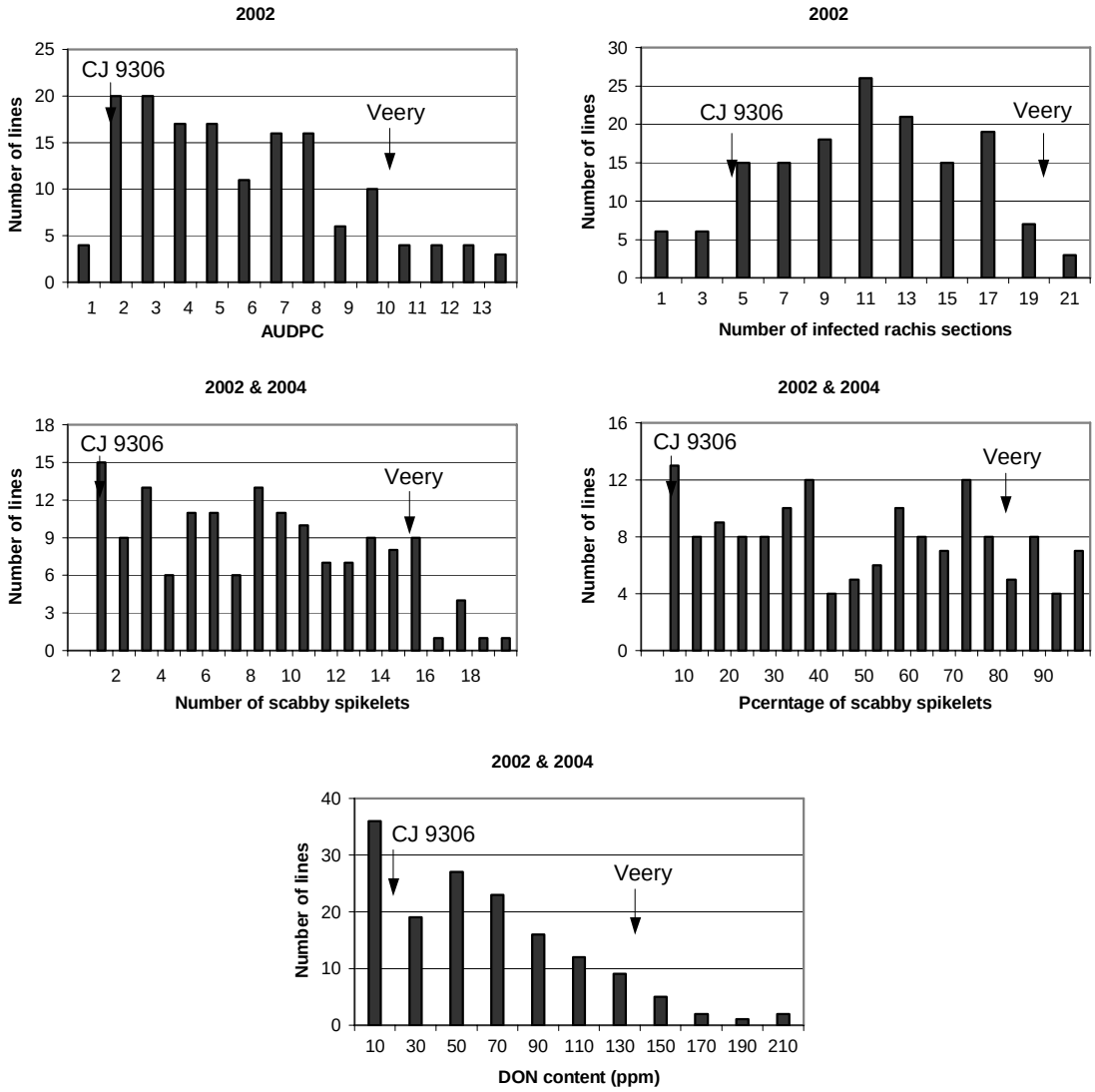

Fig. 1. Frequency distribution of 152 RILs derived from the cross Veery/CJ 9306 for resistance to fugal spread and DON accumulation of *Fusarium graminearum* by single-floret inoculation in greenhouse.

Table 1. Means, variations, *F*-values and estimates of broad-sense heritability of FHB scores and DON content in a 152-RIL population by greenhouse-based single-floret inoculation in two years (2002 and 2004).

Population	Statistics	NSS		PSS (%)		DON content (ppm)		AUDPC	NIRS
		2002	2004	2002	2004	2002	2004	2002	2002
CJ 9306	Mean	1.5±0.2	0.8±0.1	8.3±1.3	4.6±0.5	22.7±3.1	7.7±4.1	1.2±0.2	4.4±1.2
	Range	0.7–2.5	0.5–1.4	4.0–13.9	2.8–7.1	18.5–28.7	0–17.1	0.5–1.7	0.7–9.5
Veery	Mean	16.5±0.5	14.1±0.3	90.2±1.7	76.3±1.4	134.2±15.7	134.1±20.6	9.9±0.6	19.4±0.4
	Range	15.4–19.2	12.3–15.2	83.9–96.2	68.1–81.6	94.7–165.3	84.0–182.9	7.9–12.3	18–21.3
RIL population	Mean	8.7±0.4	8.2±0.4	51.0±2.4	47.4±2.4	65.0±4.4	58.9±4.4	5.4±0.3	11.1±0.4
	Range	0.7–20.2	0.6–20.7	4.0–100	3.4–100	0–265.3	0.2–274.8	0.5–13.8	0.9–22.2
	CV %	58.03	63.58	57.08	62.34	81.62	91.98	60.01	44.32
	F-value	8.04 **	13.99 **	8.13 **	14.95 **	2.98 **	13.13 **	8.79 **	5.60 **
	h_B^2	77.88	86.66	78.10	87.46	49.80	85.85	79.57	69.70

** Significant at the 0.01 probability level on the basis of one-way ANOVA.

Table 2. Means, variations, *F*-values and estimates of broad-sense heritability of the resistance to fugal spread and DON accumulation in a 152 RIL population based on a two-year combined analysis under single-floret inoculation.

Population	Statistics	NSS	PSS (%)	DON content (ppm)
CJ 9306	Mean	1.2±0.2	6.2±0.8	14.1±3.9
Veery	Mean	15.2±0.4	82.8±2.1	134.2±12.0
RIL population	Mean	8.5±0.4	49.2±2.2	62.1±3.8
	Range	1.1–19.5	6.1–100	0.1–235.6
	CV %	56.61	56.02	75.60
	F-value	6.63 **	7.12 **	2.79 **
	h_B^2	67.30	68.74	40.71

** Significant at the 0.01 probability level on the basis of two-way ANOVA.

Table 3. Correlation coefficients between FHB scores and DON content in a 152-RIL population by single-floret inoculation for single year (above diagonal: upper for 2002 and lower for 2004) and two-year combination (below diagonal), and correlation coefficients between years for the same trait (on diagonal and bolded).

	NSS	PSS	DON content	AUDPC	NIRS
NSS	**0.738** ***	0.978 ***	0.670 ***	0.948 ***	0.959 ***
		0.978 ***	0.672 ***		
PSS	0.979 ***	**0.766** ***	0.656 ***	0.967 ***	0.926 ***
			0.698 ***		
DON content	0.763 ***	0.759 ***	**0.448** ***	0.655 ***	0.657 ***
AUDPC					0.895 ***

*** Significant at the 0.001 probability level.

MARKER ASSISTED SELECTION FOR FUSARIUM HEAD BLIGHT RESISTANCE IN SOFT RED WHEAT FROM DOUBLE HAPLOID POPULATIONS

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ABSTRACT

Fusarium head blight (FHB) is caused by *Fusarium graminearum* Schwabe. The fungus can degenerate the wheat grain tissue and produce deoxynivalenol (DON) which is toxic to both human and animals. Epidemics of FHB can result in severe loss of yield and grain quality. Cultural or/and chemical control of FHB is difficult because infection of FHB occurs during the time of flowering. Chemical control could only be 60-70% effective when applied at the optimum time. Release of FHB resistant cultivars is the most effective option to control incidence of FHB. In the southeast region of the US, resistance to FHB in local adaptive soft red winter wheat is limited. Introduction of resistant genes from exotic sources could enhance the resistance of local adaptive germplasm. A Virginia line AV01W-476 with the most widely used major QTLs in chromosome 2A, 3B and 5A for FHB resistance was used as donor in our program. A total of 47 double haploid individuals were generated from backcross F1 plants induced with maize pollens. Screening with SSR markers indicated the integration of novel FHB resistant QTLs on 3BS and 5AL from donor parents and native adaptive gene pool of ASG2000 and its derivatives. Two double haploid plants from back-cross of VA01W-476/GA98186 and 4 double haploid plants from back-cross of VA01W-476/AGS2485 were identified to have VA01W-476/W14 type QTLs on 3BS and 5A. Further evaluation for agronomic traits is under investigation.

PROGRESS FROM FIVE YEARS OF SELECTING FOR RESISTANCE TO FUSARIUM HEAD BLIGHT IN SPRING WHEAT

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ABSTRACT

Some effort aimed at the improvement of resistance to Fusarium Head Blight (FHB) has been practiced within spring wheat breeding programs for many years. With the advent of the US Wheat & Barley Scab Initiative, however, such efforts have become major resource expenditures. As such, it seems worthwhile to periodically monitor progress from such endeavors. In our first attempt to gauge progress, the objective of this project was to determine whether resistance to FHB in a random sample of spring wheat germplasm selected in the upper Midwest has increased from 1998 to 2003. To facilitate such measurement, a test was composed which included 10 varieties that were released to growers between 1998 and 2003, as well as 24 breeding lines that were selected within the same time frame to continue their advancement through their respective breeding program (i.e., advanced to statewide preliminary yield trials). Two additional lines were included in the test as checks. These artificially inoculated tests were grown under mist-irrigation at Brookings, SD and Prosper, ND during the 2004 and 2005 growing seasons. *Fusarium* Head Blight severity data were collected from each four-replication test. Data were subjected to analysis of variance over years and over locations. It was found that entries were significantly different with respect to FHB severity at both locations. Analysis of severity ratings at each location over years revealed that years were significantly different in terms of severity in Brookings, but not at Prosper. The correlation coefficient for overall severity ratings among locations was highly significant ($r=0.7769$; $p=<.0001$). Year of advancement and entry means were used as independent and dependant variables, respectively, to fit a simple regression model. The slope of the simple regression line was not statistically separable from zero ($b=-0.4537$; $p=0.363$). These results suggest that much phenotypic variation for FHB severity is present within this germplasm. At the same time, it appears that the entries sampled from the 5-year time span are still too variable, with respect to FHB severity, to begin monitoring progress in the advancement of FHB resistance. Additional attempts to examine progress in FHB resistance breeding will likely be initiated in the future.

A COMPARISON OF TYPE I AND TYPE II RESISTANCE WITHIN A COLLECTION OF ELITE SPRING WHEAT GENOTYPES

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ABSTRACT

Within the SDSU Spring Wheat breeding program, FHB resistant genotypes are selected based solely on Type I resistance. Implications associated with selecting for only Type I resistance could be negative. Specifically, if independently expressed genes control the two modes of resistance, (as has been suggested) chance alone would dictate whether our selections with high levels of Type I resistance possess similarly high levels of Type II resistance. Little information exists with respect to the genetic control of Type I and Type II resistance. Our objective was to examine associations between Type I and Type II resistance measurements on an elite collection of highly inbred spring wheat germplasm. Two split-plot greenhouse experiments were carried out on seventeen genotypes. Experiment one was conducted on a greenhouse bench where experimental units were a pot of plants. Experiment two consisted of hill plots that were sown into the soil beds of another greenhouse. Two inoculation methods (single floret injection vs. whole head spray) served as the main-plot treatments that were applied to seven replications of the sub-plot treatments (genotypes) within each experiment. Environmental conditions prior to and after inoculations were consistent with our routine procedures that have been optimized for disease expression. Eighteen days after inoculation, the number of diseased spikelets/head, total number of spikelets/head, and disease severity estimates were recorded for plants within each experimental unit. Data collected from the first experiment revealed that genotypic means were significantly different for total number of diseased spikelets in addition to disease incidence and severity values regardless of the inoculation method. Significant tests of Spearman's rank-order correlation suggested that Type I resistance measures were generally similar to those attained from Type II measures, except in the case of diseased spikelets/head. Additional research must be conducted with regard to our current objective, however, it appears as though severity measures collected from spray inoculations, (Type I resistance) for example, provide similar severity data as obtained from point inoculations (Type II resistance). Although it is currently not possible to speculate on a reason for this observation, our findings provide some evidence that Type I and Type II resistances are not completely independent. Results from our second experiment will also be discussed.

IMPROVEMENT OF FHB RESISTANCE OF DURUM WHEAT

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ABSTRACT

The greatest challenge in the development of FHB resistant durum wheat (*Triticum durum*) is the extreme susceptibility of durum wheats to FHB. There are two explanations for this extreme susceptibility: 1) the inherent lack of resistance genes, and 2) the presence of super-susceptibility factors. In light of these two possibilities we are using the following approaches to insure the development of FHB resistant durum wheat. We are systematically screening durum accessions in CIMMYT GeneBank for FHB resistance. In addition, we are screening alternative sources of resistance to incorporate into durum wheat. Multiple alternative sources of resistance exist for the enhancement of durum wheat including:

1) Wild tetraploid wheat having the A and B genomes.
In a preliminary study, we found several resistance lines of *T. dicoccum* and *T. dicoccoides* show ing as few as 7% scabby spikelets when screened for type II resistance.

2) A/B genome of hexaploid wheat.
The transfer of 'Sumai #3' resistance from the 3B chromosome to durum is underway using PCR markers.

3) Ancestral species of the A and B genomes.
We have observed *T. monococcum* accessions having type II resistance responses ranging from 9.4% to 45.7% scabby spikelets. CIMMYT has produced more than 200 lines ofsynthetic wheat of A and B genomes (genome constitution=AAAABB and AABBBB), and there are several Resistance candidates where the type II resistance scores are as minimal as 9.5% scabby spikelets.

4) D genome of hexaploid/synthetic wheat.
Several synthetic (genome constitution AABBDD) wheat incorporating the D genome of *Aegilops tauschii* showed type II resistance equal to or higher than that of Sumai #3. The resistance exhib ited by these synthetic wheats is supposedly residing in the D genome. We crossed synthetic wheats with *ph1* mutant lines to transfer such resistance into durum through recombination between the A and D genomes.

In addition to utilizing alternative sources of resistance, we are removing susceptibility factors from durum wheat. We have generated a cross between a durum wheat and Sumai #3. From this cross, we will develop four kinds of populations: (1) Plants having *T. aestivum* morphology and Sumai #3 resistance(2) Plants having *T. aestivum* morphology and *T. durum* susceptibility, 3) Plants having *T. durum* morphology and Sumai #3 resistance, and 4) plants having *T. durum* morphology and susceptibility. We will create a mapping population from a cross between a resistant durum-like plant and a susceptible durum-like plant to identify and analyze the factors contributing to susceptibility.

EVALUATION OF FUSARIUM HEAD BLIGHT RESISTANCE IN SOFT RED WINTER WHEAT

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ABSTRACT

In 2005, Kentucky producers experienced exceptional yields resulting from an extremely favorable environment for wheat production and essentially no FHB pressure. Although there was very little natural disease development throughout the state, the irrigated, scabby-corn inoculated FHB nursery at Lexington, KY produced sufficient disease pressure that breeding lines could be screened for resistance. The difficulty in controlling inoculum level in the irrigated nursery in previous years prompted the addition of an alternative screening method: non-irrigated hill plots inoculated with a conidial spore suspension. Advanced breeding lines were evaluated for resistance in the irrigated FHB nursery by recording severity at 21 days after anthesis. Selected breeding lines from the irrigated FHB nursery were also evaluated in the non-irrigated hill plots. Ten seeds of each line were planted into two non-irrigated hill plots at Lexington, KY. One hill plot was sprayed at anthesis with a conidial suspension (50,000 spores μl^{-1}) until runoff and bagged with a corn shoot glassine bag for 72 hours. The second hill was used as a control and sprayed with water. Twenty-one days after inoculation the hills were rated for disease severity. Disease severity in the nursery ranged from 6 to 93%; the average of the entire nursery was 40%. This is a significant ($P<0.05$) difference from 2004 where resistant and susceptible breeding lines could not be distinguished, the range in disease severity for lines was 17 to 96% and the average severity of the entire nursery was 47%. Breeding lines included in both the irrigated FHB nursery and the non-irrigated hill plots were analyzed to determine if differences existed between average severities and incidence. The non-irrigated hill plots had a significantly ($P<0.05$) higher severity (62%) than the irrigated FHB nursery (46%). However, the irrigated nursery had a significantly ($P<0.05$) higher incidence (64%) than the non-irrigated hill plots (53%). Results from spray-inoculated spikes in the hill plots were promising. Although severity and incidence were not as consistent in the hill plots, modifications to the inoculation procedure and spore concentration can be made to produce more uniform results. This could be useful in testing advanced breeding lines in different locations across Kentucky or in years in which the irrigated nursery produces such high levels of inoculum that differentiation of resistant and susceptible lines is impossible. By simultaneously testing lines in both environments, a better estimation of resistance can be made.

PROFILING THE EXPRESSION OF GENES RELATED TO FHB PATHOGENESIS IN WHEAT WITH AFFYMETRIX GENECHIP WHEAT GENOME ARRAY

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ABSTRACT

FHB-resistant cultivars Sumai 3, Tokai 66 and Abura and FHB-susceptible landrace Y1193-6 were inoculated with *Fusarium graminearum* isolate Fg4 and water (as the mock-inoculated controls). Gene expression in the inoculated and the mock-inoculated samples was profiled with Affymetrix GeneChip Wheat Genome Array 24 hours after inoculation and was verified with Real-Time RT-PCR assay. Comparing the inoculated with their mock-inoculated controls revealed FHB-related gene expressions with a threshold of one fold difference. Some FHB-related gene expressions were cultivar-specific and were associated with FHB resistance. Also observed were *F. graminearum* genes that might only express after infection.

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NEW DNA MARKERS FOR THE CHROMOSOME 3BS FUSARIUM HEAD BLIGHT RESISTANCE QTL IN WHEAT

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ABSTRACT

A major quantitative trait locus (QTL), *Qfhs.ndsu-3BS*, for resistance to Fusarium head blight (FHB) in wheat has been identified and verified by several research groups. The objectives of this study were to construct a fine genetic map of this QTL region and to identify new DNA markers useful for marker-assisted selection (MAS) for this QTL. Two SSR markers (Xgwm533 and Xgwm493) flanking this QTL were used to screen for recombinants in a population of 3156 plants derived from a single F_7 plant heterozygous for the *Qfhs.ndsu-3BS region*. A total of 382 recombinants were identified, and they were genotyped with two more SSR markers and nine STS (sequence-tagged site) markers. A fine genetic map of the *Qfhs.ndsu-3BS* region was constructed and spanned 6.3 cM. Based on replicated evaluations of homozygous recombinant lines for Type II FHB resistance, *Qfhs.ndsu-3BS*, re-designated as *Fhb1*, was placed into a 1.2 cM marker interval flanked by STS3B-189 and STS3B-206. Assuming *Fhb1* is located in the middle of the marker interval, STS3B-256 is within 0.2 cM of *Fhb1* compared to the closest SSR markers, BARC147 and BARC133 that are approximately 0.8 and 1.0 cM from this gene, respectively. Marker STS3B-256 amplifies three loci on chromosomes 3A, 3B and 3D from Chinese Spring. Sumai 3 has a null 3BS allele for this marker. The 75 wheat lines used in our previous haplotype study (*Crop Science* 43:760-766) and 144 parental lines used in the wheat breeding program at the University of Minnesota were genotyped with marker STS3B-256. All wheat lines most likely containing *Fhb1* based on their SSR marker alleles have the null 3B allele. Among the 48 lines with all SSR alleles near *Fhb1* different from Sumai 3, only two lines, Wangshuibai and Ning894013, have the null 3B allele. Surprisingly, four of the seven lines that share only the BARC133 allele with Sumai 3 have the null 3B allele. All the four lines, Nobeoka Bozu, Nyu Bai, Abura, and Tokai 66, were originated from Japan and are well known for FHB resistance. Among the 144 parental lines genotyped with marker STS3B-256, only 20 lines have the null 3B allele, and they all have Sumai 3 or Nyu Bai in their pedigrees. Therefore, we believe the null allele of STS3B-256 is diagnostic for the Sumai 3 allele of *Fhb1* and will be useful in MAS.

MAIN EFFECTS, EPISTASIS AND ENVIRONMENTAL INTERACTIONS OF QTLS ON FUSARIUM HEAD BLIGHT RESISTANCE IN A RECOMBINANT INBRED POPULATION CS-SM3-7ADS /ANNONG 8455

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ABSTRACT

Chinese Spring-Sumai 3 chromosome 7A disomic substitution line (CS-SM3-7ADS) was reported to have a high level of FHB resistance, and an F_7 population of recombinant inbred lines (RILs) derived from the cross between CS-SM3-7ADS and Annong 8455 was evaluated for resistance to Fusarium head blight (FHB) to identify main effects, epistasis and environmental interactions of QTLs on FHB resistance. A molecular linkage map was constructed with 501 simple sequence repeat (SSR) and amplified fragment length polymorphism (AFLP) markers. The map covered a genetic distance of 2546 cM. Ten QTL were identified with significant main effects on the FHB resistance in at least one environment using MapQTL and QTLMapper software. Among them, CS-SM3-7ADS carries FHB-resistance alleles at five QTLs on chromosomes 2D, 3B, 4D and 6A. The QTL on chromosome 3BS has the largest effect on FHB resistance and explained 30.2% of the phenotypic variance when data from two locations were analyzed together. QTL was not detected on chromosome 7A that was from Sumai 3 and therefore the increased FHB resistance in CS-SM3-7ADS may not be due to a major resistance QTL on 7A of Sumai 3, but more likely due to removal of a susceptible QTL on 7A of Chinese Spring. QTLMaper detected nine pairs of additive-by-additive (AA) interactions at 17 loci that explained 26% phenotypic variance. QTL-by-environment (QE) interactions explained about 49% of phenotypic variation, indicating that the expression of the QTLs are significantly affected by the environments and multiple location tests are important for identification of stable QTL.

FRACTIONAL ANALYSIS OF CHROMOSOME 2(2H) FUSARIUM HEAD BLIGHT RESISTANCE QTL

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ABSTRACT

Two quantitative trait loci (QTL) each for lower Fusarium head blight (FHB) severity and plant height, and one major QTL each for deoxynivalenol (DON) accumulation and days to heading were mapped with recombinant inbred lines obtained from a cross between CIho 4196, a two-rowed resistant cultivar, and Foster, a six-rowed susceptible cultivar (Horsley et al., 2005). These loci reside in the barley chr. 2H region flanked by the markers ABG306 and MWG882A (bins 8-10). To date, there are a total of 26 unique loci and 67 markers in this major FHB QTL region on chr. 2. Eighteen markers have been hybridized to the 6x cv. Morex barley BAC library, identifying 131 BAC clones as part of the physical map of the region. Since the region is very large, we are using several different approaches to break down the FHB QTL to target necessary genes for resistance, and separate FHB resistance from undesirable traits to develop genetic and breeding material. Three cleaved amplified polymorphic sequence (CAPS) markers were designed for the major FHB resistance QTL to aid in development of isolines. Seventy-©four lines containing fragments of this region from CIho 4196 in a six-row susceptible cultivar, Morex, background were selected. These lines are being genotyped more extensively and submitted for phenotyping in China. Genotyping data will be presented. A Foster x CIho 4196 inbred recombinant, A171, is of special interest. It has Foster genotype throughout the proximal region of the major FHB QTL through the *Vrs1* locus, resulting in a six-rowed phenotype. However, it maintains low FBH severity, low DON accumulation, is tall, and has a late heading date, traits of CIho 4196. To facilitate development of six-rowed germplasm for breeding and identification of genes that affect these traits, A171 was backcrossed to Morex and CIho 4196. A BC1F2 population was produced summer '05 and will be screened for recombinants with desirable resistance and agronomic traits. A BC2 population was also produced and will be selected with molecular markers for additional recombinants.

In order to develop molecular markers very closely linked to the *Vrs1* locus, we initiated microarray analysis of nine six-rowed *Vrs1* mutants obtained by fast neutron mutagenesis of three two-rowed cultivars. Since these mutants were induced by fast neutrons, they are likely to have very large and possibly overlapping deletions. Such deletions can be quickly identified and genes involved in the deletions used as markers for mapping. Markers closely linked to the *Vrs1* locus are needed to identify crossovers near the *Vrs1* locus. These markers will be used to aid in breeding and identification of FHB resistance genes.

DIALLEL ANALYSIS OF FUSARIUM HEAD BLIGHT RESISTANCE IN WHEAT

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ABSTRACT

Fusarium head blight (FHB), caused by *Fusarium graminearum*, is a major biotic constraint of winter wheat production and quality in South Dakota. Breeding of resistant varieties is the most efficient approach for combating this problem. This study was performed to investigate genetic control of FHB resistance in selected winter and spring wheat genotypes. A partial diallel mating design included crosses from parents 'Nekota', '2137', 'Harding', 'ND 2710', 'BacUp' and 'Ning 7840'. Both F_1 and F_2 populations were evaluated in the greenhouse, and only F_2 populations were evaluated in the field. In the greenhouse, both F_1 and F_2 populations were artificially point inoculated at anthesis, whereas F_2 crosses evaluated under field conditions were artificially inoculated by a combination of corn spawn spread at jointing stage and inoculum suspension spray at anthesis. Disease index percentage (incidence%*severity%/100) of the crosses was analyzed using Griffing's method 4 and model 1. General combining ability (GCA) was highly significant ($P < 0.01$) in both greenhouse and field environments, but specific combining ability (SCA) was significant ($P < 0.05$) only in the F_2 crosses in the greenhouse. The ratio of combining ability variance components [$2ó^2GCA/(2ó^2GCA+ó^2SCA)$] ranged from 0.66 to 0.89. A high correlation ($r = 0.95$, $P < 0.01$) was observed for disease index between F_2 populations in the greenhouse and field environments. Deoxynivalenol (DON) content was analyzed with GC/ECD in the populations under greenhouse conditions. The DON content ranged from <0.5 to 27.2 ppm. A positive and significant correlation in the F_1 ($r = 0.71$; $P < 0.01$) and the F_2 ($r = 0.84$; $P < 0.001$) populations was found between disease index and DON content. The results showed that both additive and non-additive gene effects were involved in the inheritance of FHB resistance but additive gene effects were more important than non-additive gene effects. A significant and high correlation between disease index and DON content indicates that selecting for low disease index would be useful to indirectly select for low DON content.

EVALUATION OF ELITE HARD RED AND WHITE WINTER WHEAT FOR FUSARIUM HEAD BLIGHT RESISTANCE

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ABSTRACT

This study was performed to investigate genetic diversity for FHB resistance in selected advanced winter wheat genotypes. A total of 22 hard red and white winter wheat genotypes representing Crop Performance Testing (CPT) Variety Trial were evaluated for FHB resistance in a mist-irrigated field in 2003 and 2004 and in the greenhouse in 2003. In the field environment, genotypes varied significantly ($P < 0.01$) for flowering date, disease index and percent *Fusarium* damaged kernel (FDK) in both years. Flowering date was correlated with disease index but not with percent FDK. There was no correlation between disease index and percent FDK. This indicated that the disease index and percent FDK should be recorded separately to assess FHB resistance. The disease index and percent FDK varied significantly ($P < 0.01$) between the years. The interaction was also significant ($P < 0.05$) between year and genotype for disease index and percent FDK. Deoxynivalenol (DON) was measured in the genotypes evaluated in the field environment in 2004. The DON content ranged from 21.6 ppm to 52.4 ppm. High DON content demonstrated that these genotypes were not resistant to mycotoxin. Nivalenol accumulation was low (<0.5 ppm) in all the genotypes. The DON content was correlated ($r = 0.6$ and 0.5; $P < 0.05$) with both percent FDK and disease index, respectively. In the greenhouse, genotypes also varied significantly ($P < 0.01$) for disease index. No correlation was observed between disease index in the greenhouse and the field. The interaction effect between year and genotype in the field showed that environment changed ranking of genotypes in the two years. This suggests the need for multiple evaluations of germplasm for disease resistance. The correlations between disease index or FDK and DON content indicated that selecting for low disease index or FDK would also indirectly lead to low DON content. Since there was no correlation between the greenhouse and field environments, disease screening in the field cannot be replaced with greenhouse evaluation.

MOLECULAR CHARACTERIZATION OF WHEAT-ALIEN SPECIES AMPHIPLOIDS AND CHROMOSOME ADDITION LINES RESISTANT TO FUSARIUM HEAD BLIGHT

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ABSTRACT

Four *Triticum aestivum* L. cv. 'Fukuhokomuji (Fuku)'- *Elymus rectisetus* (2n = 6x = 42, genomes StStYYWW) chromosome addition lines (2n = 44), seven *T. aestivum* L. cv. 'Chinese Spring (CS)'- *Thinopyrum junceum* (2n = 6X = 42, genomes $E^bE^bE^bE^bE^eE^e$) amphiploids, and thirteen CS-*Th. junceum* addition lines (2n = 44) were evaluated for Type II resistance to Fusarium head blight (FHB). One Fuku-*E. rectisetus* addition line, A1034, three amphiploids,AJAP3, AJAP4, AJAP7, and two CS-*Th. junceum* addition lines, AJDAj2 and AJDAj3 exhibited resistance comparable to the resistant control 'Sumai 3'. The mean FHB severity of these resistant lines was significantly lower than the susceptible controls CS and Russ, a common spring wheat cultivar. RFLP analysis indicated that the *E. rectisetus* chromosome in three of the four Fuku-*E. rectisetus* addition lines, A1026, A1048, and A1057, belonged to homoeologous group 1. In addition, *Th. junceum* chromosomes belonging to homoeologous groups 1, 2, and 4 were identified through RFLP analysis in the CS-*Th. junceum* addition lines, including AJDAj1, AJDAj2, AJDAj3, AJDAj4, AJDAj8, and HD3515. Characterization of low molecular weight glutenin subunits in the seeds by sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) confirmed the RFLP results for the addition lines carrying a group 1 chromosome of *E. rectisetus* or *Th. junceum*. Chromosomes in the resistant amphiploids and addition are being further characterized using fluorescence in situ hybridization (FISH). These resistant lines could serve as a novel source of FHB resistance for wheat breeding. Understanding of their genetic constitutions will enhance the utilization of these resistance sources in the development of wheat varieties resistant to this devastating disease.

THE EFFECT OF GENERAL FIELD SELECTION ON WHEAT MICROSATELLITE ALLELE FREQUENCIES AT FHB RESISTANCE QTLS

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ABSTRACT

The development of FHB resistant cultivars is an important objective of Canadian common wheat breeding programs. The objective of this study was to determine whether selection for agronomic and disease response traits affected microsatellite allele frequencies at FHB resistance quantitative trait loci (QTLs). FHB resistance QTLs, derived from Nyubai, Sumai 3, and Wuhan 1, were backcrossed into three elite western Canadian spring wheat backgrounds, 98B69-L47, BW301, and Kanata, using marker-assisted selection. Eight hundred and one doubled haploid (DH) lines were produced from all possible cross combinations among BC_2F_2 lines derived from these three backgrounds. The DH lines were selected for plant type, plant height, lodging, and time to maturity in New Zealand, and response to leaf rust, stem rust, common bunt, and FHB in nurseries near Swift Current, SK, and Carman, MB. All DH lines were analyzed with microsatellite markers at FHB resistance QTLs on chromosomes 2D, 3BS, 3BSc, 4B, and 5AS. Microsatellite allele frequency changes were not significant at most loci. Selection resulted in a moderate but significant reduction in Nyubai and Sumai 3 alleles at 5AS microsatellite loci. The Wuhan 1 allele at *Xgwm608-2D* was fixed in one population. In general, selection for general field performance and response to leaf and stem rust and common bunt did not adversely affect variation at FHB resistance QTLs in the populations evaluated, except for 5AS.

THE EVALUATION OF FHB RESISTANCE QTLS INTROGRESSED INTO ELITE CANADIAN COMMON WHEAT GERMPLASM

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ABSTRACT

The effects of Fusarium head blight (FHB) resistance quantitative trait loci (QTLs), from Nyubai, Sumai 3, and Wuhan 1, were evaluated in three elite Canadian spring wheat backgrounds, 98B69-L47 (QTLs: 3BS, 3BSc, 4B), BW301 (QTLs: 2D, 3BS, 5AS), and Kanata (QTLs: 3BSc, 5AS). Microsatellite loci, linked to these QTLs, were analyzed to identify homozygous BC_2F_2 individuals. BC_2F_3 and BC_2F_4 lines were evaluated for anthesis date, plant height, FHB incidence, FHB severity, FHB index, Fusarium damaged kernels (FDK), and deoxynivalenol (DON) content in an Ottawa FHB nursery in 2004 and 2005, respectively. In the 98B69-L47 population, the 4B QTL from Wuhan 1 had the largest impact and significantly reduced FHB in 2004. However, this QTL was also significantly associated with increased plant height in both years, which may effect its deployment. In the BW301 population, the 2D QTL from Wuhan 1 decreased FHB in both years while the 5AS QTL from Nyubai decreased FHB in 2004. In the Kanata population, the 3BSc QTL from Sumai 3 was more effective than the 5AS QTL from Sumai 3. The Nyubai 3BS QTL did not have a major effect on FHB in these tests. Overall, there was a general reduction in FHB symptoms as FHB resistance QTLs were introduced into an elite genetic background.

SOURCES OF FUSARIUM HEAD BLIGHT RESISTANCE IN WHEAT: DIVERSITY AND UTILIZATION

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ABSTRACT

Fusarium graminearum Schwabe (teleomorph *Gibberella zeae* (Schwein.) which causes Fusarium head blight (FHB), also known as scab, is an increasingly important problem in the north-central region of the United States. Host resistance is considered the most practical and effective means of control but breeding has been hindered by a lack of effective resistance genes. Research funded by the National Wheat and Barley Scab Initiative (USWBSI) has led to the systematic evaluation of spring and winter wheat accessions contained in the National Small Grains Collection at Aberdeen Idaho and to the introduction of resistant germplasm from other programs globally. To date, approximately 15,000 spring and winter wheat accessions have been screened at South Dakota, Minnesota, and Missouri. Additionally, 479 spring and 308 winter wheats carrying putative resistance genes from wheat improvement programs in China, Japan, Argentina, Brazil, Uruguay, Romania, Hungary, and Mexico have been introduced through a collaborative effort between the USWBSI and CIMMYT. Although many good to excellent sources of resistance, particularly type II resistance, have been identified in winter and spring backgrounds no source of complete resistance has been identified and current sources continue to provide only partial resistance. Summarizing information generously provided by US wheat breeders it is clear that most of the FHB resistant germplasm being developed contains some level of type II resistance (reduced spread). Most spring and winter programs use Sumai 3, its derivatives including Ning 7840, or other Asian sources known to carry the 3BS QTL as a major source of type II resistance. The Sumai 3 resistance, particularly when derived from Ning 7840, has good combining ability and can be found in the pedigrees of many promising germplasm lines. More adapted germplasm lines from Virginia, Purdue, and Illinois derived from Ning 7840 are also noted by breeders as good sources of this resistance. Native genotypes that have low incidence, reduced spread, or both, are also widely used. Sources most commonly include: Freedom, Roane, Goldfield, Ernie, and Truman, and, to a lesser extent, McCormick, and Tribute. Promising germplasm combining Asian and native sources of resistance has now been developed in several programs. Some programs are also using sources of resistance identified through germplasm screening programs funded by the USWBSI. Both spring and winter wheat programs are using germplasm from CIMMYT and South America. Winter wheat programs are also using germplasm from Europe (predominantly the Romanian variety F201R). Hindering the incorporation of other sources identified through germplasm screening is the lack of adaptation of these lines. Although many contain very high levels of resistance, landraces in particular, are often late and tall and their direct utilization in main-stream breeding programs will slow the release of highly resistant soft red winter wheat rather than accelerate their release. Significant pre-breeding is needed to exploit these potentially valuable sources of resistance. Also impeding progress is the lack of genetic characterization of these lines. What FHB genes do they carry? How are the genes in these lines related to those in widely used sources from Asia and those characterized as 'native' sources of resistance? Knowledge of these genetic interrelationships is critical to making informed parental choices in programs aimed at developing lines with the multiple sources of resistance. Haplotype and molecular genetic diversity data based on AFLPs for 96 resistant genotypes from Europe, South America, the United States and Asia will be presented in an effort to provide interested breeders with preliminary information on the diversity of FHB alleles in these germplasm resources.

ENHANCING FUSARIUM HEAD BLIGHT RESISTANCE IN SPRING WHEAT: A GLANCE INTO SUCCESS AND CHALLENGES

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OBJECTIVES

To develop new improved hard red spring wheat (HRSW) cultivar and germplasm that combines resistance to Fusarium head blight (FHB), leaf diseases, and superior grain yield and bread-making quality.

INTRODUCTION

Scab disease in cereal crops has become a very important disease in recent years. The favorable environmental conditions including wet weather during flowering and grain filling and major changes in cropping system (introduction of Maize and minimum tillage) has favored disease development. Since 1993, FHB has caused serious loss of yield and quality in HRSW and durum wheat (*T. turgidum* L.) in the Northern Great Plains of the USA. Recent reports (Nganje et al., 2004) estimate combined direct and secondary economic losses caused by FHB for all crops were at $7.7 billion. Two states, ND and MN, account for about 68% ($5.2 billion) of the total dollar losses. Direct economic losses for wheat only were estimated to $2.492 billion from 1993 trough 2001 (Nganje et al., 2004). *Fusarium graminearum* Schwabe (perfect stage: *Gibberella zeae* (Schw.)Petch) has been the principal pathogen (McMullen et al., 1997; Stack, 2003). The FHB is unpredictable, and crop management generally has been ineffective for control. Chemical treatments can help reduce FHB but may not give an economic return. The only long –term and sustainable solution to FHB appears to be breeding for resistance (Meidaner, 1997). Resistance to FHB in wheat is a character of highly complex inheritance (Stack, 2003). Introducing complex resistance into commercial wheat and maintaining it through successive cycles of crossing to adapted but susceptible parents is a difficult task. This usually, requires continued effort using a reliable and repeatable disease testing procedures. Given the time and resources for that effort, however, there is no practical or theoretical reason why such a complex character such as FHB resistance cannot be added. In fact, grain yield and quality are also complex traits and they have been part and the driving force behind every wheat breeding effort. Using classical breeding techniques and various novel technologies, the NDSU HRSW breeding project aims to (1) develop improved HRSW cultivars and germplasm which combine higher levels of resistance to FHB, superior grain yield, and bread-making quality; and (2) identify, introgress, and pyramid novel FHB resistance from diverse germplasm sources into adapted HRSW germplasm base.

MATERIALS AND METHODS

Adapted FHB resistant parents developed by the NDSU and other breeding programs and the recently developed genotypes (from the scab initiative germplasm effort) are selected for planned matings in the greenhouse crossing blocks. The segregating populations generated from crosses are screened in field nursery. Breeding cycles have now progressed such that many of the new adapted parents have FHB resistance (type II mainly) based on previous field and greenhouse results plus other agronomic and quality parameters. To introduce FHB resistance into HRSW wheat cultivars adapted to ND and neighboring regions, we have combined extensive FHB screening done in an inoculated, irrigated field nursery and intensive testing of elite materials in the greenhouse. To maintain high disease pressure, a field nursery to screen wheat lines for resistance to FHB was established. Plots are elongated hill plots, each a single genotype, randomized within replicates. Throughout the nursery are multiple repeated check lines of known FHB reaction. Beginning at jointing stage *G. zeae*-colonized corn ("grain spawn") was distributed on the ground

throughout the nursery. By the time the earliest genotypes reached anthesis, blue perithecia of *G. zeae* stage were present on the grain spawn. Light mist irrigation was applied on an intermittent cycle for a period of 3 days per week. By 3 to 3.5 weeks postanthesis FHB had developed and was scored visually on 20 individual heads per hill plot using a 0-100% scale (Stack and McMullen, 1996). Grain was harvested from plots and proportion of visually scabby kernels (VSK) was determined. Deoxynivalenol (DON) levels in grain were determined by the NDSU Veterinary Sciences Laboratory using Gas Chromatographic analysis. The intensive greenhouse testing was done using the single spikelet method of FHB inoculation (Stack et al., 1997).

The advanced and elite HRSW lines generated by the breeding program which have FHB resistance are tested in preliminary, advanced, and elite yield trials at 2, 4, and 6 ND locations, respectively. The agronomic data; including grain yield and quality data, pests reactions including FHB due to natural infection, and shattering are generated from these trials. This data is also crucial to decision making on seed increase and eventual release of the new elite lines. Selected spikes/plants from the populations are sent to the winter nurseries in New Zealand to be advanced and selected for some agronomic traits such as height, maturity, shattering, and other plant type.

RESULTS AND DISCUSSIONS

The use of the FHB evaluation methods described above has enabled us to produce consistently very high FHB disease pressure (Table 1). This has facilitated the identification of improved lines. We have tested the FHB response in many lines representing progeny from first, second, third, and fourth breeding cycles. Some first and second cycle progeny showed good FHB resistance but none combined good FHB resistance with the agronomic traits and quality requirements that meet the commercial release. Several advanced cycles derived lines combined those traits however, and were released as a germplasm (Frhoberg et al., 2004; Mergoum et al., 2005) or commercial cultivars (Mergoum et al., 2005; 2006).

"Alsen"- was the first NDSU spring wheat cultivar released with good FHB resistance. It was derived from the three way cross "ND674//ND2710 /ND688". ND 674 and ND688 are two HRSW experimental lines developed by NDSU breeding program with good adaptation to ND wheat growing conditions and good end-use quality. Both lines are derived from 'Glupro' (PI 592759), a HRSW cultivar released in 1995 by NDSU for its very high grain protein content. ND2710 (Frohberg et al., 2004) is a HRSW experimental line developed by NDSU breeding program from a cross involving Sumai 3. Alsen agronomic performance and disease reactions are reported in Table 2. It has a good yield potential in general, especially in eastern ND where scab disease is prevalent. Test weight and lodging resistance are excellent for Alsen and shattering resistance appears satisfactory. Alsen is moderately resistant to predominant Upper Midwest races of leaf rust (caused by *Puccinia recondita* Rob. Ex Desm. f. sp. *Tritici*), resistant to such races of stem rust (caused by *Puccinia graminis* Per.:Pers. f. sp. *tritici* Eriks. & E. Henn), susceptible to tan spot [caused by *Pyrenophora tritici-repentis* (Died.) Drechs], moderately susceptible to the Septoria leaf disease complex [caused mainly by *Stagonospora nodorum* (Berk.) Castellani & E.G. Germano] and to common root rot (Tables 2, 4, and 5). Alsen has Fusarium head blight resistance type II expressed as reduced spreading of the disease in the spike (Table 5). Alsen has been planted on about 1 million hectares from 2002 to 2005; representing more than 30% of ND wheat acreages (N.D. Agricultural Statistics Service, USDA. 2002; 2003; 2004; 2005).

"Steele-ND"- is another HRSW wheat cultivar that was released by NDSU in 2004 with moderate FHB resistance (Mergoum et al., 2005). Steele-ND was selected from the cross 'Parshall' (PI 613587)/5/ 'Grandin' (PI 531005)/3/IAS20*4/H567.71// 'Amidon' (PI 527682)/4/ Grandin*2/'Glupro' (PI 592759). Steele-ND average FHB severity was 31.5% comparable to Alsen (28.7%) but significantly ($p<0.01$) lower than the susceptible check 'Reeder' (58.9%) (Table 5). Visual scabby kernels of Steele -ND (26.5 %) was also very low ($p<0.01$) compared to the susceptible check Reeder (37.2%), but slightly higher than Alsen (20.9%). Steele-ND does not include Sumai 3

in its pedigree and the source of resistance is believed to originate from the wheat relative *T. Dicoccoides*. A recombinant inbred lines (RIL) population derived from the cross of ND 735 with Steele-ND was developed for the purpose of mapping the FHB genes involved in Steele-ND. Grain yield of Steele-ND is similar to Reeder and, Parshall, and Alsen (Tables 3 and 4). Steele-ND is resistant to pathotype THBL, the predominant race of leaf rust in the region, and resistant to stem rust (Tables 3 and 5). Steele-ND is moderate resistant to Septoria nodorum and moderately susceptible to tan spot (Table 5).

"Glenn"- is the most recent NDSU HRSW release (2005) with improved FHB resistance compared to Alsen and Steele-ND. Glenn was selected from the progeny of the ND2831//Steele-ND cross. ND2831 is a Sumai 3 derivative line that has scab resistance similar to Alsen. This cross aimed to combine sources of FHB resistance from Alsen and Steele-ND, high yield, excellent quality, and standability into one package. Data collected during the testing period indicate that Glenn provides scab resistance (Table 4) and yield potential superior to Alsen; along with improved lodging, leaf diseases resistance, and equal or slightly better milling and baking quality (Table 4). Glenn has exceptional high grain volume (Table 4), as well as excellent end-use quality for the domestic and export wheat markets. Glenn grain yield is similar to Alsen, Parshall and Reeder, but lower than Steele-ND. Grain volume of Glenn is 811 kg m^{-3}, significantly higher than Alsen, Parshall, and Dapps. Protein content of Glenn (15.8%) is lower than Dapps (16.4%) but similar to Alsen, Parshall, and higher than Reeder (15.4%). Glenn exhibited a high level of resistance to the predominant races in the region of leaf rust and stem rust. It is medium resistant to tan spot and medium susceptible to septoria nodorum (Tables 4 and 5).

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Table 1. FHB index (Incidence x severity) and Tombstone (scabby) kernels in the North Dakota, hard red spring wheat during the 1995-2004 period.

Year	FHB Index[1]	Tombstone Kernels (VSK)[2]	DON[3]
	(Incidence X severity)	%	ppm
1995	3.0 - 56.6	3.3 - 19.6	0.6 - 7.7
1996	3.7 - 80.8	2.3 - 70.6	2.0 - 116.8
1997	36.8 - 100.	15.2 - 60.5	7.8 - 104.4
1998	19.9 - 80.7	1.8 - 95.9	0.6 - 59.7
1999	23.7 - 89.6	11.6 - 92.4	22.4 - 89.8
2000	22.3 - 92.0	13.9 - 82.8	3.2 - 62.7
2001	25.8 - 75.9	5.0 - 47.5	0.7 - 17.1
2002	32.0 - 97.4	30.0 - 92.5	18.8 - 114.
2003	3.4 - 60.6	8.6 - 65.8	-
2004	2.9 - 87.5	14.1 - 68.4	3.2 - 18.7

[1] FHB Iindex: FHB % by visual scoring at 3.5 wk after flowering; [2] VSK: Visually Scabby Kernels - proportion in harvested grain; [3] DON: Vomitoxin determined by GC analysis of harvested grain.

Table 2. Agronomic traits and reaction to FHB and leaf diseases of Alsen and five other hard red spring wheat cultivars in North Dakota , USA, during the 1998-2000 period.

				Reaction[1]					
Variety	Days to heading	Height	Lodging	Leaf rust	Septoria	FHB	Test weight	Protein	Grain yield
	Days	cm	1-9				Kgm^{-3}	%	$Kgha^{-1}$
Butte 86	59	89	1.5	MS	MS	S	757	15.5	3507
Russ	60	89	1.8	R	R	S	743	15.1	3521
Gunner	63	89	1.3	MS	MR	MS	770	14.4	3306
2375	60	84	3.8	S	S	MS	768	14.7	3467
Grandin	62	79	1.9	MS	S	S	759	15.7	3003
Alsen	61	84	0.9	MR	MR	MR	770	15.3	3279

[1]R=resistant, MR=moderately resistant, MS=moderately susceptible, S=susceptible

Table 3. Agronomic traits and reaction to FHB and leaf diseases of Steele-ND and the most grown three major hard red spring wheat cultivars in North Dakota , USA, during the 2000-2003 period.

				Reaction[1]					
Variety	Days to heading	Height	Lodging	Leaf rust	Tan Spot	FHB	Test weight	Protein	Grain yield
	Days	cm	1-9				Kgm^{-3}	%	$Kgha^{-1}$
Steele-ND	61	84	2.7	R	MS/MR	MR/MS	762	15.7	3837
Alsen	59	79	1.9	MR	S	MR	762	16	3689
Parshall	60	89	1.8	MS/S	MS	MS	764	15.8	3716
Reeder	59	79	1.5	S	MR	S	770	15.5	3910

[1]R=resistant, MR=moderately resistant, MS=moderately susceptible, S=susceptible

Table 4. Agronomic traits and reaction to FHB and leaf diseases of Glenn and five most grown hard red spring wheat cultivars in North Dakota, USA, during the 2002-2004 period.

				Reaction[1]					
Variety	Days to heading	Height	Lodging	Leaf rust	Tan Spot	FHB	Test weight	Protein	Grain yield
	Days	cm	1-9				Kgm^{-3}	%	$Kgha^{-1}$
Glenn	65	87	0.7	R	MS/MR	MR/R	806	15.8	4421
Alsen	65	84	1.1	MR/MS	S	MR	770	15.6	4317
Dapps	65	91	1.2	R/MR	MR	MS	772	16.4	4209
Parshall	65	94	1.3	MS/S	MS	MS	768	15.6	4347
Reeder	66	83	0.5	S	MR	S	755	15.4	4519
Steele-ND	66	87	1.9	R/tMR	MS/MR	MR/MS	772	15.6	4552

[1]R=resistant, MR=moderately resistant, MS=moderately susceptible, S=susceptible

Table 5. Fusarium head blight (FHB) severity, Tombstone and Deoxynivalenol toxin levels; leaf rust , stem rust , tan spot, and Septoria nodorum reactions of HRSW cultivars under natural (3 locations) and artificial (7 locations) inoculation in the filed and greenhouse conditions (4 tests) in Fargo, ND from 2001 to 2004.

Genotype	FHB Severity (Field test under natural infection) [1]			FHB Severity (Field test under artificial inoculation)			FHB GH	SR[2]	TS	SN
	SEV (%)	TMB (%)	DON (ppm)	SEV (%)	TMB (%)	DON (ppm)	SEV (%)		(1-5)	(1-5)
Glenn	7.6	0.7	0.4	18.9	16.0	4.0	16.3	R	3	3
Steele-ND	19.3	1.3	0.9	31.5	26.5	5.3	24.6	R	4	3
Alsen	7.0	0.9	0.8	28.7	20.9	4.8	10.8	R	5	5
Reeder	26.2	5.7	1.5	58.9	37.2	10.3	42.0	R	4	4
2398	41.8	5.4	2.0	75.2	51.7	9.9	55.5	R	-	-

[1]SEV=Severity; TMB= Tombstone; and DON= Deoxynivalenol toxin;
[2] SR= Stem rust; TS= Tan spot; SN= Spetoria nodorum; R=Resistant and MS=Moderate susceptible.

"GLENN" HARD RED SPRING WHEAT CULTIVAR: A NEW STEP IN COMBATING FUSARIUM HEAD BLIGHT DISEASE

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OBJECTIVES

To develop new improved hard red spring wheat (HRSW) cultivar that combines novel source of resistance to Fusarium head blight (FHB) disease and superior grain yield and bread-making quality.

INTRODUCTION

It well documented that today, Fusarium head blight (FHB), commonly known as scab, is one of the serious threat to wheat production throughout the world (Schroeder and Christenson, 1963; Bai and Shaner, 1994; McMullen et al., 1997; Stack, 2003). In North America, FHB is caused mainly by *Fusarium graminearum* Schwabe [telomorph *Gibberella zeae* (Schwein.)] (Bai and Shaner, 1994; McMullen et al., 1997). Wheat FHB has been a major disease for hard red spring wheat (HRSW) produced in North Dakota and neighboring states since 1993. Recent estimates (Nganje et al., 2004) showed that the combined direct and secondary economic losses caused by FHB for all crops were at $7.7 billion. North Dakota and MN, account for about 68% ($5.2 billion) of the total dollar losses. Direct economic losses for wheat only were estimated to $2.492 billion from 1993 trough 2001 (Nganje et al., 2004). The use of genetically resistant cultivars is believed to the most efficient and economical method of controlling this disease in HRSW. In 2002, 2003, 2004, and 2005 crop seasons, "Alsen", a moderate FHB resistance cultivar derived from the Chinese source "Sumai 3", released in 2000 by NDSU (with the support of the scab initiative funds) was planted on more than 2.1, 2.4, and 1.9 million acres representing 30.8, 37.4, 28.9, and 23.1% of the 6.8 million acres of ND wheat, respectively (N.D. Agricultural Statistics Service, USDA. 2002; 2003; 2004; 2005). Similarly, "Steele-ND", a new NDSU HRSW cultivar released in 2004 covered as much as 1.4% of ND wheat acreages. The rapid increase in acreage planted to 'Alsen' and Steele-ND indicates the desire of ND wheat growers to produce such HRSW cultivars. However, the level of resistance of both Alsen and Steele-ND needs to be improved in order to better control FHB. Therefore, any new HRSW cultivar with better FHB resistance than the actual grown HRSW cultivars is warranted.

MATERIALS AND METHODS

To introduce FHB resistance into adapted quality wheats in North Dakota, we have combined extensive FHB screening done in an inoculated, irrigated field nursery and intensive testing of elite materials in the greenhouse. To maintain high disease pressure, a field nursery to screen wheat lines for resistance to FHB was established. Plots were elongated hill plots, each a single genotype, randomized within replicates. Throughout the nursery were multiple repeated check lines of known FHB reaction. Beginning at jointing stage *G. zeae*-colonized corn ("grain spawn") was distributed on the ground throughout the nursery. By the time the earliest genotypes reached anthesis, blue perithecia of *G. zeae* stage were present on the grain spawn. Light mist irrigation was applied on an intermittent cycle for a period of 3 days per week. By 3 - 3.5 weeks postanthesis FHB had developed and was scored visually on 20 individual heads per hill plot using a 0-100% scale (Stack and McMullen, 1996). Grain was harvested from plots and proportion of visually scabby kernels (VSK) was determined. Deoxynivalenol (DON) levels in grain were determined by the NDSU Veterinary Sciences Laboratory using Gas Chromatographic analysis. The intensive greenhouse testing was done using the single spikelet method of FHB inoculation (Stack et al., 1997). As wheat

heads reached anthesis, ten heads in each replicate of each line were individually inoculated by placing a 10 ml droplet of *F. graminearum* spore suspension inside a floret near the midpoint of the head. A gentle overhead mist was applied for three nights following inoculation. FHB was scored at 3.5 wk as described above. Levels of tombstone kernels and DON were also determined in harvested grain.

RESULTS AND DISCUSSIONS

"Glenn" is the most recent NDSU HRSW release (2005) with improved FHB resistance compared to Alsen. Glenn was selected from the progeny of the cross: ND2831//Steele-ND. ND2831 is a Sumai3 derivative line that has scab resistance similar to Alsen. This cross aimed to combine sources of FHB resistance from Alsen and Steele-ND, high yield, excellent quality, and standability into one package. Data collected during the testing period indicate that Glenn provides scab resistance and yield potential superior to Alsen; along with improved standability, an improved leaf disease package, and equal or slightly better milling and baking quality. The improved scab resistance and yield advantage of Glenn is especially evident in areas where disease pressure is high (i.e. Eastern North Dakota). Glenn also exhibits exceptionally high test weight. Glenn appears to have the potential to provide producers with an alternative for 'Alsen'.

Reaction to FHB - Glenn was tested for FHB in seven location-years in the FHB nursery grown at Prosper, ND under artificial inoculation using overhead irrigation techniques. It was also evaluated in three environments under natural FHB infection and in four experiments under greenhouse conditions using the spray inoculation. On the basis of seven location-years of testing in the FHB nursery conducted under field conditions, the FHB incidence (Stack et al., 1997) recorded for Glenn (19%) was significantly higher than the most resistant line '2710' (9%) developed by NDSU (Frohberg et al., 2004); but significantly lower than the incidence for the moderately resistant checks Alsen (29%) and Steele-ND (31%); and susceptible checks Reeder (59%) and '2398' (42%). Similarly, on the basis of the three location-years of testing for FHB under natural infection conducted under field conditions, the FHB incidence recorded for Glenn was 8% compared to 2, 7, 19, 26, and 42% scored for 2710, Alsen, Steele-ND, Reeder, and Pioneer 2375, respectively. Under greenhouse conditions, the FHB incidence of Glenn based on four tests was 16% compared to 9, 11, 25, 42, and 56% for 2710, Alsen, Steele-ND, Reeder, and Pioneer 2375, respectively. Glenn was also evaluated for the levels of the trichothecene mycotoxin deoxynivalenol (DON) produced by FHB in three naturally and three artificially infected field tests. Under naturally infected conditions, the DON level of Glenn was 0.4 $\mu g\ g^{-1}$ compared to 0.5, 0.8, 0.9, 1.5, and 2 $\mu g\ g^{-1}$ for 2710, Alsen, Steele-ND, Reeder, and Pioneer 2375, respectively. Under artificial inoculation, the DON level of Glenn (4 $\mu g\ g^{-1}$) was similar to ND 2710 (2.9 $\mu g\ g^{-1}$), Alsen (4.8 $\mu g\ g^{-1}$), and Steele-ND (5.3 $\mu g\ g^{-1}$); but significantly lower that than the DON levels of Pioneer 2375 (7.4 $\mu g\ g^{-1}$), Reeder (10.3 $\mu g\ g^{-1}$), and 2398 (9.9 $\mu g\ g^{-1}$). Alsen was released in 2000 as the first NDSU HRSW cultivar with resistance to FHB from the Chinese 'Sumai 3' (PI 481542) and has been widely grown in the northern plains since 2001. Steele-ND, a NDSU HRSW cultivar released in 2004 (Mergoum et al., 2005b), has resistance to FHB comparable to Alsen but has parentage different from the Chinese Sumai 3. Compared to ND 744, Glenn has similar FHB resistance and agronomic performance. However, ND 744 has harder kernels, lower (10 g kg^{-1} in average) protein content, and grain volume than Glenn. Based on seedling and adult plant screening tests conducted under greenhouse conditions from 2000-2004, Glenn exhibited a high level of resistance to pathotype THBL, the predominant race of leaf rust in the region. Glenn was evaluated from 2000 to 2004 at the USDA-ARS, Cereal Crop Research Unit, Fargo, ND for resistance to stem rust (caused by *Puccinia graminis* Per.:Pers. f. sp. *tritici* Eriks. & E. Henn) and was found to be resistant to pathotypes Pgt-QCCJ, -QTHJ, -RTQQ, -TMLK, -TPMK, and -HPHJ. On a scale of 1 to 5 where 1 is resistant and 5 susceptible, Glenn had average scores of 4 and 3 in reaction to Septoria nodorum [caused by *Stagonospora nodorum* (Berk.) Castellani & E.G. Germano] and tan spot [caused by *Pyrenophora tritici-repentis* (Died.) Drechs] compared to 5 and 5 for the susceptible cultivar Alsen and

1 and 1 for the resistant check 'Erik' (PI 476849), respectively.

Agronomic performance and quality parameters - Data from 31 site-years in the ND Variety Trials, grain yield of Glenn (4381 kg ha^{-1}) was similar to 'Alsen' (PI 615543) (4300 kg ha^{-1}), 'Parshall' (PI 613587) (4347 kg ha^{-1}) and 'Reeder' (PI 613586) (4448 kg ha^{-1}), but lower ($P<0.05$) than Steele-ND (4885 kg ha^{-1}). In the same trials grain volume of Glenn was 811 kg m^{-3}, significantly higher ($P<0.05$) than 767, 768, and 776 kg m^{-3} of Alsen, Parshall, and 'Dapps' (PI 633862), respectively. Protein content of Glenn (166 g kg^{-1}) was lower than Dapps (171 g kg^{-1}) but similar to Alsen (163 g kg^{-1}), Parshall (164 g kg^{-1}), and higher than Reeder (161 g kg^{-1}). On the basis of 36 locations of the URN conducted in 2003 and 2004, mean grain yield, grain volume weight, and protein content of Glenn were 4340 kg ha^{-1}, 794 kg m^{-3}, and 154 g kg^{-1}, respectively, compared to Steele-ND (4515 kg ha^{-1}, 775 kg m^{-3}, and 152 g kg^{-1}), 'Pioneer 2375' (4475 kg ha^{-1}, 777 kg m^{-3}, and 143 g kg^{-1}), and 'Verde' (PI 592561) (4461 kg ha^{-1}, 762 kg m^{-3}, and 142 g kg^{-1}). Flour yield for Glenn from 13 trials grown in ND averaged 684 g kg^{-1} compared to 692, 693, and 678 g kg^{-1} for Alsen, Parshall, and Reeder, respectively. Water absorption was 65.9%, significantly higher than Reeder (64.8%), but not different from Alsen (65.1%), and Parshall (65.2%). The mixing tolerance of Glenn (20.9 min) was longer than all of the checks including Reeder (13.9 min), Alsen (16.4 min), and Parshall (17.0 min). Loaf volume was 1103 mL, comparable to Parshall (1090 mL) and Alsen (1076 mL), but superior to Reeder (1015 mL).

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FHB RESISTANCE OF THE USSRW SCREENING NURSERY WITH THE MICRO PLOT METHOD

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ABSTRACT

Forty-eight lines were tested in 2005 for Fusarium head blight (FHB) in wheat. All genotypes were sown in a 5 m^2 plot and inoculated at the same time, 31 May. In heading and flowering the genotypes differed 6 days. We thought, even the earlier genotypes were inoculated several days later than optimal, but the problems arising from the different meteorological conditions cause more problems than the several days delay in inoculation. From earlier experience we know that the window for a high infection is about 7-10 days which is significantly longer than generally accepted. For this reason we think that the comparability of the data is much better than before. This view is supported by the fact that the resistant control Ernie is among the best genotypes and the susceptible Cooker takes on of the last positions.

Four isolates were used in each plot, inoculating three groups of heads consisting of 15-20 heads for each isolate with about 15 ml inoculum. After inoculation the groups of heads were covered by plastic bags to secure the high humidity for the infection. No additional mist irrigation was used as they interfere severely with the symptom development of the genotypes. Also three check bunches were added to enable yield response analysis. FHB severity (% of infected spikelets) were rated 10, 14, 18, 22, and 26 days after inoculation. At full ripening the bunches were harvested, from each group ten average ears were separated for threshing with no wind, thereafter a cleaning were made with no loss of infected grains. Fusarium damaged kernels (FDK) was visually evaluated. In several cases also toxin analysis for DON has been made.

The most resistant genotype had 0.46 % FHB as a mean of five readings. The maximum is 30.69 %. The ratio of Fusarium damaged kernels was between 1 and 68 %, the yield loss spreads between 5.6 and 56 %. The correlation between traits is generally $r = 0.80$, significant at $P = 0.001$. This means that lower FHB data normally mean lower FDK and yield loss. Even so, at 12 % FHB we find FDK data between 3 and 38 %. For this reason an absolute forecast of the FDK values or yield loss for a given genotype is not possible as also resistance types influence the infection process. Therefore all parameters should be evaluated.

We represent the view that the American breeding made significant steps in breeding lines with good or excellent FHB resistance. In several lines, also very high resistance to Septoria tritici leaf spot and other leaf spots has been identified combined in some cases with good quality and yield potential.

Additional data for leaf diseases, quality data, plant height, yielding ability and other data help breeders to evaluate the materials properly.

THE 2004-05 UNIFORM SOUTHERN FUSARIUM HEAD BLIGHT SCREENING NURSERY

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ABSTRACT

Phenotypic estimates of host resistance to Fusarium head blight (FHB) are greatly confounded by genotype x environment interaction effects. Thus, multiple evaluations of genotypes are necessary in order to determine true genetic worth. The objectives of the Uniform Southern FHB Nursery are to provide breeders with a comprehensive set of resistance estimates on advanced generation lines in a timely fashion, and to facilitate the sharing of the best resistant materials throughout the breeding community. The 2004-05 nursery comprised 46 advanced generation breeding lies and two check cultivars, 'Ernie' (partially resistant) and 'Coker 9835' (susceptible). Seven U.S. public programs (Univ. of Arkansas, Univ. of Georgia, Louisiana State Univ., Univ. of Maryland, N.C. State Univ, VA-Tech, and USDA-ARS), two private companies (Agripro and Syngenta) and one Romanian (Fundulea) program submitted entries for evaluation. A comprehensive set of field, greenhouse and laboratory results was submitted by eight U.S., one Romanian and one Hungarian cooperator. A preliminary summary table of means for several important host resistance traits is presented below. Copies of the full report will be available at the 2005 National Fusarium Head Blight Forum and subsequently on line at the USWBSI web site: http://www.scabusa.org/.

UNIFORM SOUTHERN FHB NURSERY MEANS ACROSS LOCATIONS 2004-05

	Cultivar/ Designation	FHB Incidence	RANK	FHB Severity	RANK	FHB Index	RANK	FDK	RANK	ISK	RANK	DON	RANK	G'hse Type II	RANK	Heading Date	RANK	Plant Height	RANK	Qfhs.ndsu-3BS Xgwm533.1 161	Qfhs.ndsu-3BS XBARC133 142	Qfhs.ifa-5A Xgwm293 218	Qfhs.ifa-5A Xgwm156 342
1	ERNIE	42	3	16	5	5.7	3	16	3	24	1	1.7	12	9	3	130	5	32	6	.	X	.	.
2	COKER 9835	80	47	46	47	37.3	46	35	42	56	46	12.1	47	46	43	134	41	62	1	.	.	.	.
3	B006624	60	23	19	9	14.3	18	14	1	31	6	3.9	29	38	38	132	20	37	27	X	.	.	.
4	B990081	51	10	21	12	9.2	8	23	16	31	6	0.8	2	26	25	132	20	41	36	X	.	.	.
5	02JH000014	55	14	16	5	10.1	10	27	24	33	10	2.4	20	9	3	132	20	37	41	X	.	.	.
6	AR97002-2-1	52	11	17	8	7.9	5	24	17	33	10	1.2	6	13	9	130	5	33	21	.	.	X	.
7	AR97002-2-2	45	4	14	2	4.5	2	28	29	29	4	1.2	6	8	2	130	5	30	11	.	.	X	.
8	AR97048-1-1	59	21	26	24	14.5	19	22	14	36	18	4.5	31	18	13	133	33	46	47	X	.	.	.
9	AR97048-4-1	72	42	36	42	27.1	43	31	38	48	41	11.8	46	37	37	134	41	42	47	X	.	.	.
10	AR97048-7-1	70	38	27	27	18.8	31	30	35	44	33	5.9	38	15	11	135	46	49	46	X	.	.	.
11	AR97124-4-1	47	5	19	9	9.5	9	20	8	29	4	2.1	18	24	21	132	20	40	44	.	.	.	.
12	D00*6847-1	58	19	25	23	16.0	25	31	38	42	26	4.3	30	42	41	131	12	37	27	.	.	.	.
13	D00*6874-1	49	9	32	37	15.1	21	30	35	42	26	5.7	36	39	39	130	5	38	36	.	.	.	.
14	D00*6874-9	53	12	30	31	13.9	15	24	17	37	21	3.4	27	41	40	130	5	49	27	.	.	.	.
15	D01*7759	73	44	32	37	21.1	33	28	29	41	23	3.1	25	56	46	132	20	39	41	.	.	.	.
16	D01-7017	64	28	32	37	20.6	32	28	29	44	33	4.7	33	21	16	132	20	45	27	.	.	.	.
17	F92080G-01102	56	16	16	5	8.9	7	24	17	33	10	2.3	19	11	7	134	41	42	21	.	.	.	.
18	F95812G1-1 Fz1	70	38	30	31	24.7	40	26	21	47	39	2.9	23	18	13	133	33	35	11	.	.	.	.
19	F96035G11-2	59	21	15	3	8.2	6	20	8	33	10	2.0	17	14	10	131	12	37	11	.	.	.	.
20	GA951395-3E25	70	38	35	40	21.2	36	27	24	43	31	6.0	39	30	28	133	33	35	11	.	.	.	.
21	GA961171-3E38	64	28	35	40	25.1	41	29	33	47	39	8.0	43	25	23	129	2	44	1	.	.	.	.
22	GA961176-3A48	67	34	30	31	21.1	33	27	24	44	33	6.1	40	23	19	131	12	45	21	.	.	.	.
23	GA961526-3E15	67	34	38	44	25.6	42	38	45	50	42	9.0	45	53	45	132	20	59	21	.	.	.	.
24	GA96229-3A41	77	45	50	48	39.2	48	45	48	60	48	17.6	48	64	47	133	33	65	27	.	.	.	.
25	GA96229-3E39	69	37	42	46	29.8	45	39	46	53	45	7.5	42	69	48	132	20	62	27	.	.	.	.
26	LA9560CA22-1	66	32	30	31	21.6	37	37	44	50	42	4.6	32	36	36	133	33	40	44	.	.	.	.
27	LA97407D-17-4	48	6	24	21	13.2	13	21	13	35	16	1.1	5	32	32	128	1	40	11	.	.	.	.
28	LA97448D-27-4	48	6	26	24	13.6	14	17	4	31	6	2.5	21	50	44	129	2	49	36	.	.	.	.
29	LSU04FHB02	60	23	29	28	17.1	26	20	8	39	22	3.3	26	35	35	130	5	39	41	.	.	.	.
30	M01*1019	72	42	23	16	17.2	27	27	24	42	26	1.8	13	21	16	133	33	43	6	.	.	.	.
31	MV-5-46	71	41	31	36	22.2	39	30	35	45	37	5.5	35	29	26	132	20	48	1	.	.	.	.
32	NC03-11457	67	34	29	28	21.7	38	19	7	42	26	1.4	8	15	11	132	20	37	6	X	X	X	X
33	NC03-11458	54	13	22	14	11.6	11	15	2	31	6	1.5	9	21	16	132	20	39	6	X	X	X	X
34	NC03-11465	37	1	15	3	7.3	4	17	4	25	2	0.9	3	25	23	136	47	47	21	X	X	.	.
35	NC03-11561	55	14	23	16	15.0	20	24	17	34	14	1.5	9	31	30	137	48	34	6	X	X	.	.
36	NC03-11588	77	45	36	42	29.6	44	29	33	51	44	5.8	37	29	26	134	41	53	11	.	.	.	.
37	PI564341	86	48	40	45	37.7	47	40	47	58	47	8.1	44	34	34	132	20	53	1	.	.	.	.
38	PI564385	64	28	26	24	18.7	30	26	21	42	26	6.3	41	24	21	133	33	50	36	.	.	.	.
39	TX96D1073	57	18	23	16	13.9	15	26	21	36	18	1.8	13	31	30	131	12	43	21	.	.	.	.
40	TX98D1170	60	23	22	14	21.1	33	36	43	46	38	4.9	34	10	5	132	20	40	27	.	.	.	.
41	TX98D2423	60	23	20	11	12.9	12	20	8	34	14	1.5	9	30	28	134	41	48	27	.	.	.	.
42	TX99D4478	58	19	24	21	15.1	21	32	40	43	31	3.0	24	20	15	131	12	40	27	.	.	.	.
43	VA01W-310	66	32	29	28	18.4	29	28	29	41	23	1.8	13	44	42	133	33	53	11	.	.	.	.
44	VA04W-433	41	2	10	1	4.1	1	18	6	25	2	0.5	1	10	5	129	2	35	11	X	X	.	.
45	VA04W-503	60	23	30	31	17.8	28	27	24	41	23	3.8	28	32	32	131	12	47	36	.	.	.	.
46	VA04W-547	64	28	23	16	15.3	24	34	41	44	33	2.7	22	23	19	131	12	41	11	.	.	.	.
47	VA04W-608	56	16	23	16	15.1	21	20	8	36	18	1.9	16	11	7	130	5	34	11	.	.	.	.
48	VA04W-628	48	6	21	12	13.9	15	22	14	35	16	0.9	3	6	1	131	12	31	1	.	X	.	.
	Sumai 3																			X	X	X	X
		61		27		17.6		26.4		40		4.1		28		132		36					
		17		10		12.4		15.6		14		.		18		2		2					
		27		42		67.0		52.4		29		.		47.2		1.5		5.4					

FINE MAPPING OF A QTL REGION ASSOCIATED WITH FUSARIUM HEAD BLIGHT, KERNEL DISCOLORATION, GRAIN PROTEIN CONCENTRATION, AND HEADING DATE ON BARLEY CHROMOSOME 6H

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OBJECTIVES

To fine map a quantitative trait loci (QTL) region on chromosome 6H in barley (*Hordeum vulgare* L.) previously found to be associated Fusarium head blight (FHB) resistance, kernel discoloration (KD), grain protein concentration (GPC) and heading date (HD); and to determine if the associations can be attributed to tight linkage or pleiotropic effects of a single locus.

INTRODUCTION

In barley, numerous QTLs have been identified for FHB, KD, GPC, and HD (de la Pena et al., 1999; See et al., 2002; Canci et al., 2003; Canci et al., 2004) on all seven chromosomes. However, most of these QTLs are assigned to regions spanning 10-40 cM and therefore cannot be efficiently used for marker-assisted-selection (MAS). To be useful for MAS, the QTL must be validated and localized to a small region flanked by "breeder friendly" markers. We validated QTL for GPC and KD using two mapping populations derived from the cultivar MNBrite (Canci et al., 2003). MNBrite was developed by the University of Minnesota and carries Chevron alleles that condition KD resistance and high GPC, and is moderately resistant to FHB. Results of this study confirmed that the QTL for KD and GPC are coincident and map between markers *Bmag0807* and *Bmac0218* (26 cM) on chromosome 6H. Other studies have detected FHB and HD QTL in the same marker interval (Canci et al., 2004); suggesting that selection for KD resistance in the development of MNBrite may have resulted in increased FHB resistance. MNBrite is also unacceptably high in GPC due in part to the effects of this region on chromosome 6H. We initiated a fine-mapping study to determine the genetic relationships among FHB, KD, and GPC in this region of chromosome 6H.

In this study, we used substitution mapping approach as described by Peterson et al. (1990) to fine map the coincident QTL region. This approach uses recombinant near isogenic lines (rNILs) for mapping and is a powerful method for distinguishing linkage versus pleiotropy.

MATERIALS AND METHODS

Development of the rNILs - A donor parent from the mapping population described by de la Pena et al. (1999) and carrying the Chevron allele at the target QTL region was crossed with the recurrent elite parent M69. A marker-assisted backcrossing scheme using markers *GBM1021* and *EBmac0602* flanking the target QTL region was used to advance lines to the BC3S4. One of the BC3S4 lines (designated as C113) was backcrossed 3 more times to the recurrent parent Lacey. A population of 1200 F3 plants was then developed and screened with the two flanking markers to identify rNILs. One hundred and twenty-nine rNILs were identified. These were then genotyped using five additional SSR markers previously mapped at the *GBM1021-EBmac0602* interval. A linkage map using genotypic data from the entire population of F3s was constructed using the JoinMap version 2.0 (Stam, 1993). The 129 rNILs were advanced to the F3:5 generation for use in field evaluations.

Field Evaluations of rNILs - The 129 rNILs and parental lines Chevron and Lacey were evaluated in

the summer of 2005 at the University of Minnesota Agricultural Experiment Stations at St. Paul, Morris, and Crookston. Entries at each environment were arranged in a randomized complete block design with 2 replications. They were planted in 2.4 m long single-row plots and spaced at 30 cm apart. To inoculate plots, the macroconidia technique was used at St. Paul while the grain-spawn inoculation technique was used at Morris and Crookston as described by Mesfin et al. (2003). To enhance disease development, nurseries were mist-irrigated until the soft dough stage. Four traits, FHB, KD, GPC, and HD were measured for each plot. Data for HD was not collected at Crookston. The percent FHB severity was measured by counting the number of infected spikelets from each spike using10 randomly selected plants from each plot and the count was expressed as a percent of the total spikelets present. Kernel discoloration was measured using a scale of 1-5 (1 = no discoloration and 5 = most discolored) as described by Miles et al. (1987). Grain protein concentration was measured using the Model 6500 NIR Spectrometer (Foss North America). Heading date was recorded as the number of days from planting to 50% emergence from the boot.

Data Analysis - The Proc GLM (SAS Institute, 2000) procedure was used to conduct analysis of variance. Significant G x E effects were observed for all traits except KD and therefore further analyses were performed on individual environments. Means for rNILs and their parental lines were separated using LSD. The means of rNILs carrying similar marker profiles were averaged and compared to the mean of the rNILs carrying Lacey allele for the entire QTL region (control). A genomic region was then declared to be associated with a trait QTL when the trait mean of the set of rNILs carrying the Chevron allele at that region was significantly different from the susceptible control.

RESULTS AND DISCUSSION

The original chromosome 6H region previously found to contain the coincident QTL of interest mapped within a 34 cM segment. The construction of a fine map using 1200 F3s estimated the size of this region as 11 cM. Marker order in the fine map is consistent with the order in the original map.

There was a significant difference among rNILs for all traits measured except for FHB severity at St. Paul; suggesting that each trait QTL was segregating amongst the rNILs evaluated (Table 1). In Crookston, the mean FHB severity of subNILs carrying the Chevron allele at the entire target QTL region was significantly lower than the mean of subNILs carrying the Lacey allele across the QTL region (Fig. 1). The Chevron allele at this environment reduced FHB severity by over 50%. These results suggest that the QTL for FHB maps within the region. However, we could not precisely map the position of the QTL In Morris, we did not detect significant FHB QTL and we believe this was because we assessed FHB severity at this environment when plants were starting to show signs of senescence. The mean percent GPC for subNILs carrying the Lacey allele at the *Bmag0807-GBM1063* interval (2 cM) was 0.5-0.7% lower compared to the mean percent GPC for subNILs carrying the Chevron allele at the same marker locus in all environments tested. This suggests that the QTL for GPC is positioned at the *Bmag0807-GBM1063* interval. Similarly, the mean KD score for subNILs carrying the Lacey allele at the *Bmag0807-GBM1063* interval were found to be significantly higher than those carrying the Chevron allele; suggesting that the KD QTL maps in the same position. Although the HD QTL was generally localized in the same *Bmag0807-GBM1021* interval in the St. Paul environment, the effect was negligible (only 0.2 d).

It appears that GPC and KD map clearly within a 2 cM interval on chromosome 6H as compared to the 26 cM previously mapped by Canci et al. (2003). However, we cannot determine whether the same locus also controls FHB. Additional data collection in three more locations is planned for next year.

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Table 1. Chevron/Lacey population means and P-values for *Fusarium* head blight (FHB), kernel discoloration (KD), grain protein content (GPC), and heading date (HD).

Trait	Environments	Chevron	Lacey	Population	Population range	p-value[a]
FHB[b]	St. Paul 2005	1.3	12.2	6.5	1.9-17.7	ns
	Crookston 2005	1.4	9.7	8.0	0.3-28.0	<0.0001
	Morris 2005	1.9	14.7	16.8	2.6-30.5	<0.0001
KD[c]	St. Paul 2005	1.0	3.0	3.6	1.0-5.0	<0.0001
	Crookston 2005	1.0	3.8	3.3	2.0-5.0	<0.0001
	Morris 2005	1.0	3.0	3.3	2.0-5.0	<0.0001
GPC[e]	St. Paul 2005	15.1	12.7	12.8	11.3-15.4	<0.0001
	Crookston 2005	15.9	13.0	13.5	11.4-15.8	<0.0001
	Morris 2005	16.2	14.0	13.6	12.2-16.0	<0.0001
HD	St. Paul 2005	54.0	49.5	51.1	49.0-52.0	<0.0001
	Morris 2005	56.0	53.0	53.8	53.0-55.0	<0.0001

[a] Test for significant variation among $F_{3:5}$ rNILs
[b] FHB severity (% of infected kernels)
[c] KD score on a 1-5 scale (1=no discoloration, 5=heavily discolored)
[e] Days to anthesis

		FHB		HD		GPC		KD	
#rNILs		MO	CR	SP	MO	SP	MO	CR	SP	MO
15		18.4	11.9	50.9	53.8	12.5	13.5	13.0	4.0	3.6
58		14.4	8.6	51.0	53.8	12.5	13.1	13.3	3.7	3.5
3		16.2	**4.4***	51.3*	54.0	12.7	13.3	13.5	4.0	3.3
2		22.6	8.3	51.5*	53.5	**13.4***	**15.1***	**14.9***	**3.3***	**2.5***
19		19.7	**4.6***	51.5*	53.9	**13.7***	**14.8***	**14.3***	**2.6***	**2.6***
3		24.7	**5.9***	51.3*	54.0	**13.6***	**15.1***	**14.7***	**2.6***	**2.8***
6		17.0	8.6	50.8	53.8	12.3	13.2	13.0	3.8	3.5

= Lacey allele; = Chevron allele

Fig. 1. Mean effects for Fusarium head blight (FHB) severity, heading date (HD), grain protein concentration (GPC), and kernel discoloration (KD) across rNILs of similar graphical genotypes are shown below. Traits were measured in Minnesota at St. Paul (SP), Morris (MO), and Crookston (CR) in 2005. Asterisks indicate a significant mean difference (P=0.05) from the mean of the rNILs carrying the Lacey allele across the entire region using LSD; ns, not significant.

FUSARIUM HEAD BLIGHT RESISTANCE IN TETRAPLOID WHEAT

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ABSTRACT

Host resistance has been considered a cost-efficient and environmentally sound strategy to combat Fusarium head blight (FHB); however, progress in developing FHB-resistant wheat cultivars has been hindered by a lack of effective sources of resistance. Although resistant sources have been identified and utilized in hexaploid wheat (*Triticum aestivum* L., 2n = 6x = 42, genomes AABBDD), sources of resistance in durum wheat (*T. turgidum* ssp. *durum* L., 2n = 4x = 28, genomes AABB) are scarce. The objective of this study is to identify germplasm that may be used to enhance FHB resistance in durum wheat. The plant materials comprised 393 accessions of five cultivated subspecies under *T. turgidum*. These subspecies include cultivated emmer wheat [*T. turgidum* ssp. *dicoccum* (Schrank ex Schübler) Thell.], Persian wheat [*T. turgidum* ssp. *carthlicum* (Nevski) Á. Löve and D. Löve], Polish wheat [*T. turgidum* ssp. *polonicum* (L.) Thell.], oriental wheat [*T. turgidum* ssp. *turanicum* (Jakubz.) Á. Löve and D. Löve], and poulard wheat (*T. turgidum* ssp. *turgidum*). These accessions were evaluated for Type II FHB resistance (resistance to the spread of infection) by single spikelet inoculation over three greenhouse seasons. Approximately eighty accessions showed a level of resistance similar to 'Sumai 3', the Chinese common wheat cultivar considered the standard for FHB resistance. Sixty-seven of the 80 accessions identified as resistant to FHB in the greenhouse were further evaluated for FHB reaction in mist-irrigated field nurseries in two locations (Fargo and Langdon, ND). The grain spawn method of inoculation was used. Eighteen accessions exhibited resistance comparable to Sumai 3 in both locations. These resistant tetraploid wheat accessions represent a novel source of FHB resistance and could be utilized directly in durum wheat breeding. Introgression of FHB resistance from these tetraploid wheat accessions to durum is in progress.

WHEAT CULTIVARS WITH IMPROVED RESISTANCE TO FUSARIUM HEAD BLIGHT FOR EASTERN CANADA

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OBJECTIVES

To determine the criteria required to develop FHB-resistant wheat with low DON for the Canadian grain industry

INTRODUCTION

The reaction of wheat lines to fusarium head blight (FHB) is highly variable and is dependent on flowering dates and daily weather conditions. For this reason, it takes many years and test sites to properly assess the FHB reaction of a candidate line or cultivar. One example is the cultivar AC Napier which ranges from best to worst in artificial inoculation tests established in diverse environments, but nevertheless consistently appears somewhat resistant in commercial fields.

It was concluded that in areas where FHB is often severe, the stability of resistance over years and sites should become a key criterion. However, field symptoms are not good predictors of toxin levels in the harvested grain, and correlations may range from poor to good depending on year and site. A visual inspection of grain gives an estimate of fusarium damaged kernels (FDK) that is somewhat better correlated to toxin levels, but still variable enough to be judged inadequate.

Therefore, in areas where the disease is more prevalent, a toxin measurement is the preferred criterion. Eastern Canada has developed a tradition of judging lines for registration on the basis of both toxin (deoxynivalenol, DON) concentration and symptoms.

MATERIALS AND METHODS

Plot size was different among test sites, ranging from single-row plots to 4-row plots, 5 m long. The replicate number at sites was variable but at least 3. Plots were artificially inoculated using a sprayed suspension of *Fusarium graminearum* Schwabe macroconidia (50,000 conidia/ml), corn inoculum or both. Disease symptoms were assessed either by estimating percentage spikelets infected in the field, or else by more precise counts, by harvesting spikes 21 d after inoculation and counting number of spikelets infected. DON was measured using a competitive ELISA method developed by ECORC. The least square means were computed using in order to pool data.

RESULTS

This double-barrelled approach has paid off with lines with improved FHB reaction for the grain industry. One line to be registered (CRGB-O-623.4) has shown stable resistance across many environments (Fig. 1), similar to its FHB resistant parent Nobeoka Bouzu. It is fit for non-rust areas only, and has medium baking quality. It is a CEROM cultivar, and likely to enter the trade under the name Duo. Field observations indicate that this cultivar combines both type 1 and type 2 resistance mechanisms. Type 1 resistance is postu-

lated to be from Frontana LF 320, and type 2, from Nobeoka Bouzu.

The cultivar Nass, from CLRC, Charlottetown, has shown consistent field resistance, despite having somewhat lower type 2 resistance (Fig. 1). This cultivar combines FHB resistance with high yield, although the bread-making properties are not good enough to make it acceptable for milling. Nevertheless, considering that no other FHB resistant cultivars produce a high yield, the cultivar Nass provides evidence that FHB resistance can be combined with high yield. The cultivar was named in honour of its creator, the late Dr Hans Nass.

CONCLUSIONS

The LS means from a general linear model (Fig. 1) lead to the conclusion that field symptoms (observed on spikes) are not adequate to predict DON levels. The FDK count (not shown) provides additional information, but may still leave questions concerning DON levels. Sometimes, DON is present in grain that looks relatively sound. ELISA is providing a rapid method for DON analysis and giving confidence that breeding for FHB resistance can indeed lead to the desired goal, which is improved food safety.

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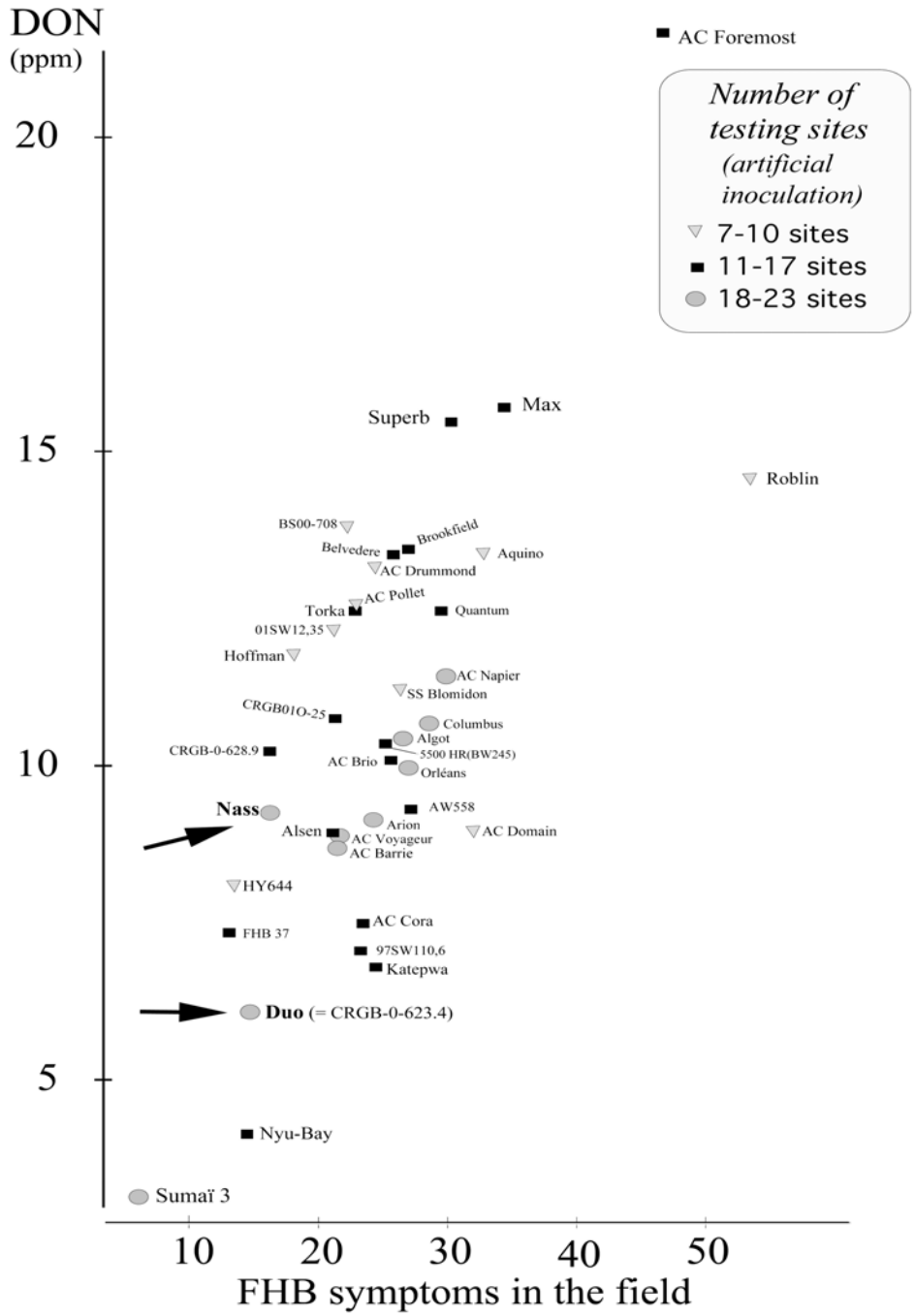

Figure 1. Least square means for fusarium head blight (FHB) symptom severity in the field and deoxynivalenol (DON) data (by ELISA) from multiple test sites and years. The data were obtained from artificial inoculation trials from 1999 to 2004. The sites included Winnipeg, Ottawa, St-Hyacinthe, Quebec City, and Charlottetown.

THE DEVELOPMENT OF SCAB (*FUSARIUM GRAMINEARUM*) RESISTANT VARIETIES OF WHEAT IN NEBRASKA FROM 2001-2005

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ABSTRACT

Fusarium Head Blight (FHB) induced by the fungus *F. graminearum*, affects to varying degrees, approximately one third of Nebraska's yearly wheat crop. The primary objective of the 2005 University of Nebraska breeding program was to select for elite lines of hard red winter wheat which had enhanced agronomic traits and showed resistance to FHB.

In 2005, 125 new crosses were made for FHB resistance using numerous resistance sources primarily from germplasm distributed by Dr. A. McKendry, who is located at the University of Missouri. This germplasm will be advanced to elite line status through modified bulk breeding or backcrossing methods.

A greenhouse screen was used to verify that lines selected from the field screen were truly FHB tolerant and not escapes. The secondary objective was to screen elite hard winter wheat lines in the Regional Germplasm Observation Nursery (RGON). This is a USDA ARS coordinated nursery that screens between 10-30 early generation experimental lines from every public and private wheat breeding program in the Great Plains.

Currently, 95 F2 populations, 44 F3 populations, 1,700 head rows, 98 F5 lines, 57 F6 lines, and 7 F7 lines (3 lines containing some of the Goldfield markers as identified by Dr. Guihua Bai and the Genotyping Center) with diverse sources of FHB tolerance some including Sumai 3 derivatives, are being advanced in the breeding program. Among the most advanced lines, an FHB tolerant line, NE01643 (which had the highest grain yield in the Nebraska State Variety Trial statewide in 2005 and consistently performs well in South Dakota) is tracking for co-release with South Dakota State University in 2006. An additional three lines with high FHB tolerance are also being considered for possible release thereafter. The most interesting of the three lines is NI02425 which has potential for dryland and irrigated production systems. The best lines for FHB tolerance and agronomic performance will be retested in 2006.

MOLECULAR CHARACTERIZATION OF A CHROMOSOME RECOMBINANT CARRYING A FHB RESISTANCE QTL FROM *LOPHOPYRUM PONTICUM*

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ABSTRACT

Robertsonian translocation 7DS.7el$_2$L wheat (*Triticum aestivum L.*) line KS24-2 was characterized as having resistance to Fusarium head blight. However, unwanted linkage drag makes it difficult to be utilized in commercial line development. Our objective is to reduce the amount of 7el$_2$L chromatin, yet retain the FHB resistance. The gene(s) for resistance to FHB is near the distal region of the long arm of 7el$_2$. KS24-2 was crossed to a genetic line of wheat cv. Chinese Spring, in which the *Ph* locus on chromosome 5B was deleted, so that there is induced homeologous pairing and recombination during meiosis in plants that are homozygous for the *Ph* deletion (genotype *phph*). We identified F_2 seedlings that were *phph*, but heterozygous for the long arm using DNA markers. Plants in subsequent generations were genotyped for markers located along 7DL/7el$_2$L and one F_3 derived F_4 plant (line 275-4) lost three *Thinopyrum*-specific markers BE403314, *Xgwm333*, BE406148, which are located in the proximal part of the group 7 chromosome, but retained the 7el$_2$-specific marker loci of the distal half of the 7el$_2$L segment, as revealed with *Xpsr129*, BE445567, and *Xcfa2240*. These three markers were associated by segregation analysis with the FHB resistance. We suggest that the7el$_2$L segment of this wheat line is shorter than that of the donor translocation line KS24-2, but the FHB resistance is retained.

EVALUATION OF BREEDING STRATEGIES FOR ENHANCING FHB RESISTANCE IN BARLEY

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ABSTRACT

Breeding for resistance to Fusarium head blight (FHB) in barley has met significant challenges resulting in relatively slow progress toward the release of new resistant varieties. Among the challenges are the lack of highly resistant sources, quantitative inheritance, large genotype-by-environment interaction, and linkages between resistance genes and undesirable traits. In retrospect, some breeding strategies have proven effective and others less so. Early generation phenotypic selection, particularly on individual plants, has proven to be unsuccessful. Greenhouse screening also has limited utility, primarily since the emphasis in barley is on type I resistance (initial infection) rather than type II resistance (spread in the spike). This is in contrast to wheat where selection for type II resistance in the greenhouse has been fairly effective. On the other hand, large scale screening efforts have identified some promising sources of resistance. All the resistant sources identified to date possess few other attributes necessary for a malting barley adapted to the Upper Midwest. Thus, at least three or more cycles of breeding will be necessary to develop a new malting variety. Extensive field-based testing in inoculated and mist-irrigated nurseries has been the main driver of progress in breeding FHB resistance to date. Breeding programs differ in the allocation of resources to screen for resistance (numbers of breeding lines, replications, and locations). For the first time, in 2005, variety candidates with enhanced FHB resistance were entered into the American Malting Barley Association quality testing program. Numerous quantitative trait loci (QTL) have been identified as potential targets for marker assisted selection (MAS). QTL that appear to have the largest and most consistent effects are linked to heading date, grain protein concentration, or spike morphology. Recent evidence of recombinants breaking some of these undesirable linkages has resulted in the initiation of MAS in collaboration with the USDA genotyping Center at Fargo, ND. Looking forward, some of the greatest gains to be made in FHB resistance in barley will be through the use of breeding data to identify QTL by association mapping. This should permit identification of markers linked to resistance alleles segregating in elite breeding germplasm and significantly expand the use of MAS to develop new malting varieties.

REPORT ON THE 2004-05 PRELIMINARY (PNUWWSN) AND ADVANCED (NUWWSN) NORTHERN UNIFORM WINTER WHEAT SCAB NURSERY

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INTRODUCTION

The PNUWWSN and NUWWSN test the Fusarium Head Blight resistance of primarily soft red winter wheat adapted to the northern areas of North America. There are a few hard and white wheat entries. Each test is conducted in multiple locations with more data collected in the NUWWSN than the PNUWWSN (Table 1). The PNUWWSN had 34 entries and the NUWWSN had 49. For the sake of brevity, this report present data on the FHB traits summarized over locations. Only location means were analyzed and a LSD was calculated using the error mean square after fitting a model with entries and locations. Additional data and analyses, including data by location, can be found in the full report posted on the USWBSI web site or from the corresponding author.

RESULTS

In there was more genotype x location interaction than in past years and ($1-R^2$) was greater than 0.29 for most FHB traits. Based on all FHB traits, the most resistant PNUWWSN entries were Ernie, ILo1-13776, IL01-11934, IL01-6243, and P.99817Rd1-7-5-5-2. The most resistant NUWWSN entries were OH903, OH904, VA04W-474, OH902, IL00-8061, IL00-8530, and P.981517A1-1-5-2.

Table 1. Traits assessed in the 2004-05 PNUWWSN and NUWWSN tests.

Code	Trait	Description	PNUWWSN Locations	NUWWSN Locations
SEV	Disease severity from field tests	% of infected spikelets in an infected head.	IL,KY,MI,MO,VA	IL,KY,MD,MI,MO,NE,NY,OH,ONT,VA
INC	Disease incidence	% of heads with at least one infected spikelets	IL,KY,MI,MO,VA	IL,KY,MD,MI,MO,NE,NY,OH,ONT,VA
IND	Disease index	IND = (SEVxINC)/100	IL,KY,MI,MO,VA,OH	IL,KS,KY,MD,MI,MO,NE,NY,OH,ONT,VA
IS	Number of infected spikelets	Number of infected spikelets from single point inolculation in the field	IN	IN
GH	Greenhouse severity	Same as SEV except from greenhouse	IL,KY,MO,VA	IL, KY, MO
KR	Kernel rating	A visual assessment of the percent infected kernels	IL,KY,MO	IL,KS,KY,MO
PSS	Percent scabby seed	Percent of scabby seed by weight	KY	KY,MD,MO
ISK	Composite of head and kernel traits	ISK Index = .3 (Severity) + .3 (Incidence)+.4 (% FDK or PSS)	IL,MO,KY	IL,KY,MD,MO
DON	DON (vomitoxin)	PPM of vomitoxin in grain	VA	MD,OH,VA

Table 2. Entries in the 2004-05 PNUWWSN

NAME	PEDIGREE	SOURCE
ERNIE	**Early, moderate resistant check**	
TRUMAN	**Most resistant check**	
FREEDOM	**Late, moderate resistant check**	
PIONEER 2545	**Susceptible check**	
981358C1-4-2-1-3-2	Acc3129/Patterson	IN
99608C1-1-3-4	95172/961331A49/4/INW9811//283-1/INW9811/3/Freedom/Acc3128	IN
99751D8-2-3	INW0123/961331A46/5/INW0123//Acc3128/3/9547B1//Patterson/Ernie	IN
99817RD1-7-5-5-2	9560RB1/92201D5//X117/Acc3128	IN
99840C4-8-3-1	961331A46/92201D5//Acc3128/Patton	IN
D9163	PIONEER_2548/3/C5023=(CHELSEA,SWD/B2141//B5219)	MI
E2001	CAYUGA/RAMROD	MI
E2052	CALEDONIA/PNR XW535	MI
E3005	RAMROD/CALEDONIA	MI
E3009	RAMROD/PIONEER_25R26	MI
IL01-11445	IL87-2834-1 / IL95-678	IL
IL01-11934	IL90-6364 / IL94-1909	IL
IL01-13776	IL94-1653 / IL95-2127	IL
IL01-5550	IL95-3245 / Ernie	IL
IL01-6243	IL90-6364 // IL90-9464 / Ning 7840 /3/ IL94-1909	IL
KY97C-0554-02	VA94-54-549/Roane//Kristy	KY
KY98C-1161-03	Patterson/2540//2552	KY
KY98C-1169-06	Patterson/2568//2552	KY
KY98C-1440-01	VA92-51-12/2540//2552	KY
KY98C-1517-01	Roane/Kristy//2552	KY
OH01-5295	IL87-1917-1/HOPEWELL	OH
OH01-6167	OH530/OH585/OH498/34586-20-1	OH
OH01-6964	5088B-D-32-1/HOPEWELL	OH
OH01-7576	38985-11-2/HOPEWELL	OH
OH01-7653	HOPEWELL/OH601	OH
VA04W-389	Ernie/3/P92823A1-1-2-3-5//Roane/Pion2643,F7	VA
VA04W-569	Roane*2//VR95B717/Roane,BC2F5	VA
VA04W-570	Roane*2//VR95B717/Roane,BC2F5	VA
VA04W-571	Roane*2//VR95B717/Roane,BC2F5	VA
VA04W-592	GA891283LE18//Er-Mai 9/GA891283LE18,BC1F6	VA

Table 3. Entries in the 2004-05 NUWWSN

NAME	PEDIGREE	SOURCE
ERNIE	**Early, moderate resistant check**	
TRUMAN	**Most resistant check**	
FREEDOM	**Late, moderate resistant check**	
PIONEER 2545	**Susceptible check**	
981238A1-11-3W	Ernie//91193D1/X117	IN
981517A1-1-5-2	Goldfield/Acc3128	IN
981542A1-10-4-5-6	Acc3128//Ernie/X117	IN
9824C1-26-2	Ernie/PF9052//INW9811/92162B8	IN
99794RA4-14-10	92201D5/4/9547C1//260-1/92367C2/3/INW9824/92829A1	IN
E0001	CLKS_CREAM/MSU LINE D1277	MI
E2017	(D3913,C4530/AUG)/3/(D0331,B9063/HILLSDALE//C113)	MI
E2042	(D3743,I4360/C5317//FRANKENMUTH)/3/(PIONEER_2555,PNR_W3017/PNR_W521	MI
E2043	(DC076,87F_INTCB_ENT#182/AUG//AUG)/3/(PIONEER_2555,PNR_W3017/PNRW521)	MI
E3012	RAMROD/PIONEER_25W33	MI
IL00-1665	IL91-13114 / Y88-3a // Foster / Pontiac	IL
IL00-8061	P8138I1-16-5-50/Foster//IL93-2489	IL
IL00-8530	IL89-1687 // IL90-6364 / IL93-2489	IL
IL01-15511	IL95-561 / IL95-4154	IL
IL01-5943	IL93-3137 / Roane	IL
KS01HW163-4	Trego/Betty sib	KS
KS950910-8-2	KSU94U284/Karl 92//Custer	KS
KY93C-0378-5-2	VA88-52-69/2510//KY84C-48-1	KY
KY96C-0399-5	2510/2580//2540	KY
KY96C-0769-7-1	2552/Roane	KY
KY97C-0304-16	Kristy/2628//2540	KY
KY97C-0574-01	VA94-54-549/L910097//2552	KY
MV-5-46	91-54-22(71-54-147/CK68-15//IN65309C7-18-2-3-2/FFR555W//93-52-55	MD
NE01643	NE94482 (=ARA/ABILENE//NE86488)/ND89744 x Karl92	NE
NE02465	NE95685 (=MO11785/NE87619//NE88492)	NE
NE02495	Wahoo/AP7601	NE
NE02549	KS940935-125-5-2 x Alliance	NE
NE02588	NE94458 (=GK-SAGVARI/COLT//NE86582) x Jagger	NE
NY91017-8080	U1266-4-11/HARUS	NY
NY91028-7085	HARUS/4/CS/A.CURVIF//GLENN/3/ALD/PVN(m-30)	NY
OH01-75	L910097 / IL87-2834-1	OH
OH01-7664	HOPEWELL/OH601	OH
OH902	ZM10782/FREEDOM//30584-37-2/VA91-54-219	OH
OH903	NING7840/GLORY//OH526	OH
OH904	ZM10782/FREEDOM//30584-37-2/VA91-54-219	OH
RCAT13/18	2737W x Ruby/Frontana #1	ONT
RCAT23/1	ACRON x ENA	ONT
RCATL24	RNA/ACRON/Ruby/Frontana #1	ONT
RCATL28	Ruby/Frontana #1 x ACRON/ EX9806 x ACRON	ONT
RCATL31	Ruby/Frontana #1 x ACRON/ AC RON x SVP72017-17-5-10-1	ONT
VA01W-99	FFR525/93-52-55(MSY*3/BALKAN//SAL),F10	VA
VA04W-439	NING 7840/PION2691//Roane(71-54-147/CK68-15//IN65309C7-18-2-3-2),F8	VA
VA04W-474	ROANE/W14/CK9134,H4	VA
VA04W-561	Roane*2//Futai8944/Roane,BC2F5	VA
VA04W-568	Roane*2//W14/Roane/3/2*Roane,BC4F4	VA

Table 4. Traits means for 2005 PNUWWSN. "l", "h" indicate means that are not significantly different from the lowest (l) or highest (h) mean in a column (LSD (0.05)). These are summed in last columns.

NAME	SEV		INC		IND		IS		GH		KR		PSS		ISK		DON	#l	#h
ERNIE	23.3	l	38.3	l	9.4	l	3.8	l	17.6	l	16.3	l	25.6	l	16.7	l	0.6	8	0
TRUMAN	14.4	l	61.5		7.6	l	4.2		4.8	l	12.0	l	11.1	l	27.3	l	0.6	6	0
FREEDOM	24.7	l	80.1	h	16.8	l	3.0	l	24.0	l	28.6	lh	18.6	l	44.1	h	0.8	6	3
PIONEER 2545	41.1	h	77.0	h	28.0	h	10.3	h	33.9	h	45.6	h	50.9	h	52.0	h	2.4	0	8
981358C1-4-2-1-3-2	27.7	l	48.7	l	11.3	l	4.2		24.9	l	20.8	l	13.2	l	29.6	l	0.1	7	0
99608C1-1-3-4	39.8	h	53.0	l	17.9	l	6.7	h	49.6	h	29.4	lh	37.4	h	37.9		1.6	3	5
99751D8-2-3	18.9	l	52.4	l	9.0	l	4.8		14.9	l	31.4	lh	43.7	h	36.2		0.3	5	2
99817RD1-7-5-5-2	19.0	l	53.1	l	9.8	l	2.9	l	19.7	l	24.7	l	36.3	h	25.4	l	0.2	7	1
99840C4-8-3-1	24.5	l	59.6	l	11.6	l	4.7		19.1	l	25.1	l	21.2	l	32.7	l	0.1	7	0
D9163	40.3	h	78.2	h	26.7		8.7	h	23.9	l	32.8	h	19.0	l	49.1	h	0.9	2	5
E2001	40.9	h	82.7	h	30.6	h	7.9	h	18.0	l	38.1	h	34.0		50.5	h	1.2	1	6
E2052	45.2	h	81.5	h	34.8	h	10.3	h	16.1	l	39.9	h	25.0	l	54.7	h	1.8	2	6
E3005	49.0	h	88.6	h	39.8	h	9.4	h	31.9		47.1	h	40.5	h	60.3	h	0.9	0	7
E3009	30.9		75.0	h	22.7		4.6		16.4	l	37.2	h	33.3		51.1	h	0.4	1	3
IL01-11445	25.1	l	54.1	l	10.8	l	8.8	h	17.7	l	11.9	l	17.7	l	24.0	l	0.3	7	1
IL01-11934	21.5	l	47.0	l	8.1	l	5.7		22.5	l	15.2	l	19.9	l	22.7	l	0.2	7	0
IL01-13776	19.1	l	45.9	l	6.8	l	8.3	h	17.6	l	13.3	l	15.1	l	21.2	l	0.4	7	1
IL01-5550	27.6	l	49.0	l	10.0	l	8.8	h	27.9		19.0	l	34.4		29.8	l	0.4	5	1
IL01-6243	20.7	l	46.3	l	8.4	l	9.2	h	22.8	l	15.3	l	21.4	l	22.0	l	0.6	7	1
KY97C-0554-02	30.2		74.2	h	18.7	l	5.6		20.6	l	25.9	l	24.5	l	41.2		0.9	4	1
KY98C-1161-03	37.6	h	73.6	h	23.2		4.3		32.3	h	33.0	h	32.9		48.1	h	1.1	0	5
KY98C-1169-06	28.9	l	55.1	l	14.8	l	8.4	h	20.9	l	30.4	lh	24.8	l	35.3		1.6	6	2
KY98C-1440-01	34.4	h	69.2	h	22.6		8.5	h	34.5	h	35.9	h	34.4		43.3		1.1	0	5
KY98C-1517-01	27.0	l	63.4		15.6	l	1.8	l	15.7	l	19.3	l	13.2	l	31.0	l	0.3	7	0
OH01-5295	23.8	l	57.5	l	11.6	l	3.5	l	18.1	l	26.7	l	29.7		36.2		0.5	6	0
OH01-6167	37.6	h	70.6	h	22.5		8.5	h	52.9	h	25.8	l	21.4	l	42.2		2.0	2	4
OH01-6964	35.1	h	69.6	h	20.5		3.6	l	32.9	h	28.8	lh	15.6	l	42.1		0.4	3	4
OH01-7576	28.1	l	58.2	l	14.8	l	4.8		43.9	h	20.6	l	14.9	l	32.2	l	0.5	6	1
OH01-7653	35.5	h	70.6	h	22.8		8.4	h	38.6	h	28.6	lh	26.6		42.1		0.1	1	5
VA04W-389	33.4		62.2		18.2	l	3.6	l	18.3	l	28.6	lh	25.0	l	42.7		0.3	5	1
VA04W-569	20.5	l	66.5	h	11.4	l	1.8	l	12.9	l	23.0	l	36.7	h	31.8	l	1.4	6	2
VA04W-570	20.3	l	60.0	l	10.4	l	2.6	l	12.3	l	21.7	l	34.2		26.2	l	0.5	7	0
VA04W-571	20.1	l	67.8	h	11.9	l	1.7	l	15.2	l	24.5	l	37.8	h	31.4	l	0.7	6	2
VA04W-592	21.4	l	49.7	l	8.1	l	6.3	h	15.6	l	22.0	l	28.3		26.8	l	0.2	6	1
AVERAGE	29.0		62.9		16.7		5.9		23.8		26.4		27.0		36.5		0.7	4.5	2.4
MAXIMUM	49.0		88.6		39.8		10.3		52.9		47.1		50.9		60.3		2.4	8	8
MINIMUM	14.4		38.3		6.8		1.7		4.8		11.9		11.1		16.7		0.1	0	0
LSD	17.7		26.9		13.8		2.4		26.3		27.7		14.8		19.6				
CV	38.1		31.8		61.0		49.0		63.9		37.6		37.3		27.1				
# LOCATIONS	5		5		6		1		4		3		1		2		1		

Table 5. Traits means for 2005 NUWWSN. "l", "h" indicate means that are not significantly different from the lowest (l) or highest (h) mean in a column (LSD (0.05)). These are summed in last columns.

NAME	SEV		INC		IND		IS		GH		KR		PSS		ISK		DON		#l	#h
ERNIE	18.5	l	50.8		11.3	l	1.4	l	12.5	l	13.8	l	23.9		31.4	l	7.4	l	7	0
TRUMAN	16.1	l	48.2		9.9	l	3.6		9.0	l	17.3	l	17.4	l	25.9	l	1.7	l	7	0
FREEDOM	21.0		65.0	h	16.1		3.1	l	17.2	l	24.1		29.7		40.3		6.4	l	3	1
PIONEER 2545	39.2	h	72.5	h	32.5	h	8.1	h	27.4	l	40.6	h	41.0	h	59.2	h	8.8	lh	2	8
981238A1-11-3W	19.4	l	50.3		14.0	l	1.4	l	6.8	l	16.4	l	26.0		30.5	l	3.6	l	7	0
981517A1-1-5-2	13.0	l	51.8		11.5	l	1.0	l	5.7	l	18.0	l	16.7	l	30.6	l	1.6	l	8	0
981542A1-10-4-5-6	25.7		64.3	h	21.5		1.6	l	16.2	l	34.2	h	41.0	h	44.4		7.5	l	3	3
9824C1-26-2	19.7	l	55.3		13.6	l	4.7		22.0	l	21.1	l	25.8		31.6	l	1.6	l	6	0
99794RA4-14-10	13.6	l	48.0		10.7	l	1.6	l	12.5	l	20.5	l	26.5		30.7	l	1.2	l	7	0
E0001	23.4		57.7		14.5	l	7.1	h	40.4		22.5	l	18.9	l	40.8		10.1	lh	4	2
E2017	34.1	h	59.9		22.5		7.0	h	24.2	l	26.4		26.4		43.3		9.5	lh	2	3
E2042	24.0		57.8		16.6		5.5		16.2	l	24.0		23.8		44.2		9.0	lh	2	1
E2043	31.3	h	67.5	h	22.9		6.4		26.6	l	34.9	h	31.1	h	54.1	h	13.1	h	1	6
E3012	30.6		66.2	h	24.6	h	4.1		18.0	l	34.5	h	29.8		55.6	h	11.7	lh	2	5
IL00-1665	20.2		54.8		13.5	l	4.6		29.0		20.5	l	22.7	l	32.1		1.2	l	4	0
IL00-8061	16.0	l	47.1		9.7	l	2.9	l	19.7	l	13.1	l	16.6	l	24.9	l	0.8	l	8	0
IL00-8530	18.3	l	53.2		13.5	l	2.9	l	12.0	l	9.6	l	14.4	l	25.7	l	1.0	l	8	0
IL01-15511	22.2		55.7		13.8	l	4.4		27.9	l	16.5	l	19.8	l	34.0		1.4	l	5	0
IL01-5943	15.9	l	51.1		10.6	l	5.8		14.1	l	13.7	l	20.0	l	31.3	l	2.7	l	7	0
KS01HW163-4	41.2	h	70.2	h	34.0	h	6.7		43.7		32.2	h	43.8	h	58.2	h	18.3	h	0	7
KS950910-8-2	28.6		58.2		22.8		8.9	h	54.2	h	29.4	h	32.1	h	48.2	h	4.6	l	1	5
KY93C-0378-5-2	27.1		72.1	h	23.9		2.1	l	33.8		32.1	h	29.3		51.4	h	6.3	l	2	3
KY96C-0399-5	28.1		70.5	h	25.7	h	4.7		25.5	l	33.8	h	32.9	h	55.0	h	9.7	lh	2	6
KY96C-0769-7-1	23.0		64.1	h	18.9		2.1	l	25.0	l	24.0		30.0		46.7	h	4.6	l	3	2
KY97C-0304-16	21.6		63.3	h	20.1		4.6		28.1	l	30.7	h	29.5		46.0	h	4.4	l	2	3
KY97C-0574-01	27.4		62.1	h	21.4		3.1	l	34.4		32.2	h	28.8		45.6	h	3.9	l	2	3
MV-5-46	32.6	h	68.2	h	29.8	h	9.5	h	75.9	h	29.8	h	30.4		49.3	h	5.2	l	1	7
NE01643	23.1		56.7		16.1		7.4	h	26.9	l	32.5	h	26.4		43.3		4.8	l	2	2
NE02465	29.0		56.4		24.5	h	8.5	h	29.2		29.8	h	30.6		43.3		1.5	l	1	3
NE02495	29.2		59.7		22.4		5.6		39.8		27.1		26.9		43.5		4.2	l	1	0
NE02549	28.7		58.8		19.7		7.7	h	60.0	h	22.7	l	32.4	h	43.1		8.4	l	2	3
NE02588	31.9	h	64.0	h	25.4	h	6.5		43.6		34.1	h	37.3	h	56.1	h	8.2	l	1	6
NY91017-8080	26.6		58.0		19.6		6.6		10.5	l	23.6		24.3		45.0		11.8	lh	2	1
NY91028-7085	33.8	h	75.2	h	26.9	h	7.1	h	39.9		27.4	h	32.5	h	54.0	h	20.6	h	0	8
OH01-75	22.2		63.5	h	18.5		6.6		22.0	l	20.7	l	20.0	l	40.6		2.3	l	4	1
OH01-7664	25.0		59.0		18.4		5.7		33.0		20.1	l	25.8		41.4		6.0	l	2	0
OH902	20.3		42.2	l	9.0	l	1.2	l	8.2	l	15.5	l	16.7	l	27.1	l	2.3	l	8	0
OH903	10.0	l	32.1	l	6.2	l	0.9	l	14.9	l	12.4	l	10.6	l	17.8	l	0.8	l	9	0
OH904	17.6	l	35.1	l	6.7	l	3.0	l	9.2	l	13.5	l	14.0	l	22.1	l	1.2	l	9	0
RCAT13/18	32.6	h	61.8	h	23.6		6.2		57.9	h	25.7		27.6		48.0	h	8.3	l	1	4
RCAT23/1	25.4		61.7	h	19.2		4.0		24.2	l	22.3	l	22.6	l	44.9		7.0	l	4	1
RCATL24	27.0		63.7	h	21.7		6.0		12.1	l	16.9	l	20.1	l	40.1		17.3	h	3	2
RCATL28	34.3	h	57.6		22.3		7.5	h	33.7		35.9	h	36.1	h	48.3	h	11.2	lh	1	6
RCATL31	18.7	l	51.5		14.2	l	4.1		25.1	l	15.3	l	20.3	l	35.6		2.6	l	6	0
VA01W-99	24.3		62.5	h	21.3		5.0		38.5		26.6		24.9		42.0		1.7	l	1	1
VA04W-439	18.5	l	57.6		14.1	l	4.9		16.1	l	27.9	h	27.6		39.5		2.1	l	4	1
VA04W-474	11.7	l	48.1		8.7	l	2.3	l	3.7	l	18.1	l	17.9	l	27.5	l	0.8	l	8	0
VA04W-561	20.1	l	57.8		14.9	l	1.9	l	7.7	l	19.1	l	23.7		36.1		1.0	l	6	0
VA04W-568	20.2		60.6		15.7	l	2.6	l	3.5	l	14.6	l	23.5		34.8		1.1	l	5	0
AVERAGE	24.1		58.2		18.1		4.7		25.3		23.8		25.9		40.5		5.8		4	2
MAXIMUM	41.2		75.2		34.0		9.5		75.9		40.6		43.8		59.2		20.6		9	8
MINIMUM	10.0		32.1		6.2		0.9		3.5		9.6		10.6		17.8		0.8		0	0
LSD	10.1		13.8		9.5		2.5		25.4		13.4		12.7		14.1		11.9			
CV	16.7		26.6		61.4		57.3		61.3		39.7		30.2		24.9		127.2			
# LOCATIONS	10		10		11		1		3		4		3		4		3			

SPRING WHEAT LINE TOKAI-66, A SOURCE OF HERITABLE KERNEL RESISTANCE TO FUSARIUM HEAD BLIGHT

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ABSTRACT

The spring wheat line 'Tokai-66' (T-66) (PI#382161) was originally developed in Japan in the late 1960's. We tested T-66 in three years of field trials in our *Fusarium*-inoculated high-disease-pressure FHB nursery. While its visual FHB severity was slightly better than average, the percentage of scabby kernels in harvested grain was consistently among the lowest, as was the level of DON. In greenhouse tests, we included T-66 as one parent in two replicated half-diallele trials. The FHB severity score of T-66 was similar to the other FHB-R parents and the FHB-R checks. The F-1 lines with T-66 as a parent were distributed through the range of FHB severity scores. By comparison, the level of scabby kernels in T-66 was lowest of all parents, and the scabby kernels of F-1's involving T-66 were among the lowest of all the F-1's. Using Griffing's formula for calculating general combining ability (GCA) effects in these trials, the GCA of T-66 for FHB severity was significant, and similar to the two other FHB-R parents. In contrast the GCA of T-66 for scabby kernels was the greatest of any of the parents. T-66 has been used as a parent in the ND spring wheat breeding program and several ND advanced lines which include T-66 in their pedigree are in early field trials. (This poster was presented at the Crop Science Soc. of America meeting on November 6-10, 2005 in Salt Lake City, UT.)

FUSARIUM HEAD BLIGHT RESISTANCE IN BARLEY ACCESSIONS FROM THE N. I. VAVILOV INSTITUTE

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ABSTRACT

The deployment of resistant cultivars is one of the best means for combating Fusarium head blight (FHB) in barley (*Hordeum vulgare*). To source additional *Hordeum* germplasm for FHB evaluation, we initiated a cooperative research project with the N. I. Vavilov Institute of Plant Industry (VIR) in St. Petersburg, Russia. The VIR genebank contains over 20,000 barley accessions, comprising 24 *Hordeum* species. Their barley collection is very diverse and contains accessions from regions not represented in the USDA National Small Grains Collection. Since 2003, we evaluated nearly 1100 cultivated (six-/two-rowed as well as winter/spring types) and wild barley accessions from VIR for FHB resistance. These evaluations were made in disease nurseries established in China (Hangzhou) and/or in Minnesota (St. Paul and Crookston). The grain spawn (with ascospores) method of inoculation was used for the nurseries in Hangzhou and Crookston, whereas the foliar spray (using macro-conidia) method was used in the St. Paul nursery. FHB severity assessments were made at the mid-dough stage. From these evaluations, we identified four six-rowed spring-type accessions that exhibited FHB severity levels comparable to Chevron (the six-rowed standard) over multiple locations. The resistant accessions were 15130 and 15133 from Russia and 20731 and 20738 from Afghanistan. Additional screening tests will be made in the field over multiple locations and replicates to confirm the resistance of these accessions and their ability to reduce the accumulation of deoxynivalenol. Moreover, we also will genotype these accessions with molecular markers to determine whether they possess the same alleles as other reported sources of FHB resistance in barley.

COMMON RESISTANCE OF WHEAT TO MEMBERS OF THE *FUSARIUM GRAMINEARUM* SPECIES COMPLEX AND *F. CULMORUM*

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ABSTRACT

Fusarium head blight caused mainly by *Fusarium graminearum* and *F. culmorum* is the most important disease of wheat in Central-Europe. Previous studies clarified that *F. graminearum* is an assemblage of at least 9 geographically separated species (O'Donnell et al., 2004). Among these, *F. graminearum sensu stricto, F. boothii* and *F. vorosii* sp. nov. occur in Hungary (Tóth et al. 2005). Geographical structuring has also been observed in *F. culmorum* (Tóth et al. 2004). Although common resistance of wheat against several *Fusarium* species has been proposed recently (Mesterházy et al., 2005), no data are available in this respect for these recently described species/lineages. In this study, 20 wheat genotypes with highly differing resistance were tested under field conditions by spraying inocula of isolates of eight species of the *F. graminearum* species complex, and 3 *F. culmorum* lineages representing geographically isolated populations in 2003–2004. The severity of Fusarium head blight (FHB), *Fusarium* damaged kernels (FDK), the yield reduction and the deoxynivalenol (DON) contamination were also measured to describe the nature of the resistance. *Fusarium culmorum* isolates were in general more aggressive to wheat than those belonging to the *F. graminearum* species complex. *Fusarium meridionale, F. boothii* and *F. mesoamericanum* were found to be the least pathogenic to wheat. The various wheat genotypes exhibited similar reactions against the different *Fusarium* isolates, indicating that resistance to *F. graminearum sensu stricto* was similar to that for the other species of *F. graminearum sensu lato* examined. This is an important message to breeders as the resistance relates not only to any particular isolate of *F. graminearum*, but similarly to isolates of other *Fusarium* species. This holds true for all the parameters measured. The DON contamination refers only to DON-producing isolates of the *F. graminearum* species complex and *F. culmorum*. Highly significant correlations were found between FHB, FDK, yield loss and DON contamination.

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DEVELOPING A MULTIPLE DISEASE RESISTANCE LINKAGE BLOCK ON WHEAT CHROMOSOME *3BS*

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ABSTRACT

Four wheat (*Triticum aestivum*) fungal resistance genes/QTL have been localized to the region on chromosome 3BS between the markers *Xgwm493* and *Xgwm389*. The durable stem rust (caused by *Puccinia graminis*) resistance gene *Sr2*, Fusarium head blight (caused by *Fusarium graminearum*) resistance QTL *Qfhs.ndsu-3BS*, Septoria tritici blotch (caused by *Mycosphaerella graminicola*) resistance gene *Stb2*, and Stagonospora blotch (caused by *Stagonospora nodorum*) resistance QTL *QSng.sfr-3BS* are all in current cultivars, but the tight repulsion linkage will require recombination events to occur. The initiative to build a 3BS linkage block with all four traits is in progress. Crosses were made between the stem rust resistant parent Ocoroni 86 and the Fusarium head blight resistant cultivar Alsen. Additionally crosses were made between the Septoria tritici blotch resistant parent DH 115 and the Stagonospora blotch resistant cultivar Arina. Recombinant stem rust and Fusarium head blight resistant and susceptible phenotypes were observed in F_2 and verified in F_5 from the cross of Ocoroni 86 x Alsen. The phenotypic marker for *Sr2*, pseudo-black chaff, and DNA markers *Xgwm533* and *Xgwm493* were also used to assist in the selection of *Sr2* and *Qfhs.ndsu-3BS* for recombinant plants. Recombinant plants resistant to both Septoria tritici blotch and Stagonospora blotch from the cross of DH 115 x Arina were observed in F_2 and F_3 and will be verified in the F_4 plants. Respective resistant recombinant plants will be crossed in order to develop plants with resistance to all four diseases.

MAPPING OF QUANTITATIVE TRAIT LOCI FOR FUSARIUM HEAD BLIGHT RESISTANCE IN TWO-ROWED BARLEY

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ABSTRACT

Fusarium head blight (FHB) of barley (*Hordeum vulgare* L.), primarily incited by *Fusarium graminearum*, has been a major production problem in the midwestern part of USA due to deoxynivelenol (DON) contamination of grain. To identify quantitative trait loci for FHB resistance in two-rowed barley, a recombinant inbred line population was developed from the cross ZAU 7/ND16092. ZAU 7 is an early, semidwarf cultivar from Zhejiang University, Hangzhou, China and ND16092 is a line from North Dakota. A single-seed decent population was evaluated for FHB resistance in replicated field experiments over six environments in China and North Dakota. A linkage map of Diversity Array Technology (DArT) and SSR markers was constructed for the population using QTX20. One QTL for FHB resistance was found at the distal region of 2H long arm at all tested locations using both simple interval mapping (SIM) and simplified composite interval mapping (sCIM). The negative effect from ND16092 might be associated with a lax spike gene located in this region of 2H long arm. QTL for FHB resistance were not observed in proximal region of 2H long arm. Four other QTL for FHB resistance were found in one or two environments. Three QTL for DON accumulation, each in a different environment, were identified.

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DISCLAIMER

Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the authors and do not necessarily reflect the view of the U.S. Department of Agriculture.

MAPPING QTLS FOR DIFFERENT TYPES OF RESISTANCE TO FUSARIUM HEAD BLIGHT IN WANGSHUIBAI

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ABSTRACT

Fusarium head blight (FHB), caused by *Fusarium graminearum*, can significantly reduce both grain yield and quality. Growing FHB resistant varieties is an effective means to reduce losses caused by the disease. However, currently used FHB resistance sources are mainly Sumai 3 and its derivatives. Utilization of FHB resistance sources different from Sumai 3 may enrich the genetic pool of FHB resistance sources. Wangshuibai is a FHB resistant Chinese landrace unrelated to Sumai 3. To map QTLs for Type I (initial infection), Type II (spread), and Type III (low mycotoxin deoxynivalenol accumulation) FHB resistance, 139 F_6 derived recombinant inbred lines (RILs) was developed from a cross between FHB resistant Wangshuibai and FHB susceptible Wheaton. More than 1300 simple sequence repeat (SSR) and amplified fragment length polymorphism (AFLP) markers were analyzed in this population. FHB was evaluated in the greenhouses of both Kansas State University and University of Illinois. Type I resistance was evaluated by spraying about 500 conidiaspores over spike and Type II resistance was evaluated by injecting 1000 conidiaspores into a central floret of a spike. Percentage of symptomatic spikelets in each inoculated spike was calculated at 7th days (Type I) and 21st day (Type II) after inoculation. Five QTLs, located on chromosome 3BS, 4B, 4A, 3A, and 1A for Type I resistance were detected. Seven QTLs, located on chromosome 3BS, 7A, 3D, 3A, 1A, and 5A for Type II resistance were detected. Seven QTLs, located on chromosome 3BS, 7A, 1B, 1A, 5D, and 5A for Type III resistance were detected. These QTLs could jointly explain as much as 24.4% of phenotypic variation for Type I resistance, 60.2% of phenotypic variation for Type II resistance, and 55.6% for Type III resistance. Among these QTLs, five of them involved in more than one type of wheat FHB resistance. Two QTLs located on 3BS (near the distal end of 3BS) and 1A contributed to all of the three types of wheat FHB resistance. Three QTLs located on 3BS (close to the centromere), 7A, and 5A showed effects on both Type II resistance and low DON accumulation. The remaining QTLs only showed effects on one type of FHB resistance. Three QTLs located on 3A, 4A, and 4B showed effects on Type I resistance. Two QTLs located on 3A and 3D showed effects only on Type II resistance. Two QTLs located on 1B and 5D showed effect on Type III resistance. The broad-sense heritabilities estimated in 2003-2005 were 34.2%, 82.1%, and 71% for Type I, Type II, and Type III resistance, respectively. New QTLs for FHB resistance identified in Wangshuibai have potential to be used in developing cultivars with enhanced FHB resistance by pyramiding FHB resistance QTL from different sources.