

## Genetic dissection of a major Fusarium head blight QTL in tetraploid wheat

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### Abstract

The devastating effect of Fusarium head blight (FHB) caused by *Fusarium graminearum* has led to significant financial losses across the Upper Midwest of the USA. These losses have spurred the need for research in biological, chemical, and genetic control methods for this disease. To date, most of the research on FHB resistance has concentrated on hexaploid wheat (*Triticum aestivum* L.) lines originating from China. Other sources of resistance to FHB would be desirable. One other source of resistance for both hexaploid wheat and tetraploid durum wheat (*T. turgidum* L. var. *durum*) is the wild tetraploid, *T. turgidum* L. var. *dicoccoides* (*T. dicoccoides*). Previous analysis of the 'Langdon'-*T. dicoccoides* chromosome substitution lines, LDN(Dic), indicated that the chromosome 3A substitution line expresses moderate levels of resistance to FHB. LDN(Dic-3A) recombinant inbred chromosome lines (RICL) were used to generate a linkage map of chromosome 3A with 19 molecular markers spanning a distance of 155.2 cM. The individual RICL and controls were screened for their FHB phenotype in two greenhouse seasons. Analysis of 83 RICL identified a single major quantitative trait locus, *Qfhs.ndsu-3AS*, that explains 37% of the phenotypic or 55% of the genetic variation for FHB resistance. A microsatellite locus, *Xgwm2*, is tightly linked to the highest point of the QTL peak. A region of the LDN (Dic-3A) chromosome associated with the QTL for FHB resistance encompasses a 29.3 cM region from *Xmwm14* to *Xbcd828*.

**Abbreviations:** *bcd*, barley cDNA clone; *cdo*, oat cDNA clone; FHB, Fusarium head blight; LDN(D), Langdon-D genome disomic substitution; LDN(Dic), Langdon-*T. dicoccoides* disomic substitution; LDN(Dic-3A), Langdon-*T. dicoccoides* disomic chromosome 3A substitution; LRR, leucine-rich repeat; *mwm*, barley genomic clone; QTL, quantitative trait locus; RFLP, restriction fragment length polymorphism; RGA, resistance gene analogue; RICL, recombinant inbred chromosome line; sCIM, simplified composite interval mapping; SIM, simple interval mapping; SSR, microsatellite,  $V_i$ , interaction effect variation;  $V_m$ , main effect variation;  $V_p$ , phenotypic effect variation

### Introduction

Annual worldwide grain production of durum wheat (*Triticum turgidum* L. var. *durum*) is estimated at 27.5 million metric tons. This estimate varies slightly due to environmental and pest problems such as drought, flooding, and diseases including Fusarium head blight (FHB) (*Fusarium graminearum*), leaf rust (*Puccinia recondita*), and tan spot (*Pyrenophora tritici-repentis*),

as well as insects and rodent pests (Blanco *et al.*, 1998). The presence of FHB has been a major problem in North Dakota since 1993. The economic loss due to FHB in North Dakota alone, in the period 1993–1997, totaled over three billion dollars (McMullen *et al.*, 1997). FHB is an important issue from a production point of view since over 80% of the acreage of durum produced in the USA is in North Dakota (U.S. Department of Agriculture, 2000).

The most prevalent causal agent of FHB is the species *Fusarium graminearum*, referred to as *Gibberella zeae* in its sexual stage. Aside from FHB, this fungus is also associated with stalk rot in maize and root rot in small grains, including durum, hexaploid wheat (*T. aestivum*), oat (*Avena sativa*), barley (*Hordeum vulgare*), and rye (*Secale cereale* L.) (McMullen *et al.*, 1993). *Fusarium* head blight-resistant varieties of durum wheat are needed since cultural and management practices have not provided adequate control against this disease. At present, the most effective fungicide application treatments reduce damage from FHB by about 50% (McMullen *et al.*, 1999).

Sources of genetic resistance in wheat that have been analyzed by other research groups include, but are not limited to, 'Sumai-3', 'Ning 7840', and 'Frontana'. Sumai-3, a Chinese hexaploid wheat, is one of the most prominent sources examined for type II resistance (spread of disease in the infected heads). Waldron *et al.* (1999) identified 5 regions of the genome that were associated with type II resistance. The major QTL identified in this study were later verified in different populations (Anderson *et al.* 2001). Bai *et al.* (2000) examined six wheat cultivars as sources of type II FHB resistance; these sources included Sumai-3 and Ning 7840, the latter containing Sumai-3 in its pedigree. It was determined that the resistance from Sumai-3 and Ning 7840 is inherited primarily through additive-dominant gene actions. The heritability was significant and accounted for a large portion of the genetic variation in the populations examined. Frontana, a variety from Brazil, is being explored as a source for type I resistance (resistance to the initial onset of infection; Tamburic-Ilicic *et al.*, 2000). Genetic analysis on these, along with other sources of FHB resistance in both wheat and barley, have resulted in the identification of several QTL associated with FHB resistance (Kolb *et al.*, 2001).

One species that appears to show promise as a source for resistance to FHB in durum wheat is the non-adapted tetraploid wheat *T. turgidum* L. var. *dicoccoides* (AABB,  $2n = 4x = 28$ ). This wild species has also been shown to be a good genetic source for increased grain protein concentration and other agronomic traits (Steiger *et al.*, 1996; Joppa *et al.*, 1997; Chee *et al.*, 2001). However, *T. dicoccoides* (Dic) has many drawbacks due to its exotic nature. It is normally unadapted, has a propensity for shattering, and is later-maturing (Cantrell and Joppa, 1991).

Chromosome substitution lines permit the examination of each individual chromosome and its influ-

ence on plant characteristics (Joppa 1993; Joppa and Williams, 1998). A series of Langdon-*T. dicoccoides* [LDN(DIC)] chromosome substitutions were included in trials evaluating durum germplasm for FHB (Stack *et al.*, 1999). The LDN(DIC-3A) substitution was identified as resistant to FHB Type II infection (Stack *et al.*, 1999). The LDN(DIC) recombinant inbred chromosome lines allow a focused investigation on a smaller area of the genome and a detailed mapping and analysis of certain quantitative traits or morphological characteristics.

Objectives of this research included the development of a molecular linkage map for chromosome 3A, the identification of genomic regions significantly associated with FHB resistance, the identification of molecular markers linked to FHB resistance, and the identification of lines with FHB resistance that can be used as sources for breeding programs.

## Materials and methods

A population of 83 RICL individuals for chromosome 3A of *T. dicoccoides* in a 'Langdon-16' durum background was used in this study (Joppa, 1993). This population, along with Langdon-16 (LDN-16), LDN (Dic-3A), and several durum check lines were screened over two greenhouse seasons with three replicates per greenhouse season using a randomized complete block design.

Lines were inoculated with three highly aggressive isolates of *Fusarium graminearum* (R010, R1267, and R1322) (Stack and McMullen, 1985) at anthesis following the procedures described by Waldron *et al.* (1999) with a few modifications. The plants were misted once with a misting head in the evening and tented overnight for three consecutive nights after inoculation. For each inoculated head, the severity and incidence was scored 21 days after inoculation to evaluate the spread of infection. The score for infection in this study was based on a visual (0–100%) scale (Stack and McMullen, 1995).

Resistance in the host to the initial infection of the head is classified as Type I FHB resistance. Because Type I resistance suffers from large environmental effects, Type II resistance was examined in this study. Type II resistance is the resistance to spread of disease in the head once infection has occurred. Type II resistance is the primary research focus of most investigators and has been shown to be rather stable and

easily tested in experimental environments (Bai *et al.*, 2000).

The molecular markers utilized in this study were those reported to map on Triticeae chromosome 3A (Nelson *et al.*, 1995; Röder *et al.*, 1998). The markers utilized for the analysis of the LDN(Dic-3A) RICL population included restriction fragment length polymorphisms (RFLP), simple sequence repeats (SSR), and resistance gene analogues (RGA). Survey filters containing DNA from the parental lines, LDN (Dic-3A) and LDN-16, digested with the restriction enzymes *EcoRI*, *EcoRV*, *DraI*, *HindIII*, and *XbaI* were used to screen for RFLP polymorphisms. Once these polymorphisms were identified, they were screened on the entire population filters. Probes were labeled by random hexamer labeling with <sup>32</sup>P-labeled dCTP (Feinberg and Vogelstein, 1983). In the screening of the RFLP, a total of 33 clones were initially screened for polymorphisms. Sources of the RFLP probes were oat cDNA clones (*cdo*), barley genomic clones (*mwg*), and barley cDNA clones (*bcd*).

A series of wheat microsatellite primers were also screened according to the protocol published in Röder *et al.* (1998). Products were separated on a 6% denaturing polyacrylamide gel and silver-stained. Band patterns were scored for polymorphisms. Any primers that produced polymorphisms were then tested on the full RICL population.

RGA primers are PCR-based primers derived from common motifs identified in disease resistance genes. The primer sequences for RGA were based on sequences published by Chen *et al.* (1998). Reactions were run based on the specifications of Chen *et al.* (1998), and band patterns were visualized on silver-stained polyacrylamide gels.

A linkage map was developed using Mapmaker (Lander *et al.*, 1987) through the Group, Compare, Try, and Ripple commands. A total of 19 markers were mapped on chromosome 3A using the LDN(Dic-3A) RICL population. Phenotypic trait data were statistically analyzed by SAS (SAS Institute, 1990), while MQTL software was used to perform simple interval mapping (SIM) and simplified composite interval mapping (sCIM) for QTL analysis (Tinker and Mather, 1995a, b). Parameters for analysis were a chromosome walking pace of 1 cM and an experiment-wise Type I error rate of 5%, with 1000 permutations run to calculate the significance threshold. For a single environment, the test statistic can be converted to a LOD score by multiplying with 0.22 or dividing by 2 ln(10) (Tinker and Mather, 1995b).

Heritability of type II FHB resistance in the LDN(Dic-3A) RICL population was calculated with the components of variance from the combined RCBD analysis, using the following formula:

$$h^2 = \frac{\sigma_G^2}{\sigma_G^2 + \frac{\sigma_{GE}^2}{e} + \frac{\sigma_\epsilon^2}{re}}$$

The heritability was then used along with the phenotypic variance explained by the most significant locus ( $Vm_p^2$ ) to determine the amount of genetic variance ( $Vm_g^2$ ) explainable in the population through the formula  $Vm_g^2 = Vm_p^2/h^2$ .

## Results

### Mapping data

From an initial screening of 33 RFLP probes, a total of 13 clones were determined to possess potential polymorphisms on the parental DNA digests and were then screened on the full population of the 83 RICL for LDN (Dic-3A). Of the thirteen clones screened on the population filters eight yielded clear polymorphisms (8/33 or 24%). From eight SSR primer sets tested, five gave clear polymorphisms in the population (5/8 or 63%). The six RGA primers screened resulted in four clear polymorphisms (4/6 or 67%).

A linkage map (Figure 1) was constructed with 19 loci, viz. 8 RFLP loci, 5 SSR loci, and 6 RGA loci. Of the 19 loci mapped to chromosome 3A, only one (RGA locus *Xs2as3b*) showed a significant deviation from the expected segregation ratio of 1:1 (the actual ratio was 28:53) according to a  $\chi^2$  test for fitness. The length of chromosome 3A was determined to be 155 cM, calculated using the Haldane mapping function (distance threshold set at 80 cM) in the Mapmaker software. The average distance between markers on 3A is 7 cM. The largest gap on the map is 42 cM between the RFLP locus *Xbcd115* and the RGA locus *Xs2as3b*, found on the long arm of the chromosome. This chromosome length for 3A is slightly more than half the length previously reported (Nelson *et al.*, 1995).

### *Fusarium head blight resistance screening*

Type II disease severity means of parental lines and the entire mapping population for two seasons is presented in Table 1. Differences in FHB severity between the

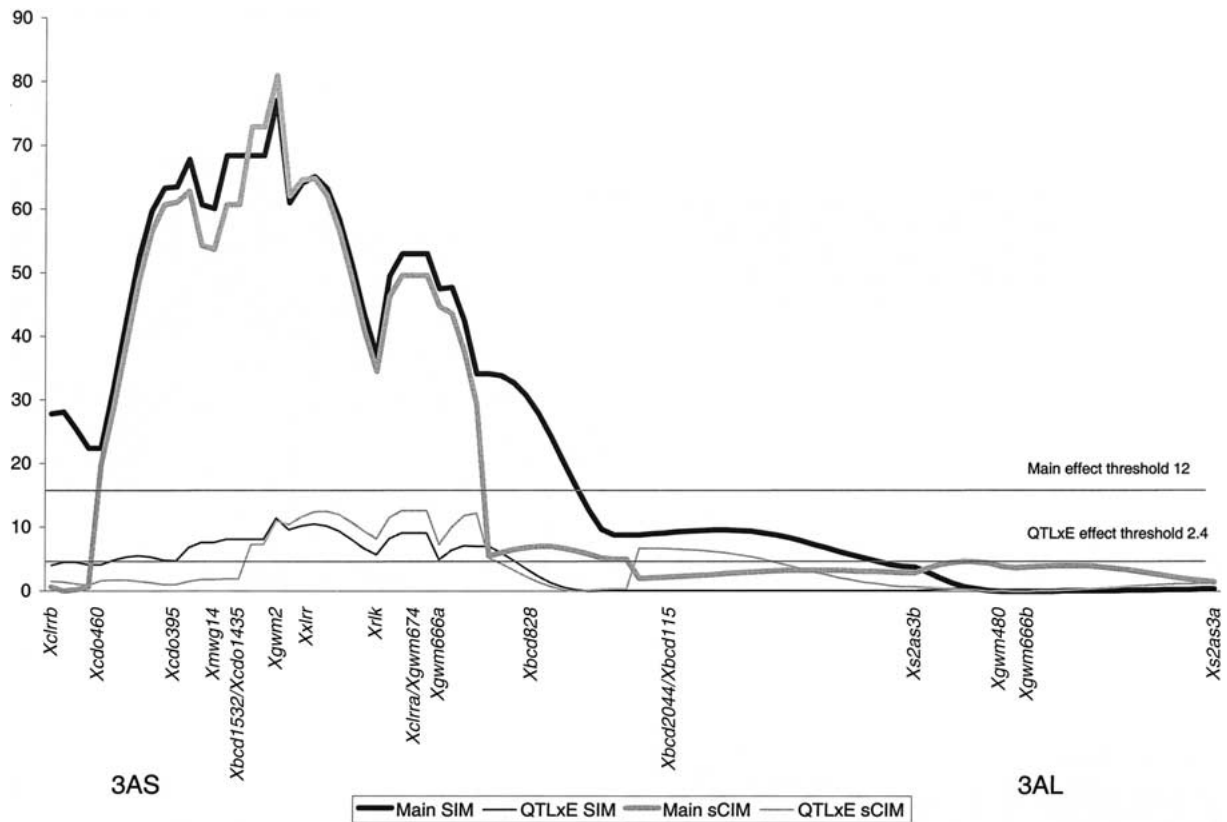


Figure 1. Test statistic plot for QTL main effect and QTL×E effect for FHB resistance generated using the linkage map of *T. dicoccoides* chromosome 3A. Plots determined for simple interval mapping (SIM, black lines) and simplified composite interval mapping (sCIM, gray lines). The experiment-wise type I error rate for declaring significance of SIM results is indicated.

seasons were observed. Disease severity of the 1998 spring greenhouse evaluation was higher than for the 1999 spring greenhouse evaluation. This discrepancy was most likely due to environmental conditions such as the onset of powdery mildew infection in the 1999 spring season. Due to the competitive infection some of the lines were not scored for FHB severity.

The severity data for each season were analyzed independently and also combined across seasons. The LDN(Dic-3A) parental line was significantly lower in disease severity than the LDN-16 parental line when evaluated in the 1998 spring season. In 1999, the disease severity of the LDN(Dic-3A) is not reported because the plants were lost before heading.

In both greenhouse seasons, a wide range of disease severity was observed (Table 1). Several RICL had disease severity scores higher than the susceptible parent, indicating transgressive segregation for susceptibility. In 1998, individuals with disease severity lower than the low parent were observed; however this was not significant. A test for homogeneity (Steel

Table 1. FHB severity means for type II disease reaction of parental lines, and LDN (Dic-3A) RICL population collected under controlled conditions in two greenhouse seasons.

Individuals	Spring 1998	Spring 1999
LDN-16	69.73	54.64
LDN(Dic-3A)	18.86	–
Population mean	53.08 ± 18.30	26.77 ± 13.19
Population range	93.32 ± 17.96	93.32 ± 7.00
C.V.	38.99	69.91
LSD (0.05)	32.42	31.36

*et al.*, 1997) was conducted on the two data sets to determine if it was possible to combine them. The  $\chi^2$  value calculated for the homogeneity of the two seasons was a non-significant value of 0.19. Thus, the habitability ( $h^2$ ) for the LDN(Dic-3A) population was calculated based on the combined analysis for the two

Table 2. FHB severity means for Type II disease reaction for the top five individuals in the LDN(Dic-3A) RICL population and parental lines averaged across spring 1998 and 1999 greenhouse evaluations.

Individuals	FHB severity means (%)
LDN-16	62.18
LDN (Dic-3A)	18.86
LDN (Dic-3A)-10	15.89
LDN (Dic-3A)-22	18.00
LDN (Dic-3A)-72	18.86
LDN (Dic-3A)-52	19.57
LDN (Dic-3A)-63	20.81
LSD (0.05)	31.89
C.V.	54.45

seasons. The  $h^2$  for this population for Type II FHB resistance was determined to be 66.9%.

#### Quantitative trait locus analysis

Both simple interval mapping (SIM) and simplified composite interval mapping (sCIM) were performed (Figure 1). The peak of the main effect QTL is located at or near *Xgwm2*. The *Xgwm2* locus has a test statistic of 77.1 for SIM analysis and 80.9 for sCIM analysis for the main effect QTL (significance threshold of 12; used only for SIM analysis). The total phenotypic variation ( $V_m/V_p$ , the main effect variation over the phenotypic effect variation) for the LDN(Dic-3A) RICL population explained by the *Xgwm2* locus was determined to be 37%. Based on the habitability estimate of 66.9%, the *Xgwm2* locus was determined to explain over 55% (37/66.9) of the genetic variation in this population. This major QTL for FHB type II resistance associated with *Xgwm2* is designated as *Qfhs.ndsu-3AS*.

#### Graphical genotyping

Graphical genotyping of the top five FHB resistance lines across 1998 and 1999 spring seasons of screening is presented in Figure 2. The means of the top five lines identified along with LDN-16 and LDN (Dic-3A) are represented in Table 2. LDN (Dic-3A)-72 possessed the smallest fragment from *T. dicoccoides* (29.3 cM), while LDN(Dic-3A)-63 possessed the largest fragment from *T. dicoccoides* (149.9 cM). All individuals in this selected group possessed the region from *T. dic-*

*occoides* spanning chromosome 3A from *Xmwig14* to *Xbcd828* loci, a genetic distance of ca. 30 cM.

## Discussion

#### *Fusarium head blight screening*

Screening for FHB resistance is labor-intensive, costly, and the phenotype is highly influenced by the environment (McMullen *et al.*, 1997). Greenhouse conditions must be carefully monitored to maintain proper environmental conditions. Confounding the data are common greenhouse diseases, which may skew screening results. Disease severity is dependent upon temperature and humidity from inoculation at anthesis to the soft dough stage (a Zadoks growth stage range of 60 to 85; Zadoks *et al.*, 1974). *Fusarium head blight* screening results are also subject to errors in classification. Even when using a pictorial scale, such as that in Stack and McMullen (1995), consistent and repeatable results require that the same person evaluates all plants in an experiment.

Environmental effects were evident among the two greenhouse seasons of screenings. Marker-assisted selection (MAS) to determine the presence of FHB resistance genes could potentially reduce the subjectivity associated with evaluation of heads for severity. Marker-assisted selection also allows for screening of a larger number of individuals in a shorter amount of time and with reduced expense.

#### Mapping

The LDN(Dic-3A) RICL chromosome 3A population map was 155 cM long. This is slightly longer than half the length of the published map for chromosome 3A (Nelson *et al.*, 1995). Reduced recombination in LDN (Dic) RICL population has been reported for chromosomes 5B and 6B (Chee *et al.*, 2001; González-Hernández, 2000). Possible explanations for this reduction in recombination are the number of meiotic events leading to the development of populations used in each study (one event for generation of RICLs while many events lead to the generation of RILs), or the relative distance of parents (i.e. chromosome homology) used in each cross.

#### Quantitative trait locus analysis

Graphs of the QTL maps for the main effect were generated by SIM and sCIM (Figure 1). While SIM

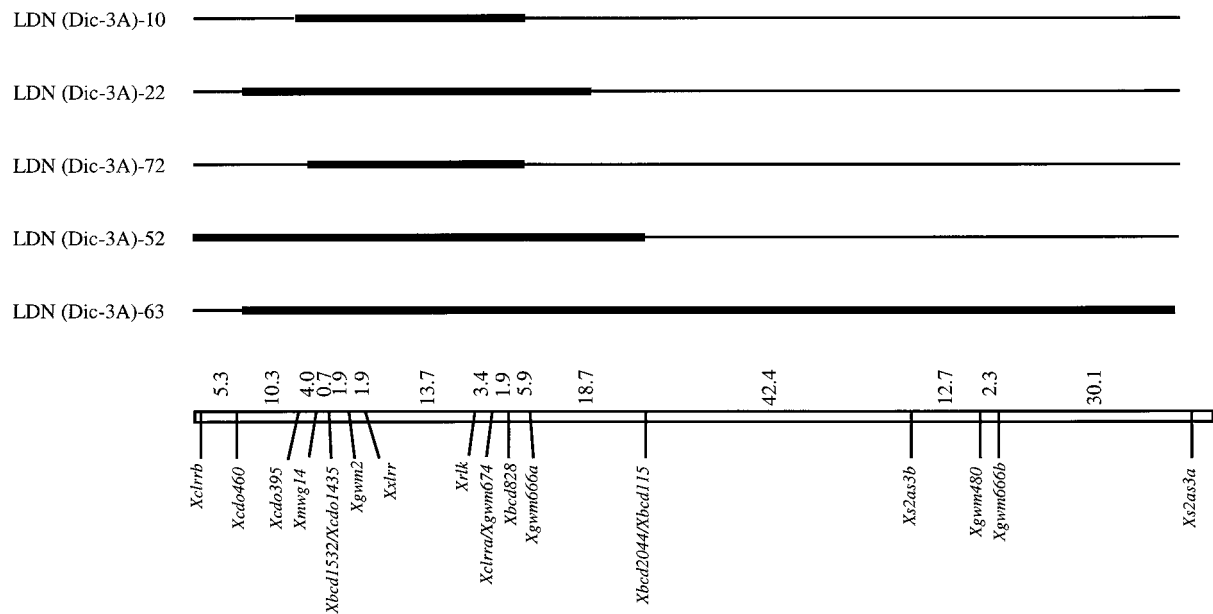


Figure 2. Graphical genotyping for the top five individuals in the RICL population showing resistance to FHB infection for regions of chromosome 3A based on mapped loci. In these individuals parental chromosomal segments (Dic-3A, thick line; and LDN-16, thin line) are depicted. A 29.3 cM region from *Xgwm14* to *Xbcd828* of *T. dicoccoides* chromosome is significantly associated with FHB resistance. All map distances are in cM calculated with the Haldane mapping function.

uses a linear model for analysis and gives results similar to the maximum-likelihood model, it does not determine the effect of other QTL in the vicinity of the QTL of interest (Tinker and Mather, 1995a, b). Analysis by sCIM accounts for the interaction of other QTL in the vicinity of the QTL of interest (Tinker and Mather, 1995a, b). Since there is no significant difference between the SIM and sCIM analyses that were performed, it can be assumed that no other QTL on this linkage group affected FHB resistance in this population. Thus, a single QTL for FHB resistance is present on chromosome 3A. We have proposed the designation *Qfhs.ndsu-3AS* for this QTL.

The *Qfhs.ndsu-3AS* region associated with FHB resistance in the LDN(Dic-3A) RICL population has a relatively large effect on the trait. The QTL explains 37% of the phenotypic variance and 41% of the variance when including the environmental interaction. The locus most closely associated with the peak of *Qfhs.ndsu-3AS* is *Xgwm2*. The presence of a significant peak for the QTL by environment interaction (QTL  $\times$  E) for FHB resistance in the LDN(Dic-3A) population indicates that the main effect QTL is influenced by the environment, as expected from cursory observation of the raw data. Heritability for this trait in this population was calculated to be 66.9%. Using this estimate it is possible to partition the variation in this

population to the genetic and environmental effects. The genetic variation explained by *Qfhs.ndsu-3AS* in this population can then be estimated to be 55.3%.

Several QTL associated with FHB have been identified in hexaploid wheat populations derived from Sumai-3 (Waldron *et al.*, 1999; Anderson *et al.*, 2001). The locations of these QTL were on chromosomes 3B, 2A, and 6B. One of the identified QTL, *Qfhs.ndsu-3BS*, explained 24.8–41.6% of the total variation depending on the population examined (Anderson *et al.*, 2001). It may be postulated that the 3A QTL reported here and the 3B identified in hexaploid wheat represent homoeologous loci. However, due to their map locations on the respective chromosomes this seems unlikely.

It is interesting to note that distal to the *Xgwm2* locus on the long arm of chromosome 3A, there are two RGA loci mapping within 15 cM of the peak of the QTL. The two RGA loci closest to the peak are both derived from leucine-rich repeat (LRR) motifs of known disease resistance genes from rice.

#### Graphical genotyping

The results of the genotyping show that there is a region from the *T. dicoccoides* parental line spanning the *Xmwg14* locus to the *Xbcd828* locus present in all

of the resistant RICL. This region covers a distance of 29.3 cM spanning both the short and long arms of chromosome 3A and contains *Qfhs.ndsu-3AS*. The top five most resistant lines in the RICL population all contain this region. Identifying the lines with highest FHB resistance and the smallest region of *T. dicoccoides* may aid in reducing the amount of linkage drag associated with the use of this wild species in breeding programs.

#### Value in breeding

The RICL population, specifically lines carrying the smallest segment of *T. dicoccoides* chromosome while carrying *Qfhs.ndsu-3AS*, and the SSR loci identified closely linked to this QTL show great promise in breeding resistant varieties of durum and bread wheat. This RICL population contains a new source of FHB resistance unique from that detected in other populations. Even though the source is a wild accession of tetraploid wheat, the RICLs have the region of interest introgressed into an adapted, identified durum background. A second advantage of the substitution line is that only a single chromosome from the wild species is transferred. This reduces the amount of linkage drag that is associated with the introduction of genes from non-adapted species into adapted cultivars. Marker-assisted selection not only reduces the time that is spent on screening populations carrying this source of FHB resistance, but it could also remove the variation associated with phenotypic screening.

Zhu *et al.* (1999) showed that height and head morphology in barley were associated with the QTL determined for FHB resistance in the double-haploid population they evaluated. Hilton *et al.* (1999) discussed the relationship between plant height and resistance and concluded that there is a correlation between plant height and resistance in wheat. Mesterházy (1995) also reported that susceptibility of plants to FHB varied in natural field inoculations based on the height of the lines. Because the screening process used here was under controlled greenhouse conditions, influences of any morphological differences were limited. The resistance identified in this report is independent of such morphological characteristics.

#### Conclusions

Wild species have been used for transferring genes to a cultivated species and derived germplasm are in

use in many plant-breeding programs. In wheat, one example is the introduction of a *T. dicoccoides* gene or gene cluster from chromosome 6B for high grain protein concentration into durum and hexaploid wheat (Khan *et al.*, 1989; Chee *et al.*, 2001). Outside of wheat, an example of employing wild species for introduction of valuable genes is that of fruit size in tomato (*Lycopersicon esculentum*; Frary *et al.*, 2000). The use of genes from wild relatives enables an increase in germplasm diversity and represents new sources for disease resistance. Each of the previously published papers describes FHB QTL identified in different germplasm sources than the *T. dicoccoides* used in this research. This research has identified a new source of FHB resistance, and *Qfhs.ndsu-3AS* that can be used for breeding in not only tetraploid wheat but also by introgression into hexaploid wheat.

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