VARIATION FOR RESISTANCE TO FUSARIUM HEAD BLIGHT IN *TRITICUM DICOCCOIDES*

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ABSTRACT

Head blight of wheat (FHB, scab) caused by *Fusarium* spp. is a severe fungal disease problem worldwide. Apart from yield and grain quality losses, the contamination of the harvest with toxic fungal metabolites, known as mycotoxins, is of serious impact. In spite of the fact that several sources for resistance against FHB have been found and utilized in hexaploid wheat, virtually no resistant tetraploid wheat cultivar has been identified so far.

Wild emmer wheat, *Triticum dicoccoides*, previously identified as a rich source for disease resistance genes to several pathogens, was tested for resistance to FHB. Single point inoculations were applied to evaluate a set of 151 *T. dicoccoides* genotypes, originating from 16 habitats in Israel and one habitat in Turkey, for resistance to fungal spread (Type II resistance) in replicated greenhouse experiments. A considerable level of diversity was found among the tested genotypes, the broad sense heritability for Type II FHB resistance was 0.71. Among the eight *T. dicoccoides* lines with the lowest relative infection rates, five originated from the Mt. Gerizim population, and three from the Mt. Hermon population. These two habitats are characterized by a relatively cool and semi-wet climate. Hence, it may be possible that *Fusarium* occurrence in these habitats was responsible for natural selection in favor of resistance.

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DESIGNATING TYPES OF SCAB RESISTANCE: A DISCUSSION

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At a meeting of the Germplasm Introduction and Enhancement Group of the U.S. Wheat and Barley Scab Initiative, held Sept. 12-13, 2002 in St. Paul, MN, the status of terminology for types of scab resistance was reviewed and discussed. The two principal types of resistance described by Schroeder & Christensen (1963), resistance to initial infection and resistance to spread of infection in the head (usually designated types 1 and 2, respectively) have been widely used. However, several additional types have been postulated without agreement among laboratories on either their definitions or in the sequence of numbering (or lettering) to be used. Several other factors contribute to confusion among designated types of resistance (Bushnell 2000). These include:(1) differences among laboratories in the way disease development, toxin accumulation, and kernel yield and quality are measured; (2) the need to deduce the amount of some postulated types of resistance from two measured qualities as, for example, disease severity and yield reduction must be measured to determine tolerance, or toxin concentration and yield loss must be measured to deduce insensitivity to toxin; (3) differences in objectives among laboratories; e.g. a focus on mechanisms of resistance can lead to postulated types of resistance that are not feasible to measure routinely in breeding for resistance; (4) uncertainty about the role of trichothecene toxins in pathogenesis; and (5) limited available information on the physiology and (in most cases) the genetics of resistance.

Lively and candid discussion by the group led to the following results: About half the participants favored continued use of “type 1” to designate resistance to initial infection and “type 2” for resistance to spread in the head. The remaining participants did not favor use of type 1 or type 2 alone to designate the type of resistance. This subgroup recommended that each worker describe both what was measured and the inferred type of resistance in words instead of depending only on use of “type 1” and “type 2”. For resistances other than types 1 and 2, the group was nearly unanimous that it is premature to codify them into a standardized list. Too little is known about them, methods for measuring them are not standardized, and there is lack of agreement among workers on how to designate them. Postulations of resistance mechanisms are valuable as a basis for experimental investigation, but should not be designated by number (or letter) until they are well established and until practical, uniform methods of measuring them are available. The group hopes these conclusions will lead to further discussion by the larger FHB research community.

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INHERITANCE OF FUSARIUM HEAD BLIGHT RESISTANCE (TYPE II) IN NEW WHEAT GERMPLASM CJ 9306 AND CJ 9403

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ABSTRACT

Fusarium head blight (FHB or scab) caused by *Fusarium graminearum* is a worldwide serious disease in wheat. Exploitation and genetic studies of elite resistance sources can speed up the development of resistant cultivars. Two new resistance sources CJ 9306 and CJ 9403 developed in a recurrent selection program of Nanjing Agricultural University, China were crossed to two susceptible cultivars. Experiments with P_1, P_2, F_1, F_2, BC_1 and BC_2 generations for four crosses and with $F_{6:7}$ RILs for one cross were carried out in greenhouse to evaluate FHB resistance to fungal spread within a spike. Single-floret inoculation was conducted at heading and flowering stages, and the inoculated plants were subsequently misted for three days. The number and percentage of scabby spikelets (NSS and PSS) on the 25th day after inoculation were scored. The frequency distribution in F_2s and BC_1s showed continuous with two major peaks and one minor peak between them, indicating that scab resistance in wheat should be a qualitative-quantitative trait. A high level of resistance in CJ 9306 was mainly attributed to co-presence of two genes. The major gene expressed at a moderate to resistant level and was the prerequisite for the expression of the second or minor gene that enhanced the resistance to a high level. In CJ 9403 there might be three major genes and two to three minor genes governing the resistance. The fittest genetic model varied depending on specific crosses. A four-parameter model with additive × dominance interaction provided the most complete and precise elaboration in the two crosses with CJ 9306. A simple additive-dominance model was best fitted for the data from Veery/CJ 9403 and NSS in Norm/CJ9403. For PSS in Norm/CJ 9403, a five-parameter model with additive × additive and dominance × dominance effects seemed to be more adequate than others. The additive effects always significantly increased the resistance and played a major role in the inheritance of scab resistance. The estimates of broad-sense and narrow-sense heritabilities were 60%-86% and 32%-65%, respectively. As new improved germplasm of scab resistance, CJ 9306 not only has a high level of Type II resistance as well as a feature of simpler inheritance, but also possesses well-improved agronomic traits. Therefore, it should be a good choice for breeding scab resistant cultivars. CJ 9403 could be directly applied in production in adapted areas and breeding programs because of its excellent agronomic traits and high yielding potential even if its resistance is a little lower.

SCREENING WINTER AND FACULTATIVE WHEATS FOR FUSARIUM HEAD BLIGHT INFECTION

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ABSTRACT

Severe epidemics of Fusarium head blight (FHB) occur regularly in the Southern Cone region of South America, especially in Argentina, Brazil, Paraguay and Uruguay. Historically the National Wheat Programs have identified sources of resistance to FHB in the spring wheats, including Frontana, Alvarez 110, Encruzilhada, Klein Atlas etc. In the recent years, these have been combined with the Chinese germplasm derived from Sumai#3 and Chuan Mai#18 to achieve higher level of resistance in the wheat programs.

In order to broaden the spectrum of resistance to include winter and facultative wheats, CIMMYT's regional program, based in INIA La Estanzuela, Uruguay, screens local and introduced germplasm under naturally occurring and artificially inoculated conditions. The level of naturally occurring FHB infection during 2001 was extremely severe, which allowed excellent evaluation of the introduced germplasm (Table 1).

Table 1. Classification of winter and facultative wheat germplasm for FHB, 2001.

Source	Total entries	Scab classification*				
		R	MR	MS	S	VS
4th WONIR	112		2	3	29	77
10th FAWWON	62		6	8	18	30
Georgia	86		6	8	45	27
Louisiana**	14	1	3	4	5	1
Kansas	118		2	20	81	15
Oklahoma	30		2	2	17	9
Texas	20		2	3	5	10
Mexico (winter)	19			8	5	6
TOTAL	**461**	**1**	**23**	**56**	**205**	**175**
%	**100**	**0.2**	**5.0**	**12.1**	**44.5**	**38.0**

**R=Resistant; MR= Mod. Resistant; MS= Mod. Susceptible; S= Susceptible; VS= Very susceptible*
*** Collection of parent lines from the crossing block*

The evaluation of the CIMMYT international nurseries and winter wheat germplasm from various collaborators in the US, confirms the presence of large variability in the FHB resistance. In spite of the fact that FHB infection was uniform throughout the cycle, later heading germplasm tended to show lower levels of infection. The lines from this group will need to be checked through artificial inoculations. Other lines, early or intermediate for their heading, selected for lower level of FHB infection are presented in Table 2.

While these results confirm the resistance of some parent lines (Shou Chou), they also demonstrate that other lines such as CIMM1FHB#5, Coker 960208 and ND 2928 are, in fact, moderately susceptible under Uruguayan field conditions. Two lines (X950412-F-7 and Pioneer 26R61) were rated at par with local check *INIA Tijereta,* considered to be moderately resistant and will need to be confirmed in future evaluations. Several other lines (Bezostaja, Irneria/ Mukkab hib., Star/Bwd, OK 98637 and X950446-F-1), rated moderately resistant to moderately susceptible, represent germplasm with very wide genetic backgrounds which can be useful in the breeding programs.

Table 2. Fusarium head blight reaction of selected facultative and winter wheat lines

Entry	Heading	FHB		Origin
		(1-5/1-5)	Reaction	
I. TIJERETA (Local check)	**E***	**22**	**MR****	**Uruguay**
ND2928	E	24	MS	Louisiana
CIMM1FHB#5	E	25	MSS	Louisiana
SHOU CHOU	E	11	R	Louisiana
OK97508	E	24	MS	Oklahoma
TX98V6610	E	23	MS	Texas
I. TORCAZA (Local check)	**I**	**32**	**MS**	**Uruguay**
BEZOSTAJA	I	22	MRMS	10FAWWON
STAR/BWD	I	22	MRMS	10FAWWON
IRNERIA/MUKKAB HIB.	I	22	MRMS	10FAWWON
SULTAN	I	23	MS	10FAWWON
93435-1-10	I	13	MS	Georgia
UGA 931463E27	I	23	MS	Georgia
PIONEER 26R61	I	12	MR	Louisiana
APD99-5627	I	23	MS	Louisiana
COKER960208	I	14	MS	Louisiana
LA422	I	13	MS	Louisiana
9388D22-1-3	I	23	MS	Louisiana
X950337-II-2	I	23	MS	Kansas
X950446-F-1	I	22	MRMS	Kansas
X950412-F-7	I	11	RMR	Kansas
OK98637	I	22	MRMS	Oklahoma
OK95571	I	14	MS	Oklahoma
I. GORRION (Local check)	**L**	**45**	**S**	**Uruguay**

** E= Early (150d), I= Intermediate (160d), L= Late (170d)*

Field screening of facultative and winter wheat germplasm under naturally occurring epidemics of FHB at a hot spot site such as La Estanzuela, Uruguay, provides an excellent opportunity to identify new sources of resistance. These, in addition, can also be screened for foliar blights and leaf rust diseases. CIMMYT, in collaboration with the National Agriculture Research Institute, INIA, is trying to incorporate these and other sources of FHB resistance into locally adapted wheats. The preliminary results are very encouraging.

TYPES I, II AND FIELD RESISTANCE TO FUSARIUM HEAD BLIGHT IN WINTER AND SPRING WHEAT GERMPLASM

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OBJECTIVE

The objective of this research was to evaluate and determine the relationship among Type I, II and field resistance in spring and winter wheat germplasm that we had previously identified as having potentially useful levels of resistance to scab.

INTRODUCTION

Fusarium graminearum Schwabe (teleomorph *Gibberella zeae* (Schwein.), also known as scab, is a devastating disease of wheat and barley in warm and humid regions of the world. Host plant resistance has long been considered the most practical and effective means of control but breeding has been hindered by a lack of effective resistance genes and by the complexity of the resistance in identified sources. No source of complete resistance is known, and current sources provide only partial resistance, often in unadapted types. The identification of different sources of resistance in winter wheat through a systematic evaluation of accessions maintained in the National Small Grains Collection at Aberdeen, ID has been identified as a key objective of the US Wheat and Barley Scab Initiative's (USWBSI) germplasm research area. As such, approximately 4600 winter wheat accessions have been evaluated at Missouri. Additionally, spring and winter wheat germplasm has been introduced into the United States through a collaborative effort established between the USWBSI and CIMMYT. Both initiatives have resulted in the identification and introduction of wheat germplasm with high levels of Type II resistance. Less is known, however, about the Type I resistance in these lines, how the two types of resistance are correlated, and whether those types of resistance relate to field resistance.

MATERIALS AND METHODS

Germplasm: Germplasm was selected for evaluation from two sources. Winter wheat germplasm was acquired from the National Small Grains Collection in Aberdeen, ID and was kindly provided by Dr. Harold Bockelman. Germplasm was selected that had functional levels of Type II resistance in each of 3 successive cycles of greenhouse evaluation. Winter wheat germplasm included was from China, South Korea, Japan and Italy and included land races, cultivated genotypes and cultivars. Spring wheat germplasm included most genotypes introduced into Missouri in 2000 through the CIMMYT/USWBSI collaboration. Lines included were from the CIMMYT breeding program and included advanced breeding lines and wide crosses. Genotypes also included lines introduced from China and from Romania. Of the Romanian wheat introduced, 6 had a winter wheat growth habit.

Greenhouse Evaluations: Vernalized seedlings were arranged in a split-plot design with genotype as the main plot and type of resistance as the sub-plot. For each accession, 10 plants per treatment were planted and the experiment was replicated six times. For evaluation of Type II resistance, plants were inoculated at first anthesis with 10µL of a macroconidial suspension of *Fusarium*

graminearum concentrated to 50,000 macroconidia/mL. Inoculum was placed in a single central floret at first anthesis using an Oxford 8100™ repeat dispensing syringe. For all inoculations, a single isolate was used which had been previously determined to be aggressive on the resistant cultivar, Ernie. Plants were incubated in a mist chamber (100% relative humidity) for 72 h post-inoculation to promote disease development and then returned to the greenhouse bench. Ratings for Type II resistance (disease spread in the spike) were made at 21 d after inoculation. A Fusarium head blight index (FHBI) was also determined at 21 d post-inoculation as the ratio of diseased spikelets to total spikelets in the inoculated head.

For evaluation of Type I resistance, heads were again inoculated with a macroconidial suspension of *Fusarium graminearum* concentrated to 50,000 macroconidia/mL. Inoculum was sprayed directly on the head at full anthesis using a Pulmo-Aide nebulizer as the power source and an atomizer (Model 163, DeVilbiss Sunrise Medical, Somerset, PA15501-0635, USA). Inoculum was delivered to each head, spraying one side and then the other. Plants were incubated in a mist chamber as described above. At 10 d post-inoculation heads were rated for symptoms of Fusarium head blight. Total spikelets in the head were recorded followed by the number of spikelets in the head showing disease. Incidence was determined as the total number of spikelets on the inoculated head showing disease symptoms. The Type I FHBI rating for each head was determined as the number of spikelets with disease divided by the total number of spikelets on the head. Ratings were taken again at 21 d post-inoculation to determine the scab index for the head. The 21-d rating (total number of infected spikelets/total spikelets in the inoculated head) provided an estimate of severity on the inoculated head.

Field Evaluations: The field scab index was determined from unreplicated spray inoculations or winter wheat germplasm. Individual rows were inoculated at 75% anthesis with a macroconidial suspension of *Fusarium graminearum* concentrated to 50,000 macroconidia /mL using a CO_2 backpack spray system. Plants were maintained under overhead mist irrigation throughout the inoculation period (approximately 2 wk). Twenty heads from each row were evaluated for symptoms of scab 18-21 d post-inoculation. Infected spikelets were counted on each head. Incidence was determined as the number of heads with visible symptoms of disease. Severity was determined as the ratio of diseased spikelets to total spikelets in the inoculated heads. The field scab index was calculated as incidence*severity.

RESULTS AND DISCUSSION

Data presented in Table 1 are those from winter wheat germplasm screened with high levels of Type I, II and field resistance. Superior lines included land races and cultivated lines from China, South Korea and Italy. Of 45 lines evaluated, 12 lines had good levels of Types I and II resistance, coupled with good field resistance. Data for spring wheats with good levels of resistance are given in Table 2. Of 57 wheat genotypes introduced through the CIMMYT collaboration in 2000, 23 genotypes had excellent levels of Type II resistance (< 10%) while 15 had good Type I resistance (<40%). Nine genotypes combined good levels of both Type I and Type II resistance. Four of these lines were introduced from Romania, while two were introduced from China. Type I and Type II resistances were not highly correlated. Complete data for all lines evaluated will be available at the Scab Forum.

Name	Designation	Origin	Heading date	Mean spikelets by accession	Greenhouse Point Inoculation 21 d		Greenhouse Spray Technique 10 d		Greenhouse Spray Technique 21 d		Field Scab Index
					21 d		10 d		21 d		
Average			320	13.4	2.7	0.21	7.7	0.57	8.6	0.64	30
LSD at 0.05			3.1	1.6	1.3	0.09	2.9	0.23	2.9	0.19	18
c.v.%			3.9	11.0	41.4	37.6	33.3	30.7	29.2	26.6	

	Greenhouse spray (incidence)				Greenhouse point	
	10 Day	21 Day	10 day	21 day	21 day	21 day
CIMMYT 2000 - Pedigree/Designation	# diseased	# diseased	FHBI	FHBI	# diseased	FHBI

RESISTANCE IN HEXAPLOID WHEAT TO FUSARIUM HEAD BLIGHT

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When Fusarium head blight (FHB) re-emerged as a major disease of wheat in North America about 15 years ago, it was evident that most cultivars were susceptible. Small grain breeders and pathologists began working with Sumai 3 and related resistant lines from China as sources of resistance. Although there seem to be no races of *Fusarium graminearum* that have adapted to a particular source of resistance, numerous examples from other pathogens suggest that we should be cautious about reliance on one source of resistance. Moreover, Sumai 3 is not completely resistant. The discovery of other resistance genes may allow us to create genotypes with a greater level of resistance than any that are currently known. To further these objectives, the USWBSI created a Germplasm Introduction and Enhancement (GIE) research area "to identify new sources of FHB resistance and to facilitate the utilization of resistant germplasm".

ACCOMPLISHMENTS

Participants in the USWBSI, as well as workers in other areas of the world, have identified many wheat lines resistant to FHB (e.g., 2,4,6,9). Snijders hypothesized 3 main pools of resistant germplasm (12). The GRIN database includes 34 lines that have FHB index values of 2 or 3. McKendry and Bestgen identified 17 lines from the CIMMYT/USWBSI collaborative germplasm effort that had 1 spikelet or less blighted following point inoculation (McKendry, personal comm.). No line of common wheat is completely resistant, but several have a high degree of resistance.

SCREENING TECHNIQUES

Most germplasm screening efforts employ point inoculation, in which an aqueous suspension of conidia is placed in a single floret at the middle or near the tip of the spike. Point inoculation is designed to detect resistance to spread of the fungus throughout the spike, based on spread of symptoms, referred to as Type II resistance. Resistance to initial infection (Type I resistance) may be as important as Type II resistance. If weather is favorable for infection throughout anthesis and early grain fill, there may be multiple primary infection events, leading to severe head blight without the need for spread of the pathogen through the rachis. Workers evaluate germplasm for Type I resistance by spraying heads at full flowering with a spore suspension.

Mesterhazy proposed 5 types of active resistance mechanisms in wheat to *Fusarium* infection, based on his own and previous work (6). This classification of resistance types (now referred to as Types I to V) has had a major influence on how researchers view resistance and its genetic control. As more has been learned about the interaction between *Fusarium* and wheat, there are questions about how some of these types of resistance are defined and measured. Participants at a recent workshop of the GIE group agreed that Type I and Type II

resistance are reasonably straightforward, as first defined (8). The other 3 types of resistance (III-V) pose problems.

Type III resistance (resistance to kernel infection) is a valid concept, but operationally it poses problems. What is the appropriate way to measure it? Point inoculation is not suitable. If a line has a high degree of Type II resistance, then the kernels from a head on which a single floret was inoculated will show a low frequency of infection and damage because the fungus never reached them or reached them too late to cause visible damage. For evaluation of Type III resistance, every kernel evaluated should be exposed to infection. Spray inoculation might be more suitable, but a line with resistance that impedes progress of the fungus from the stamen to the ovary could mask whatever resistance or susceptibility kernels might have to invasion. There is a more fundamental question about this kind of resistance. Does it refer to the ability of the fungus to penetrate a kernel or to the degree to which the fungus ramifies the grain? If Type III resistance is meant to measure differences in the amount of mycelium in grain, then a test that measures fungal biomass in kernels should be used.

Mesterhazy defined Type V resistance (active resistance mechanism “e”) as “resistance to toxins in ears by decomposing them” and cited Miller *et al*. 1985 (7) and Snijders and Perkowski 1989 (sic) (13) as sources (The citation of the Snijders and Perkowski paper is incorrect in Mesterhazy’s paper. The correct citation is given in the reference list below). Miller at al. suggest 2 reasons for low DON levels in grain: the plant has factors that prevent formation of toxin, or factors that promote degradation of toxin. Accurate measurement of resistance to toxin accumulation poses difficulties. If lines have Type I or Type II resistance, or resistance to invasion of kernels from other tissues in the head, then they would have lower levels of DON in kernels compared to lines that lacked these forms of resistance. Detection of resistance to toxin accumulation requires that grain from different wheat lines has not only been equally exposed to infection, but that fungal biomass in the grain can be measured and related to DON content (7).

Correlation analysis of DON content versus fungal biomass in kernels may reveal that some lines have less DON than would be expected. Such lines should be investigated for presence of genes that act to influence the accumulation of DON. It would be desirable to combine these genes with genes for other types of resistance. However, selection for resistance to DON accumulation against a background of resistance that reduces the frequency or extent of kernel infection would be difficult. Genes for resistance to DON accumulation would be ideal candidates for marker-assisted selection.

The concept of tolerance (Type IV, or “d” in Mesterhazy’s list of active defense mechanisms) has traditionally been applied to foliar or root diseases, in which grain is not directly infected, but its mass and quality are reduced by the stress of infection of vegetative organs. A tolerant cultivar sustains less yield reduction for a given severity of disease than an intolerant cultivar. What does tolerance mean for a pathogen that infects reproductive organs? If grain is relatively sound despite severe head blight symptoms, the plant may have a resistance mechanism that interferes with invasion of the grain. Mesterhazy used yield relative to uninoculated controls as a measure of tolerance, i.e. if a group of lines had equivalent head blight severity scores, but differed substantially in relative yield, he considered the line(s) with higher yield to

be tolerant. Without direct evidence of comparable timing and extent of kernel invasion (fungal biomass per kernel), conclusions about tolerance in FHB must remain tentative.

INHERITANCE OF RESISTANCE

Discovery and phenotypic characterization of resistant lines are only the first steps in using germplasm. Resistance must be incorporated into cultivars adapted for each region where FHB is a threat. Breeders can use germplasm without knowing the genetic basis of resistance, but the work is more efficient if they have this knowledge.

Original accessions are often heterogeneous for resistance and it is necessary to reselect from this germplasm to obtain lines that consistently express resistance. Once this has been done, genetic studies can be undertaken.

Most studies published so far have evaluated Type II resistance in response to point inoculation. Two or more genes condition resistance in Sumai 3 or its derivative, Ning 7840. A major QTL has been mapped to 3BS (1,14). Results from a number of studies with various wheat lines indicate QTLs for FHB resistance may reside on most chromosomes of the wheat genome (see 14). Many of these genes show additive action. This suggests that as new genes are identified in other sources of resistance, they will act additively, or in more complicated manners, with the genes already in hand, to give higher levels of resistance. Even moderately susceptible lines have contributed useful genes for resistance (1,3,11).

FUTURE DIRECTIONS OF THE GIE PROGRAM

If Types I and II resistance are inadequate to protect the crop, resistance to kernel invasion or to DON accumulation could provide another layer of protection. The germplasm program of the USWBSI should give more attention to discovery of germplasm with these other forms of resistance. With respect to Type I and Type II resistance, emphasis should shift to genetic characterization of the germplasm already in hand. We need to know more about inheritance of resistance in various sources, the uniqueness of their genes, and how genes interact to affect the resistance phenotype.

We may be approaching the limit of Type II resistance. Several lines show only slight necrosis in the inoculated floret. It will be difficult to detect higher levels of Type II resistance when such sources are combined. It may be more useful to combine other types of resistance with a high degree of Type II resistance. Evaluation of the same wheat lines by both point and spray inoculation suggests that different genes control Type I and Type II resistance. If yet other genes control resistance to kernel infection and toxin accumulation, then it should be possible to combine all of these into a single cultivar. For reasons outlined above, phenotypic selection would not work. This is clearly a project that requires marker-assisted selection technology. First we need to carefully characterize, phenotypically and genetically, resistance other than Type II, and then find reliable markers for these genes.

To accomplish this efficiently within the USWBSI, I propose creation of a coordinated program analogous to programs in other research areas. Many accessions with Type II resistance have been identified in hexaploid wheat. The most resistant accessions, particularly any that lack the major QTL on 3BS (see e.g., 5), need to be thoroughly studied genetically. We also need to identify sources of Type I resistance and sources of resistance to kernel invasion and DON

accumulation. For this, we need to develop reliable methods of phenotypic screening. These will likely be more complicated and costly than the point inoculation technique currently used to identify Type II resistance.

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NOVEL SOURCE OF TYPE II RESISTANCE TO FUSARIUM HEAD BLIGHT

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ABSTRACT

Sources of resistance in wheat (*Triticum aestivum*) to Fusarium head blight (FHB) of wheat are limited despite extensive screening of germplasm since Arthur first reported this disease in 1891. Wheatgrass has been demonstrated to be an important resistance source for wheat leaf rust, stem rust, and powdery mildew diseases. Here we report the excellent resistance to FHB of *Lophopyrum elongatum* (2n = 2X =14, genome EE).

A series of Chinese Spring- *L. elongatum* substitution lines from 1E(1A) to 7E(7D) except 4E(4D) and 5E(5A) (provided by J. Dvorak, Department of Agronomy and Range Science, University of California, Davis, CA), were evaluated for Type II resistance to *Fusarium graminearum* in a greenhouse, February-April 2002. The recipient parent Chinese Spring was also included in the experiment. In a completely randomized design, 12 – 24 plants per line were evaluated for disease severity (DS), the percentage of diseased spikelets in inoculated spikes. The mean DS of Chinese Spring was 41%. The mean DSs of the substitution lines ranged from 5% - 74%. Pairwise comparisons of means showed that three lines had significantly higher DSs than Chinese Spring. They are 3E(3D), 2E(2D), and 6E(6A) with respective DSs of 74%, 71%, and 62%. Three lines had DSs that were significantly lower than Chinese Spring. They are 7E(7A), 7E(7B), and 7E(7D), with respective DSs of 5%, 5%, and 6%. The disease did not spread beyond the inoculated spike in all tested plants in these three lines. Our data shows that the 7E chromosome of *L. elongatum* conditions Type II FHB resistance. Chinese Spring itself has resistance to FHB. The resistance of Chinese Spring may be located on chromosomes 2D, 3D, and 6A, because when these chromosomes were replaced with their respective homoeologous *L. elongatum* chromosome, these substitution lines were more susceptible to *Fusarium graminearum* than Chinese Spring.

The experiment is being repeated in the greenhouse, October-December, 2002. Results to date are consistent with our results in the test of February-April, 2002.

EVALUATION OF THE NATIONAL SMALL GRAINS COLLECTION OF BARLEY FOR RESISTANCE TO FUSARIUM HEAD BLIGHT AND DEOXYNIVALENOL ACCUMULATION

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INTRODUCTION

Barley is a major crop in the Red River Valley of Minnesota, North Dakota and Manitoba. Production peaked in the 1980s and decreased to its lowest level in 30 years in 1999. Many factors have contributed to barley's decline in the region, including Fusarium head blight (FHB) (McMullen, *et al*).

Grain affected by FHB, caused primarily by *Fusarium graminearum*, has reduced quality and may be contaminated with unacceptable levels of deoxynivalenol (DON) (Salas, *et al.*). With the advent of the FHB epidemics of the 1990s, some brewing companies imposed strict limits on the levels of DON present in grain going to the malt houses. Barley exceeding the DON specifications is relegated to feed grade with a corresponding drop in price. The risk involved with potential grade reduction is a primary factor in declining barley hectarage. This has lead to increased awareness of the problem and a need for barley varieties that are resistant to FHB and to toxin accumulation.

In 1998, Busch Agricultural Resources, Inc. (BARI) scientists began a study to identify sources of resistance to FHB and resulting DON accumulation. This effort concentrated on screening the entire 6-rowed spring barley collection held by the National Small Grains Collection. Public and private barley breeders may be able to utilize accessions identified as having high levels of resistance to disease and toxin accumulation to improve malting barley.

MATERIALS AND METHODS

Initially 7,475 accessions were received from the USDA-ARS, National Small Grains Collection, Aberdeen, Idaho. Field evaluation sites included Casselton, ND and Crookston, MN in 1998, Park River and Osnabrock, ND in 1999 and 2000, and Park River in 2001. Accessions were planted mechanically as single, non-replicated rows. In July 1998 accessions with few or no visible symptoms were selected and then hand harvested in August. In 1999, 2000 and 2001 all accessions were hand harvested regardless of visible symptoms. In 2000 percent FHB was determined in July by counting number of infected kernels vs. total kernels on 10 heads. In all years harvested material was transported to Fort Collins, CO and threshed. Grain samples were submitted to the Barley DON Diagnostic Laboratory located in the Department of Cereal and Food Sciences at North Dakota State University for DON analysis (Tacke and Casper). Selections were made for further testing based on both percent FHB and ppm DON.

Selected accessions were planted in the greenhouse in Fort Collins, CO during the winters of 1999/2000 and 2000/2001. Heads were inoculated at anthesis with an isolate of *F. graminearum* collected from Midwest-grown barley. Inoculations were carried out late in the day and plants placed in clear plastic chambers equipped with a humidifier for approximately 36 hours. Heads were rated for percent FHB by counting the total number of kernels and the number of visibly infected kernels at 14- and 21-days post inoculation. In the 2000/2001 season, greenhouse samples also were submitted for DON testing.

RESULTS AND DISCUSSION

A number of the 7,475 rows planted in 1998 were winter, hulless, black, hooded, dwarf, 2-rowed or other types of barley and were discarded. Because all the barley at the Crookston nursery appeared to be free of disease, selections were made and harvested from plants at the Casselton nursery. The same accessions were harvested at Crookston. Based on visual selection, the top 98 accessions and 2 checks were submitted for DON analysis from both locations. DON levels ranged from non-detectable to more than 40 ppm across locations.

Eighty-two accessions were advanced to the 1999 field screening. Toxin levels were generally lower than the previous year and ranged from 0.2 to 11.0 ppm (avg. 2.9 ppm) at Park River and non detectable to 12.5 ppm (avg. 1.9 ppm) at Osnabrock.

Fifty-six accessions were selected for testing in the greenhouse in 1999/2000. An additional 13 accessions that had been discarded in 1998 but were identified as resistant by North Dakota State University (NDSU) in 1999 were returned to the study. Twelve accessions did not develop any symptoms of FHB. In all, 9 of the 56 accessions were eliminated from further testing based on high DON, high FHB or very poor agronomic traits. All of the NDSU selections that were returned to the study were carried over to the next field season.

In 2000 sixty accessions were planted at Park River and Osnabrock. Toxin analysis and disease ratings were possible on 48 accessions. Toxin levels averaged 0.3 ppm DON at Park River but averaged 1.6 ppm at Osnabrock. None of the 12 accessions that had 0% FHB in the greenhouse remained completely free of disease. However, these and other accessions low in disease continued to perform well.

A total of 47 accessions were tested in the greenhouse in 2000/2001, 15 from previous years' tests and 32 selected by NDSU in 2000. Disease averaged 18% and was high (25 to 95%) in many accessions. Toxin levels ranged from non-detectable to 13.6 ppm with a mean of 2.1 ppm. All accessions were carried over for another year of field screening.

DON level for 2001 averaged 2.4 ppm. Levels ranged from 0.6 ppm to 3.3 ppm for the 15 accessions remaining in the study.

We have selected 15 accessions to recommend for inclusion in breeding for resistance (Table 1). Several of these accessions selected were already reported to have resistance and have been used in various breeding programs over the years (including the 2-rowed types, Svanhals and Svansota). Steffenson and Scholz also selected a number of accessions in their studies (Steffenson and Schulz). As a result of these studies, several new accessions can be

added to the list of resistant germplasm. The geographic sources of these accessions cover four continents. However, at least 2 accessions from the US have Chevron in their background. Another US selection has a Svanhals parent. The full diversity of the most resistant accessions needs to be assessed through molecular genetics. This can further focus breeding efforts on numerous sources of resistance.

Table 1. Accessions from the National Small Grains Collection selected for resistance to Fusarium head blight and Deoxynivalenol.

ACCESSION	CIho	PI	ORIGIN	PEDIGREE
Mammoth Winter	CIho 220		Ukraine	
Wisconsin Pedigree	CIho 835		USA	
Abyssinninan Intermediate	CIho 2414		Ethiopia	Selection from PI 25674
Hietpas 3	CIho 6611		USA	Selection from Oderbrucker
Seed Stocks 1148-1	CIho 6613		USA	
Markhinstz	CIho 7279	PI 149782	Russia	
Iowa 5286	CIho 9539		USA	Manchuria, CI 4471/Chevron
ELS 6402-302	CIho 12904	PI 298751	Ethiopia	
1948D		PI 371317	Switzerland	
UNA 8392		PI 477854	Peru	
Cross	CIho 2492		Sweden	
Svansota	CIho 1907		USA	No. 456/Svanhals
Svanhals	CIho 2274		Sweden	Selection from PI 5474
Peatland	CIho 2613		USA	Selection from same landrace as Chevron
Chevron	CIho 1111		Switzerland	Selection from landrace

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FUSARIUM HEAD BLIGHT TYPE II RESISTANCE OF A SPRING WHEAT POPULATION DERIVED FROM A HUNGARIAN WINTER WHEAT

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ABSTRACT

Fusarium head blight (FHB) of wheat, caused mainly by *Fusarium graminearum*, has plagued farmers in the spring wheat region for nearly a decade, causing substantial loss. Disease management by crop rotation, tillage, or fungicides has been marginally successful at best. The best long term solution to the FHB problem in this region is by incorporation of resistance into adapted cultivars. Several cultivars resistant or moderately resistant to FHB have been released in the region over the past several years, and many more advanced lines are being tested. The resistance to FHB in nearly all such adapted spring wheats has come from Chinese germplasm sources such as Sumai 3 and its derivatives. Other sources of resistance need to be explored. In the 1980's, Akos Mesterhazy in Hungary identified non-Chinese lines which showed resistance to FHB. He produced several advanced resistant lines by intercrossing these sources. One of us (RWS) obtained several of his lines in 1996 and crosses were made to adapted spring wheats. A population derived from crossing Mesterhazy's line 'Ringo Sztarr/Nobeoka Bozu' by the ND cultivar 'Grandin' was advanced by single seed descent to F-6, selecting only for spring habit. We grew 182 lines from this population in the greenhouse in two randomized replicates. At anthesis, ten spikes per replicate were inoculated by single spikelet inoculation and then given 3 days of intermittent mist treatment. At 3.5 weeks post-anthesis, FHB symptom development on each spike was scored on a 0-100% severity scale. FHB severity scores of the 182 lines ranged from 8% to 85%. Based on FHB severity, 14 of the 182 lines were resistant as or better than our standard resistant check line 'ND2710' and other best Sumai 3 derived lines. We conclude that lines derived from other resistance sources may be as resistant to FHB as those from presently used Chinese germplasm. This can serve to diversify the germplasm base for FHB resistance.

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PROPOSED CHROMOSOMAL LOCATION OF FHB RESISTANCE GENES IN ADDITIONAL SETS OF DURUM DISOMIC SUBSTITUTION LINES DERIVED FROM DIFFERENT *T. DICOCCOIDES* ACCESSIONS

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ABSTRACT

Fusarium head blight (FHB), caused mainly by *Fusarium graminearum*, has been a serious disease problem on spring wheat in North Dakota and surrounding states for nearly a decade. North Dakota is the principal durum growing state in the USA and durum has been especially hard hit by FHB. Development of cultivars with FHB resistance has been much slower for durum than for the spring bread wheats, in part because the best known sources of FHB resistance are in hexaploid backgrounds and effective transfer to the tetraploid durum seems to be difficult. In the 1980's, USDA geneticist L.R. Joppa had produced a set of durum disomic chromosome substitution lines derived from a wild emmer (*Triticum dicoccoides*, TDIC) selection "Israel A", identified for high grain protein levels. We recently reported (Crop Sci. 42:637-642) the finding of FHB resistance on chromosome 3A in the durum disomic substitution line from this series. Other researchers have found molecular markers for this gene. In searching for potential sources of FHB resistance, we previously had screened 290 accessions of TDIC from the USDA world collection, and we had identified several lines with useful levels of FHB resistance. Two of these accessions were used to produce new sets of chromosome substitution lines in 'Langdon' durum following the method used for the original TDIC chromosome substitution series. The purpose of the present study was to determine which chromosomes held the resistance loci in these FHB resistant TDIC accessions. Each substitution line was grown in replicated trials in the greenhouse and inoculated at anthesis with *Fusarium graminearum* by the single spikelet method. FHB response was determined visually 3.5 weeks after inoculation. LDN(DIC) substitution lines representing five different chromosomes (1A, 3A, 5B, 7A, 7B) had significantly less FHB than the Langdon checks. The other LDN(DIC) lines showed intermediate responses, not significantly different from the Langdon durum parent. The five TDIC chromosomes substituted in the lines with significantly reduced FHB are proposed as sites of FHB resistance genes in these two accessions.

WILD EMMER, *TRITICUM DICOCCOIDES*, AS A SOURCE OF FHB RESISTANCE FOR TETRAPLOID AND HEXAPLOID WHEATS

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ABSTRACT

Wild emmer, *Triticum turgidum* L. var. *dicoccoides* (a.k.a '*T. dicoccoides*') (TDIC), is wild tetraploid wheat found throughout the Middle East. Because it shares the AB genome with modern durum (*T. turgidum* L. var. *durum*), it readily crosses with it and also crosses with hexaploid wheat - with some care as to choice of parent. TDIC has long been known as a source of novel disease resistance including genes for resistance to stem rust, stripe rust, leaf rust and powdery mildew, among others. Our research with TDIC as a source of FHB resistance began as two separate lines of inquiry which have since come together. One area of study was the evaluation of a set of disomic chromosome substitution lines developed by Leonard Joppa in the 1980's to study a gene for high grain protein. In each of these lines, one chromosome pair from TDIC replaces the corresponding pair in a durum background. We tested this set of substitution lines for FHB. The entire story of this aspect of the work was recently reported in Crop Science (42:637-642). We found a major FHB resistance gene on 3A and a major gene on 2A that appears epistatic to FHB resistance. Somewhat less strong resistance was present on 1A and 6B. Another research group at NDSU has identified molecular markers for the 3A QTL. Research on the 2A epistatic locus is currently underway. Concurrently, we began screening the USDA world collection of TDIC for FHB. Between 1995 and 1997 we tested 449 TDIC collections. Of these, 33 (7.3%) showed levels of FHB substantially lower than durum check lines. About half have held up as moderately to highly resistant upon repeated testing. In direct crosses between these TDIC selections and durum, the FHB resistance appears in the offsprings but along with many undesirable traits. From that point the two lines of research joined together. Two TDIC accessions from among those confirmed as having FHB resistance were selected upon which USDA cytogeneticist Leonard Joppa would base new sets of durum disomic substitution lines. An abstract elsewhere in this proceeding describes that process and the results. From the new series of substitution lines those which showed FHB scores significantly lower than the Langdon durum background parent were those with TDIC chromosomes 1A, 3A, 5B, 7A, and 7B; however none of these by itself is likely to confer adequate resistance to FHB. In a diallele study on the original disomic lines, we found that the strong resistance gene on 3A showed positive combining ability with those on 1A and 6B. In a field trial in 2002 we also confirmed that the FHB resistance on 3A will effectively reduce FHB in a hexaploid wheat background. Several of the chromosomes in these substitution lines are not among those previously recognized to bear FHB resistance genes in hexaploid wheat.

EFFICIENCY AND EFFICACY OF MARKER ASSISTED SELECTION OVER PHENOTYPIC SELECTION FOR FHB RESISTANCE IN DURUM WHEAT

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ABSTRACT

We are studying the efficiency of Marker Assisted Selection (MAS) for Type II Fusarium Head Blight (FHB) resistance in two durum wheat populations derived from a Chinese bread wheat source 'Sumai 3'. This study is based on the hypothesis that for a trait such as FHB, the use of molecular markers for MAS would reduce the time involved in selection along with a reduction in cost. The first population consisted of 1,814 $F_{2:4}$ lines that were developed from crossing a cultivar Ben to Sumai3/Sceptre//D88816 line. The second population consisted of 320 $F_{2:5}$ that were derived from backcrossing cultivar Lebsock to the line Lebsock//Sumai3/Lebsock. These two populations were screened for FHB resistance in the greenhouse in spring 2002 by inoculating the heads with *Fusarium graminearum* and later scoring the diseased heads. Screening for the resistance QTL located on the chromosome 3BS was done using the microsatellite locus *Xgwm533*. In the greenhouse evaluation, 1,124 lines in the first population and 180 lines from the second population were found resistant with scores of less than 21%. Microsatellite marker identified the resistant QTL in 524 lines from population I and131 lines from population II. Apart from the lines that were found to be resistant in the presence of marker and susceptible in its absence, some lines had the marker but were susceptible and some did not have the marker and still were resistant to the disease. Lines representing these four groups will be evaluated in summer 2003 in a replicated scab nursery and the efficacy of both the selection methods will be calculated. In the present study the molecular data showed that using MAS the population size could have been reduced from 1,814 lines in population I to 524 and 131 from 320 lines in population II, thus saving a significant amount of greenhouse space, resources and time in screening. We calculated the efficiency of each selection process so far and found that, with MAS it took us 44 working days to screen the two populations with an approximate cost of $1.43 per data point and with phenotypic selection in the greenhouse; it took 141 days with an approximate cost of $0.99 per data point. In terms of time involved, MAS was found to be 3.2 times quicker saving 97 days. With the use of high throughput non-denaturing gel system, the efficiency of MAS in terms of time and labor will be higher at much reduced cost. In our next step of study, we plan to advance the agronomically desirable lines by repeated backcrossing to cultivars Ben and Lebsock. These lines will then be further analyzed for their FHB resistant phenotype and agronomic performance.

PUTATIVE SOURCES OF FUSARIUM HEAD BLIGHT RESISTANCE IN SPRING WHEAT IDENTIFIED FROM THE USDA SMALL GRAINS COLLECTION

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INTRODUCTION

The use of host resistance will likely be one of the major components in managing Fusarium head blight (FHB) of wheat. Germplasm improvement and varietal development for FHB resistance will depend upon continued efforts in discovery and characterization of diverse resistant sources. Since 1998, we have evaluated 4,400 accessions of spring wheat from the USDA small grains collection. This report summarizes putative sources of resistance that underwent three consecutive years of field evaluations in replicated trials.

MATERIALS AND METHODS

Spring wheat germplasm from the USDA collection (Aberdeen, ID) were first evaluated in a preliminary screening nursery (PSN). This is a non-replicated nursery with entries planted into rows (ca. one meter in length). ND 2710 and BacUp were used as resistant checks and Sonalika and Wheaton as susceptible checks with a check-to-entry ration of 1:28. The nursery was inoculated with infected corn grain and conidial suspension. Details in nursery management, inoculation, and data collection were as described previously (Zhang *et al*. 2000; 2001). Accessions or plants within an accession with a low FHB index (incidence*severity) and/or low percentage of Fusarium damage kernels (FDK) were selected. Selections were further evaluated in subsequent years in elite germplasm nurseries (EGN). Entries of EGN were planted in row-plots with three replicates and arranged into split-plot design, with maturity as the main plot and genotype as the subplot. Maturity groups were determined based on days between planting and flowering: early ($\leq$55), intermediate (55-65), and late ($\geq$66).

RESULTS AND DISCUSSION

In each of the three evaluation years, high disease pressure was generated by artificial inoculation and mist-irrigation. FHB indices on the susceptible checks (Wheaton and Sonalika) consistently exceeded 80%.

Table 1 lists selections with low FHB indices ($\leq$ 40%) and low FDK ($\leq$ 40%). This group of materials generally exhibited stable low FHB reaction over years. Selections with low FHB indices ($\leq$ 40%) but high FDK (>40%) or high FHB indices but low FDK ($\leq$40%) are given in Table 2. The first group of materials from Table 2, namely Sin Chunaga, Norin 61 and several other lines originated primarily from Japan, consistently showed lower disease indices, but high visual FDK ratings. Kernels rated as FDK in this group were mostly bleached, but remained plump. A recent study on Fusarium infection of seed harvested from the 2002 field FHB screening nursery suggested that discoloration (bleaching) of plump kernels might not be

due to fungal infection (Zhang and Jin, unpublished). Although FHB indices of second group in Table 2 were high, lines in this group generally had low FDK scores and might contribute useful resistance/tolerance genes in breeding.

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Table 1. Spring wheat germplasm selections with low Fusarium head blight indices and low percentage of damaged kernels.

		FHB index (%)				FDK (%)		
Accession	ID	2000	2001	2002	**mean**	2000	2001	**mean**
PI 382161	Tokai 66	10.5	5.9	12.8	**9.7**	16.0	12.0	**14.0**
PI 382154	Nyu Bai	13.7	9.4	11.9	**11.7**	15.0	15.3	**15.2**
PI 382153	Nobeoka Bozu	17.0	9.8	12.2	**13.0**	13.8	15.7	**14.7**
	ND 2710 (CK)	14.2	10.2	14.7	**13.0**	22.8	19.5	**21.2**
	Sumai 3 (CK)	15.0	17.0	15.8	**15.9**	28.3	25.0	**26.7**
PI 182568	Norin 43	21.5	16.1	28.0	**21.9**	46.7	30.0	**38.3**
PI 462151	Shu Chou W. 3	18.5	29.8	18.9	**22.4**	18.8	20.0	**19.4**
Citr 12002	Renacimiento	25.5	21.2	22.2	**23.0**	41.7	36.7	**39.2**
PI 345731	Tezanos P.P.	20.2	19.3	30.0	**23.2**	20.0	23.0	**21.5**
PI 519790	274-1-118	19.8	28.9	24.8	**24.5**	40.0	35.3	**37.7**
PI 434987	Estazuela Young	22.2	23.4	31.2	**25.6**	58.0	15.5	**36.8**
CItr 5103	274	14.5	27.2	35.1	**25.6**	19.0	23.3	**21.2**
PI 81791	Sapporo H.K.J.	24.4	40.0	15.9	**26.8**	21.7	17.7	**19.7**
PI 596533	**BacUp (CK)**	35.0	16.5	30.2	**27.2**	31.9	17.0	**24.5**
PI 192660	Prodigio Italiano	39.0	20.2	24.1	**27.8**	22.5	18.7	**20.6**
PI 185380	Prodigio Italiano	35.7	25.0	26.2	**28.9**	27.5	16.0	**21.8**
PI 285933	Chudoskaja	36.2	30.5	22.5	**29.7**	26.7	21.7	**24.2**
PI 382167	16-52-9	10.9	28.5	49.8	**29.7**	23.3	18.0	**20.7**
PI 351256	Japon 2	21.2	36.2	38.3	**31.9**	41.7	18.7	**30.2**
CItr 12021	Centenario	32.2	31.8	34.0	**32.6**	41.7	25.0	**33.3**
PI 163429	PI 163429	27.0	31.8	40.2	**33.0**	30.0	28.7	**29.3**
PI 351221	Newthatch Sel.	34.0	35.2	30.0	**33.1**	20.0	20.0	**20.0**
PI 382144	Encruzilhad	29.6	37.3	35.6	**34.2**	45.0	33.3	**39.2**
PI 294975	Artemowska	24.5	67.0	16.8	**36.1**	20.0	20.0	**20.0**
Citr 13136	Rio Negro	42.8	35.2	32.5	**36.8**	50.0	23.7	**36.8**
PI 264927	220	31.9	48.3	30.5	**36.9**	16.7	20.7	**18.7**
PI 104131	Excelsior	36.3	34.2	44.5	**38.3**	21.7	14.0	**17.8**
Citr 17427	16-52-2	34.5	43.5	39.9	**39.3**	33.3	24.0	**28.7**
PI 83729	Magyagovar 81	51.0	49.8	18.2	**39.7**	46.0	22.7	**34.3**
PI 469271	**Wheaton (CK)**	87.6	88.8	83.5	**86.6**	93.3	83.7	**88.5**

Table 2. Spring wheat germplasm selections with low Fusarium head blight indices and high percentage of damaged kernels or vice versa.

		FHB index (%)				FDK (%)		
Accession	ID	2000	2001	2002	**mean**	2000	2001	**mean**
	ND 2710 (CK)	14.2	10.2	14.7	**13.0**	22.8	19.5	**21.2**
	Sumai 3 (CK)	15.0	17.0	15.8	**15.9**	28.3	25.0	**26.7**
PI 596533	**BacUp (CK)**	35.0	16.5	30.2	**27.2**	31.9	17.0	**24.5**
PI 469271	**Wheaton (CK)**	87.6	88.8	83.5	**86.6**	93.3	83.7	**88.5**
PI 478282	**Sonalika (CK)**	87.1	84.3	87.8	**86.4**	76.4	84.0	**80.2**
PI 382140	Abura	15.1	17.7	17.5	**16.8**	38.3	47.3	**42.8**
PI 182561	Sin Chunaga	22.7	22.0	22.0	**22.2**	86.7	76.7	**81.7**
PI 182586	Norin 43	30.0	20.7	26.7	**25.8**	50.0	56.7	**53.3**
PI 197128	Shinchunaga	17.0	36.7	27.8	**27.2**	80.0	76.7	**78.3**
PI 182583	Chuko	19.1	39.8	23.2	**27.3**	78.8	75.0	**76.9**
PI 411132	Gogatsu-Komugi	28.3	24.2	34.2	**28.9**	77.5	66.7	**72.1**
PI 351816	Froment Du Japon	32.0	29.0	33.0	**31.3**	70.0	33.3	**51.7**
PI 182591	Norin 61	37.0	29.2	31.0	**32.4**	66.7	41.0	**53.8**
PI 192634	Trintecinco	53.7	23.1	49.2	**42.0**	41.7	34.7	**38.2**
PI 351743	CLUJ 49-926	40.2	65.0	21.5	**42.2**	26.0	30.0	**28.0**
PI 185843	Surpresa	57.7	39.7	30.2	**42.5**	42.5	23.3	**32.9**
PI 362437	III/14-B	35.5	52.3	43.0	**43.6**	33.3	22.3	**27.8**
PI 264998	628	43.7	47.8	40.5	**44.0**	30.0	26.7	**28.3**
PI 264940	111a	55.3	47.7	30.0	**44.3**	41.7	16.0	**28.8**
PI 168727	Bahiense	36.8	28.2	68.5	**44.5**	25.0	19.7	**22.3**
Citr 2492	Manchurian	57.0	23.0	56.0	**45.3**	25.0	23.5	**24.3**
PI 344467	Oncativo Inta	48.0	45.3	48.5	**47.3**	38.8	31.7	**35.2**
PI 256958	Academia 48	42.4	59.0	46.2	**49.2**	20.0	26.7	**23.3**
PI 163439	PI 163439	59.0	38.6	51.0	**49.5**	40.0	27.7	**33.8**
PI 132856	Mentana	48.3	41.8	60.3	**50.1**	36.7	29.7	**33.2**
PI 351993	Z.88.54	54.2	57.2	39.5	**50.3**	30.0	25.3	**27.7**
PI 168716	Klein Condor	54.8	64.7	32.2	**50.6**	35.0	31.0	**33.0**
PI 349534	533B	54.3	67.3	34.0	**51.9**	26.7	20.0	**23.3**
PI 351476	Vaulion	55.8	75.3	35.4	**55.5**	25.0	40.0	**32.5**
PI 184512	H 51	76.7	37.7	52.5	**55.6**	33.3	19.0	**26.2**
PI 344465	Laureano Alv. L.	48.3	62.5	61.2	**57.3**	36.7	30.0	**33.3**
PI 192219	Hatvani	48.8	79.3	44.4	**57.5**	36.7	25.3	**31.0**
CItr 11215	Belgrade 4	39.6	84.3	54.3	**59.4**	35.0	29.0	**32.0**
PI 344454	Buck Austral	64.5	81.5	34.6	**60.2**	28.8	30.0	**29.4**
PI 351187	Taillens Velu Sel.	46.5	79.5	58.1	**61.4**	26.7	34.0	**30.3**
PI 113949	Stepnjachka	63.0	70.8	50.7	**61.5**	38.3	24.7	**31.5**
PI 519798	PF 79782	35.9	67.0	82.5	**61.8**	27.5	24.0	**25.8**
PI 225160	Mentana	39.0	68.0	81.2	**62.7**	30.0	27.5	**28.8**
PI 584934	Whestphalen	62.5	69.3	58.0	**63.3**	41.3	31.7	**36.5**
PI 362043	Arnaut de Toam.	59.6	61.3	79.5	**66.8**	23.3	33.3	**28.3**
PI 352000	Z.89.37	52.2	82.2	68.5	**67.6**	26.7	43.3	**35.0**
PI 192229	Gran Com. Ung.	57.7	77.8	74.5	**70.0**	31.7	40.0	**35.8**
PI 113948	Kooperatorka	77.7	88.3	61.7	**75.9**	26.3	43.3	**34.8**