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Identification of QTLs Associated with Fusarium Head Blight Resistance in Barley Accession CIho 4196

R. D. Horsley, D. Schmierer, C. Maier, D. Kudrna, C. A. Urrea, B. J. Steffenson, P. B. Schwarz, J. D. Franckowiak, M. J. Green, B. Zhang, and A. Kleinhofs

ABSTRACT

Fusarium head blight (FHB), incited by *Fusarium graminearum* Schwabe [teleomorph *Gibberella zea* (Schwein)], reduces quality of harvested barley (*Hordeum vulgare* L.) because of blighted kernels and the presence of deoxynivalenol (DON), a mycotoxin produced by the pathogen. CIho 4196, a two-rowed type, is one of the most resistant accessions known in barley; however, it possesses many undesirable agronomic traits. To better understand the genetics of reduced FHB severity and DON accumulation conferred by CIho 4196, a genetic map was generated using a population of recombinant inbred lines derived from a cross between Foster (a six-rowed malting cultivar) and CIho 4196. Quantitative trait loci (QTL) analyses were performed using data obtained from 10 field environments. The possible associations of resistance QTLs and various agronomic and morphological traits in barley also were investigated. The centromeric region of chromosome 2H flanked by the markers ABG461C and MWG882A (bins 6–10) likely ($P < 0.001$) contains two QTLs contributing to lower FHB severity and plant height, and one QTL each for DON accumulation, days to heading, and rachis node number. The QTL for low FHB severity in the bin 8 region explained from 3 to 9% of the variation, while the QTL in the bin 10 region explained from 17 to 60% of the variation. A QTL for DON accumulation that explained 9 to 14% of the variation was found in the bin 2 region of chromosome 4H. This may represent a new QTL not present in other FHB resistant sources. Resistance QTLs in the bin 8 region and bin 10 region of chromosome 2HL were provisionally designated *Qrgz-2H-8* and *Qrgz-2H-10,* respectively. The QTL for DON accumulation in chromosome 4H was provisionally named *QDON-4H-2*.

FUSARIUM HEAD BLIGHT adversely affected the quality of barley grown in North Dakota and northwestern Minnesota in 11 of the last 12 yr. The quality and value of harvested grain were reduced because of blighted kernels and the presence of DON, a mycotoxin produced by the pathogen. Widespread contamination of grain with DON made large portions of it unsuitable for use in malting and brewing (Schwarz, 2003). This resulted in significant revenue losses to growers because they were not able to realize malting barley premiums and had to sell their barley at discounted feed prices. The U.S. Government Accounting Office estimates that barley losses in North Dakota from 1993–1997 due to FHB were about $200 million (U.S. General Accounting Office, 1999). This amount is equal to about 17% of the $1.2 billion in total barley revenues that North Dakota growers received during this period. Nganje et al. (2001) estimated losses of $136 million in the same region from 1998–2000.

The relationship between visual FHB symptoms and DON concentration is not strong; thus, a grain sample with acceptable levels of plump kernels, grain protein, and kernel color can have high levels of DON (Schwarz et al., 1995). Deoxynivalenol concentrations ≤ 0.5 $\mu g\ g^{-1}$ are desired in barley by the malting and brewing industries because the mycotoxin can carry through the malting and brewing process into finished beer (Schwarz et al., 1995).

Tillage, crop rotation, and chemical control have been suggested as methods for reducing FHB severity, but success using these approaches has been limited. Genetic resistance offers the greatest potential for reducing FHB. Over 100 barley accessions have been reported to carry some FHB resistance (Steffenson, 2003; Scholz et al., 1999; Prom et al., 1996). Takeda (1992) identified CIho 4196 as one of the most resistant two-rowed barley accessions. Urrea et al. (2005) and Prom et al. (1996) confirmed his observations and found that CIho 4196 also accumulates low concentrations of DON. CIho 4196 originates from China and is being used extensively as a parent by the six-rowed barley breeding program at North Dakota State University (NDSU). CIho 4196, like most of the other described sources of resistance, heads and matures later, and is taller than cultivars currently grown in the Upper Midwest region of the United States (Urrea et al., 2005).

Urrea et al. (2002) evaluated $F_{4:5}$ and $F_{4:6}$ families from the cross between the FHB susceptible six-rowed cultivar Foster and CIho 4196 to gain knowledge about the inheritance of reduced FHB severity and DON concentration. Heritability of FHB severity and DON concentration was 0.65 and 0.46, respectively. They observed moderately strong positive genetic correlations between FHB severity and DON accumulation and found FHB severity and DON concentration to be negatively associated with plant height, days to heading, spike angle, and relative spike density. In other genetic studies, similar negative associations between FHB resistance and days to heading, plant height, and/or relative spike density were observed in segregating popula-

R.D. Horsely, P.B. Schwarz, J.D. Franckowiak, and M.J. Green, Dep. of Plant Sciences, North Dakota State Univ., Fargo, ND 58105-5051; D. Schmierer, C. Maier, and A. Kleinhofs, Dep. of Crop and Soil Sciences, Washington State Univ., Pullman, WA 99164-6420; D. Kudrna, Plant Sciences Dep., Univ. of Arizona, Tucson, AZ 85721-0036; C.A. Urea, Univ. of Nebraska, Scottsbluff, NB; B.J. Steffenson, Dep. of Plant Pathology, Univ. of Minnesota, 495 Borlaug Hall, 1991 Upper Buford Circle, St. Paul, MN, 55108; B. Zhang, Dep. of Plant Protection, Zhejiang Univ., Hangzhou City 310029, China. Received 21 Mar. 2005. *Corresponding author (richard.horsley@ndsu.edu).

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677 S. Segoe Rd., Madison, WI 53711 USA

Abbreviations: cM, centimorgans; DON, deoxynivalenol; FHB, Fusarium head blight; LRS, likelihood ratio statistic; QTL, quantitative trait locus; SIM, simple interval mapping; SSR, simple sequence repeat.

tions derived from crosses between adapted genotypes and the FHB-resistant cultivars Chevron (Ma et al., 2000; de la Pena et al., 1999), Gobernadora (Zhu et al., 1999), Fredrickson (Mesfin et al., 2003), and Zhedar 2 (Dahleen et al., 2003). The centromeric region of chromosome 2H was identified as a region of importance for these negative associations in each of these studies.

To determine if CIho 4196 carries the same genes for FHB resistance as the aforementioned cultivars, and to gain a better understanding of the genetics conferring reduced FHB severity and DON accumulation, and the association of these traits with other traits, a genetic map was generated using a population of recombinant inbred lines from the cross Foster × CIho 4196. This information is needed to develop an effective strategy for breeding agronomically acceptable cultivars with reduced FHB severity and DON concentration.

MATERIALS AND METHODS

Plant Materials

One spike was harvested from each of 250 F_2 plants from the cross Foster × CIho 4196 grown at Fargo, ND, during summer 1996. Two cycles of generation advancement were done in the greenhouse during the fall of 1996 and the spring of 1997. In each cycle, seed from one plant of each line was harvested and advanced to the next generation. Finally, three plants per line from the spring 1997 greenhouse increase were harvested in bulk to generate the $F_{4:5}$ generation. This generation was used as the seed source for the FHB and DON assays in Langdon, ND, in 1997. Seed harvested from these families ($F_{4:6}$) was the seed source for the trials grown in 1998 at Fargo, Langdon, and Osnabrock, ND. One $F_{4:6}$ seed from each family also was sown in the greenhouse. Leaf tissue for DNA extraction and seed harvested from each plant ($F_{6:7}$) were collected and sent to the laboratory of A. Kleinhofs at Washington State University. Remnant seed was used for additional disease and mycotoxin assays at Fargo, Langdon, and Osnabrock in 1999. During the initial stages of map construction, the level of heterozygosity in the population was greater than desired; thus, two generations of single seed descent were performed to obtain $F_{8:9}$ families. Seed from these families was used to sow field experiments at Zhejiang University in Hangzhou, China, in fall 2000 and at Langdon and Osnabrock in 2001.

Field Experiments

One hundred and fifty of the 250 $F_{4:5}$ recombinant inbred lines were randomly selected and sown at Langdon, ND, on 17 May 1997 in a FHB disease nursery. Experimental units were a single, 1-m row. Entries were assigned to experimental units using an augmented block design (Federer, 1993), and each experiment was repeated twice. The block size in each experiment was 25 entries, including the parents Foster and CIho 4196. The FHB disease nurseries were inoculated four times with *F. graminearum*, beginning 1 wk before heading, and then once a week thereafter for three consecutive weeks using the method of Prom et al. (1996). Because of the tall height of many entries, the population was prevented from lodging by supporting the plants with two strings of twine held up by metal posts located throughout the field. The experiment was repeated in FHB nurseries at Fargo, Langdon, and Osnabrock, in spring 1998; at the same locations in 1999; at Zhejiang University in Hangzhou, China, during the 2000–2001 winter; and at Langdon and Osnabrock in 2001. The same experimental design, plot size, and FHB inoculation methods employed in 1997 also were used for all subsequent experiments. Experiments were sown in 1998 on 27 April at Fargo and on 15 May at Langdon and Osnabrock; in 1999 on 21, 17, and 18 May at Fargo, Langdon and Osnabrock, respectively; from 5 to 7 November 2000 in Hangzhou, China; and on 14 May and 16 May 2001 at Langdon and Osnabrock, respectively.

Inoculum Preparation

In the North Dakota FHB nurseries, *F. graminearum* inoculum was prepared according to the methods of Xia (1956) as modified by Prom et al. (1996). Equal parts, by volume, of barley and maize (*Zea mays* L.) grain were soaked separately in water for 48 h. After soaking, the grain was placed in stainless steel pans and covered with aluminum foil. Next, the barley and maize grain were autoclaved for 20 min at 121°C, in each of two consecutive days, to sterilize the grain substrate. Pieces of agar containing five isolates of *F. graminearum* (KB-172, KB-173, KB-176, KB-582, and KB-672) (Salas et al., 1999) were added to each pan containing the sterilized grain, and the grain was incubated at 25°C for 14 d in complete darkness. Ten days following this treatment, the inoculum in each pan was mixed by hand.

Before inoculation, the colonized seeds of barley and maize were mixed in equal proportions and scattered over the barley plots at a rate of 50 g colonized grain m^{-2} for four consecutive weeks, beginning 1 wk before heading of the earliest entries. At Osnabrock, plots were irrigated at a rate of 1.20 L h^{-1} using model XS-360 F xeri-spray sprinklers (Rain Bird, Glendora, CA). Sprinkler heads were spaced 4 m apart and 120 cm above the ground. To provide favorable conditions for ascospore liberation and infection, irrigation was for 30 s every 30 min in the morning (0600 to 0800 h) and in the afternoon (1600 to 1800 h). At Fargo and Langdon, plots were irrigated at a rate of 0.19 L h^{-1} using 1800-SAM-PRS sprinklers (Rain Bird, Glendora, CA). Sprinkler heads were spaced 3 m apart and 120 cm above the ground. Irrigation was done once for 10 min at 0700 and 1700 h.

In the nursery at Zhejiang University, the inoculum preparation methods and sprinkler irrigation methods were modified slightly. Only autoclaved barley was used as a grain substrate for the inoculum production. A single isolate of *F. graminearum* from Zhejiang Province, China, was used for inoculum. Sprinkler irrigation was done in the morning at 0700 to 0800 h and in the afternoon at 1800 to 2000 h using locally purchased irrigation equipment.

Morphological, Agronomic, and Fusarium Head Blight Data

Data collected on progeny lines and parents in the field were days to heading (number of days after 31 May when 50% of spikes were fully emerged from the boot), plant height (distance from soil surface to the tip of spikes, excluding awns), spike angle (1 = erect spike, 90° from horizontal; 3 = bent spike, 0° from horizontal), spike density (spike length/no. of rachis nodes with fertile spikelets), and number of rachis nodes with fertile spikelets. Spike angle data were collected at harvest maturity.

Disease assessments were made at the soft dough stage (Zadoks 85) (Zadoks et al., 1974) of development. Ten to fifteen spikes within each row were harvested at random, and the infected kernels per spike were counted. Percentage FHB severity was calculated by dividing the total number of infected kernels by the total number of kernels and multiplying by 100.

At maturity, each plot was harvested using hand shears.

Grain samples were dried at 35°C in a forced air dryer to approximately 100 g kg^{-1} moisture, deawned, and cleaned. Deoxynivalenol accumulation ($\mu g\ g^{-1}$) of harvested grain was determined using the gas chromatograph method of Tacke and Casper (1996).

Statistical Analyses

Data for parents and progeny in each experiment in an environment were analyzed as an augmented block design. Adjusted means (i.e., least square means) were calculated for each entry and these means were used to perform the analysis of variance across experiments as a randomized complete block design. Within a year, each experiment at an environment was considered a replicate. Mean separation between parents was done using *t* tests and were considered significant at $P \leq 0.05$.

Map Creation

Leaf tissue was collected from 144 of the 150 $F_{8:9}$ recombinant inbred lines (RILs) (six plants did not grow) grown in the greenhouse in 2000. Plant DNA extraction, Southern blotting, and RFLP hybridization procedures were done using standard protocols (Kleinhofs et al., 1993), except that 1.0% agarose gels were used to separate digested barley DNA. DNA probes were screened against the parents (Foster and CIho 4196) for polymorphisms using six restriction enzymes (*BamH*I, *Dra*I, *Eco*RI, *Eco*RV, *Hin*dIII, and, *Xba*I).

Parental and progeny DNA also were screened using PCR with published simple sequence repeat (SSR) sequences purchased from Integrated DNA Technologies (Coralville, IA) and from the Scottish Crops Research Institute (Ramsay et al., 2000). The SSR loci were amplified in a 20-μL reaction mixture containing 50 ng of template DNA, 0.3 μM of each primer, 200 μM of each dNTP, 2 m*M* of $MgCl_2$, 1 unit of Taq DNA polymerase, and 1 × Taq buffer supplied with the enzyme using the conditions described by Ramsey et al. (2000). PCR products were separated on 4 to 6% denaturing polyacrylamide gels (Cambrex, East Rutherford, NJ). For product visualization, gels were silver stained using the DNA Silver Staining System (Promega Corporation, Madison, WI).

Map Manager QTXb20 (Manly et al., 2001) was used to create the skeletal genetic map. Recombination values were converted to centimorgans (cM) using the Kosambi function (Kosambi 1944). When possible, map positions of previously mapped markers were confirmed using the Steptoe × Morex 150 doubled haploid line population (Kleinhofs et al., 2005). To facilitate map saturation in the target region of chromosome 2HL, 56 RILs with recombinations in the bin 8 to bin 12 region were selected and used for subsequent mapping.

Quantitative Trait Loci Mapping

The software program Map Manager QTX version 0.27 (Manly et al., 2001) was used for identification of QTLs for all traits at the 10 environments. For each analysis, regression analysis was used to identify chromosomal regions associated with FHB resistance at $P < 0.001$. Permutation tests (500 iterations) were performed at $P < 0.001$ to determine the thresholds at which the likelihood ratio statistic (LRS) became significant for QTL identification. Next, simple interval mapping (SIM) was done for the chromosomes associated with FHB resistance for all traits in 1-cM steps using the critical LRS values identified by the permutation tests. Those regions with LRS values above the significant level were determined to contain putative QTLs. The percentage of variation explained by the QTLs and the additive regression coefficient at the peak LRS value for the trait were obtained from the SIM analysis. When it appeared multiple putative QTLs for FHB were in the same region, composite interval mapping (CIM) was done using the marker with the greatest coefficient of variation in the regression analysis as the background marker.

RESULTS AND DISCUSSION

Data collected in 1997 and 1998 were used to determine the heritability of FHB resistance and DON accumulation, and the correlated response of other traits when selecting for reduced FHB (Urrea et al., 2002). The population was grown again in 1999 at three North Dakota locations; at Zhejiang University in Hangzhou, China, during the 2000–2001 winter; and in 2001 at two North Dakota locations. In the barley-FHB pathosystem, FHB severity and DON concentration often exhibit a high degree of variation; thus, it is important to collect phenotype data across several environments. The Foster × CIho 4196 population was phenotyped in 10 environments to more rigorously identify and define putative QTLs. As such, Foster × CIho 4196 is the most intensively characterized population for FHB resistance to date.

Disease pressure varied from low (2000–2001 Hangzhou) to very high (2001 Langdon and Osnabrock) across the 10 FHB nurseries. However, the susceptible controls in each augmented block had infection levels greater than CIho 4196, indicating that the chance for disease escapes was low.

Responses of the Parents and Progeny

Least square mean FHB severity, DON accumulation, days to heading, plant height, spike angle, relative spike density, and number of fertile rachis nodes of the progeny and two parents and the range of the progeny from each environment where data were collected are presented in Table 1. Significant differences in FHB severity were found between the parents at all 10 environments. On average, mean FHB severity of CIho 4196 and Foster was 14.2 and 43.9%, respectively. Mean FHB severity of the progeny (31.8%) was closer to that of the susceptible parent Foster. Negative and positive transgressive segregation was observed in all environments, indicating that both parents contribute loci for FHB resistance.

Data on DON accumulation were collected for entries grown at four environments. DON accumulation was measured from only four environments because of the large number of samples and the high cost of the assay. Significant differences between the parents were found at two of the four environments. At Osnabrock in 2001, the DON accumulation was 61.5 $\mu g\ g^{-1}$ for CIho 4196 and 64.1 $\mu g\ g^{-1}$ for Foster. The similar DON concentrations found for the two parents at two of the environment indicates that the resistance to DON accumulation of CIho 4196 can be overwhelmed if disease pressure is high. The range in DON accumulation (36.3–193.1 $\mu g\ g^{-1}$) in the progeny at 2001 Osnabrock was large. This indicates that, even though the disease pressure was high, segregation for DON accumulation in the population could still be detected. Negative and positive transgressive

Table 1. Least square mean Fusarium head blight (FHB) severity, deoxynivalenol (DON), days to heading, plant height, spike density, spike angle and number of fertile rachis nodes of parents and progeny from the cross Foster/CIho 4196, and range of the progeny.

		FHB severity		DON		Days to heading		Plant height		Spike density		Spike angle score (1–3)†		No. of rachis nodes with fertile spikelets	
Environment		Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
		%		g μg^{-1}		d		cm		mm rachis node^{-1}					
1997 Langdon	CIho 4196	1.0b‡		7.3b				113.3a						14.7a	
	Foster	21.9a		43.2a				90.8b						9.7b	
	Progeny	9.2	−0.9–35.7§	25.9	−17.4–123.3			106.0	76.5–134.2					11.9	5.8–20.5
1998 Fargo	CIho 4196	2.0b				37.2a		93.2a						14.7a	
	Foster	44.9a				29.2b		80.2b						9.9b	
	Progeny	23.1	−3.7–60.1			35.1	28.9–40.9	87.1	47.2–117.4					12.3	4.7–25.5
1998 Langdon	CIho 4196	6.0b				37.2a		112.3a						16.8a	
	Foster	43.8a				32.0b		94.5b						10.2b	
	Progeny	30.6	0.1–93.7			36.1	29.8–42.6	108.9	82.4–132.9					12.3	4.8–21.8
1998 Osnabrock	CIho 4196	21.1b		9.1b		34.9a		116.6a				2.7a			
	Foster	48.7a		32.8a		31.4b		96.7b				1.2b			
	Progeny	37.6	−4.8–87.6	22.5	−7.5–84.2	34.3	28.0–43.0	110.6	88.6–127.9			2.0	0.4–3.4		
1999 Fargo	CIho 4196	2.1b								2.4b		1.8a		16.5a	
	Foster	20.8a								3.6a		1.0b		9.4b	
	Progeny	8.9	−6.7–39.7							3.3	1.7–5.1	1.5	0.4–3.3	11.5	6.3–22.5
1999 Langdon	CIho 4196	21.5b						119.3a		2.5b		3.0a			
	Foster	49.0a						98.4b		3.6a		1.0b			
	Progeny	44.9	17.6–75.1					115.9	92.0–136.7	3.0	1.7–4.2	2.2	1.0–3.0		
1999 Osnabrock	CIho 4196	5.2b								2.6b		3.4a			
	Foster	55.2a								3.5a		1.0b			
	Progeny	30.8	−1.6–70.3							3.1	1.7–5.1	1.9	0.9–3.4		
2000–2001 China	CIho 4196	5.2b						121.0a				2.9a			
	Foster	11.7a						113.2b				2.0b			
	Progeny	13.9	−0.8–30.8					117.6	93.1–149.6			2.1	0.9–3.1		
2001 Langdon	CIho 4196	20.7b		62.5a										12.8a	
	Foster	64.3a		57.5a										10.7b	
	Progeny	48.4	3.6–97.4	94.9	32.2–231.5									12.4	7.2–20.0
2001 Osnabrock	CIho 4196	57.2a		61.5a								2.4a		12.9a	
	Foster	79.8b		64.1a								1.4b		9.1b	
	Progeny	71.9	33.2–99.4	95.6	36.3–193.1							1.8	0.9–2.7	12.0	7.5–26.4

† Spike angle score where 1 = erect spike, 90° from horizontal and 3 = bent spike, 0° from horizontal.
‡ Means within a column for an environment followed by the same letter are not significantly different at $P = 0.05$ as determined by a t test.
§ Least square means with a negative number are not incorrect, but occur due to the analysis.

segregation was observed in all four environments, indicating that both parents are contributing loci for DON accumulation.

The number of days to heading was determined at three environments. Averaged across the three environments, CIho 4196 headed about 4.4 d later than Foster, and the mean of the progeny was skewed toward that of CIho 4196. The range in days to heading of the progeny was much greater than the difference observed between the parents (average nearly 13 d). This indicates that both parents were contributed different genes that affected days to heading.

Plant height was measured at six environments, and CIho 4196 was significantly taller than Foster at all environments. Averaged across all environments, CIho 4196 was nearly 20 cm taller than Foster. Height of the progeny was skewed toward that of CIho 4196, and positive and negative transgressive segregation was observed at all environments. Again, it appears that both parents are contributing genes controlling plant height.

The population also segregated for spike density; thus, data for this trait were collected at three environments in 1999. Since this character can alter the microclimate of the spike, thereby affecting disease severity, we wanted to determine if compact spikes were more susceptible to FHB in our population. Zhu et al. (1999) mapped coincident QTLs for FHB severity and spike density using a population derived from the cross Gobernadora × CMB643 (note: CMB643 is now known as 'Azafran'). At all environments, spike density of CIho 4196 was more compact than Foster. On average, the distance between rachis nodes with fertile spikelets was 2.5 and 3.6 mm for CIho 4196 and Foster, respectively. The range in spike density of the progeny was from 2.1 to 4.6 mm. Again, the transgressive segregation observed in the progeny indicates that both parents are contributing genes that affect this trait.

Spike angle was determined at six of the 10 environments. Data for this trait were collected to determine if an upright spike would be more susceptible to FHB than a nodding spike, the former could provide a microclimate conducive for disease infection. An upright spike would be expected to stay wet longer after precipitation or deposition because the water would collect in the spike. In contrast, water would be shed more efficiently from a nodding spike. Foster had a more upright spike than CIho 4196 at all environments. Mean spike angle of the progeny was intermediate to that of the parents, and the range of progeny generally fell within the values for the parents. This suggests that the inheritance of this trait may not be as complex as that of the other traits measured.

The number of rachis nodes with fertile spikelets was determined at six of the 10 environments. The spike of CIho 4196 had significantly more rachis nodes than Foster (14.0 vs. 9.7), and the mean number of rachis nodes of the progeny was generally intermediate to the two parents. In environments where tip sterility did not occur (i.e., 2001 Langdon and Osnabrock), the range in rachis nodes was 7.3 to 22.0 nodes. As with the other traits, transgressive segregation was observed.

Quantitative Trait Loci Analysis

A linkage map of all seven barley chromosomes is presented in Fig. 1. A total of 206 molecular markers were mapped, generating a linkage map of 142 unique loci consisting of 115 RFLP (genomic DNA, cDNA, resistance gene analogs, and known function genes) and 26 SSR marker loci. One morphological marker for two-rowed vs. six-rowed spike type (*vrs1*) was also mapped. The genetic map spanned a distance of 1137 cM and compares favorably with the Steptoe × Morex map of 1250 cM (Kleinhofs et al., 1993) and the most recently published Steptoe × Morex DArT map of 1137 cM (Wenzl et al., 2004). Thus, this map is adequate for QTL analyses across most regions of the chromosomes. Once the initial map was developed and preliminary data suggested that chromosome 2H contained the main FHB resistance QTLs, it was targeted for more extensive map development. As a result, the chromosome 2H map is well developed with 62 markers, including 35 new ones. Twenty six of the new markers mapped within the critical region containing the FHB QTL. In addition, lines with recombination in the chromosome 2H, ABC306 to MWG882A, bin 7 to bin 10 region were selected and used for saturation genetic and eventual physical mapping. This map contains 26 unique loci and a total of 73 markers, including those that cosegregate (Fig. 2).

Because the purpose of this research is to identify QTLs associated with FHB resistance and DON accumulation, only chromosome regions with these QTLs are discussed. Also, because all data were not collected at all environments, separate QTL analyses were performed first for each environment. This allowed us to determine if QTLs for FHB resistance and DON accumulation were detected in most or all environments. In order for a QTL to be a candidate for marker assisted selection and map-based cloning, it should be expressed in most environments where the progeny segregated for the trait and data were collected. Thus, QTLs detected in <75% of the environments where a trait was measured will not be discussed. The QTL analyses also were done using means averaged across environments to determine if the results were consistent with those observed from the individual environment analyses. For all traits, the results based on individual environments and means were consistent. Plots of the test statistics based on the QTL analyses of means are presented in Fig. 3. In all analyses, a chromosome region was considered likely ($P < 0.001$) to contain a QTL when the LRS value calculated using Map Manager QTX was greater than the threshold value obtained using the permutation tests function. To facilitate comparisons of our results with those from other studies, we aligned our map with the Steptoe × Morex barley bin map (Kleinhofs et al., 2005).

The region of the long arm of chromosome 2H (i.e., 2HL) near the centromere was found to have QTLs associated with both FHB severity and DON concentration at $P < 0.001$ (Fig. 3). These QTLs were detected in all environments where FHB severity and DON were determined. This region of chromosome 2HL also had QTLs for days to heading, plant height, and number of

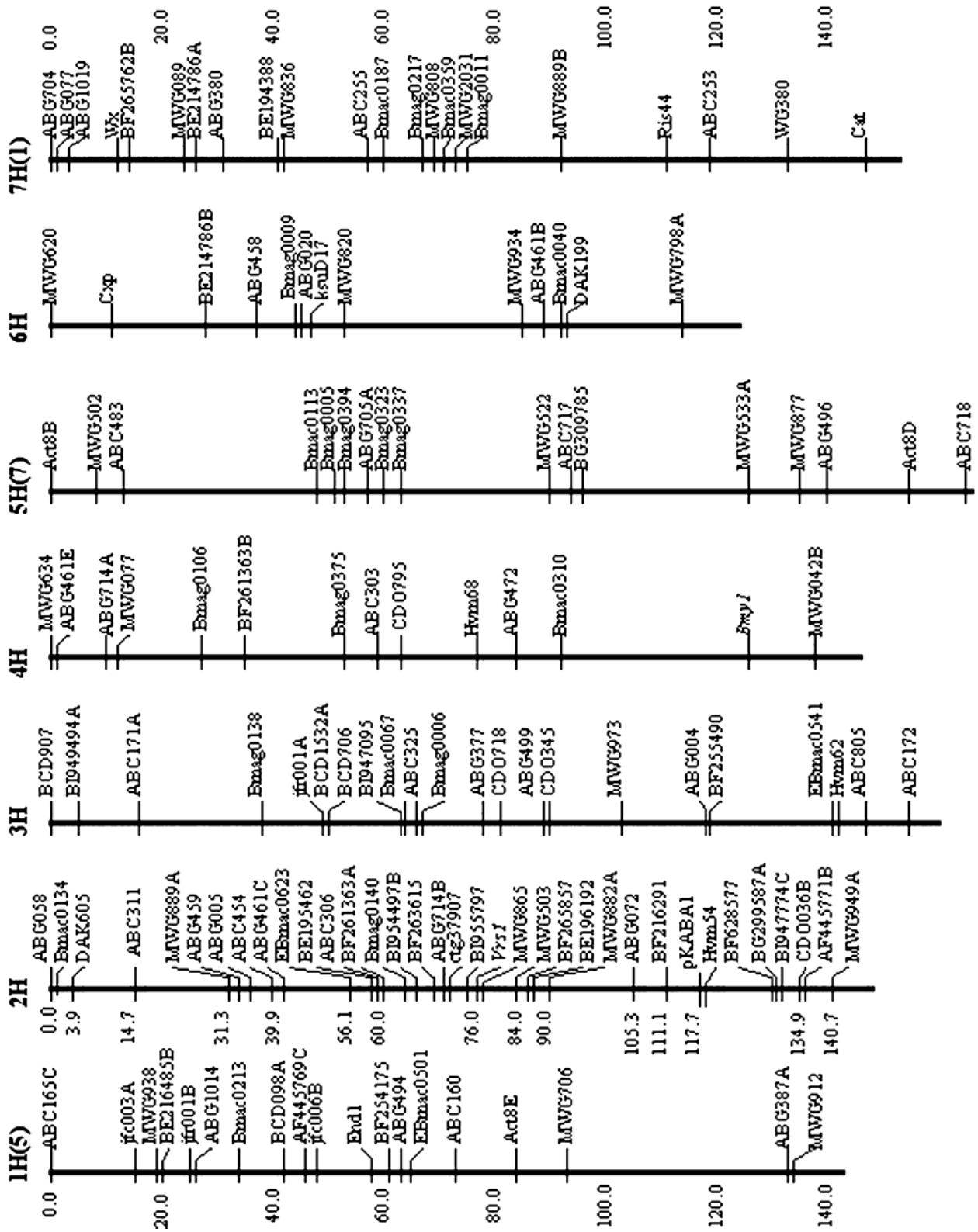

Fig. 1. Foster × CI4196 map. Map was generated based on 144 recombinant inbred lines. Only a single marker is shown at each locus, although in some cases multiple markers segregated at the same locus. For an expanded map of the *Vrs1* region showing all markers, see Fig. 2. Expressed sequence tag markers are identified by their accession number; jfr or jfc markers are wheat RGAs from Dr. John Fellers (Kansas State University) and refer to locus designations used in his laboratory; ctg markers refer to contig numbers in HarvEST (http://harvest.ucr.edu, cited 21 June 2005, verified 9 Aug. 2005).

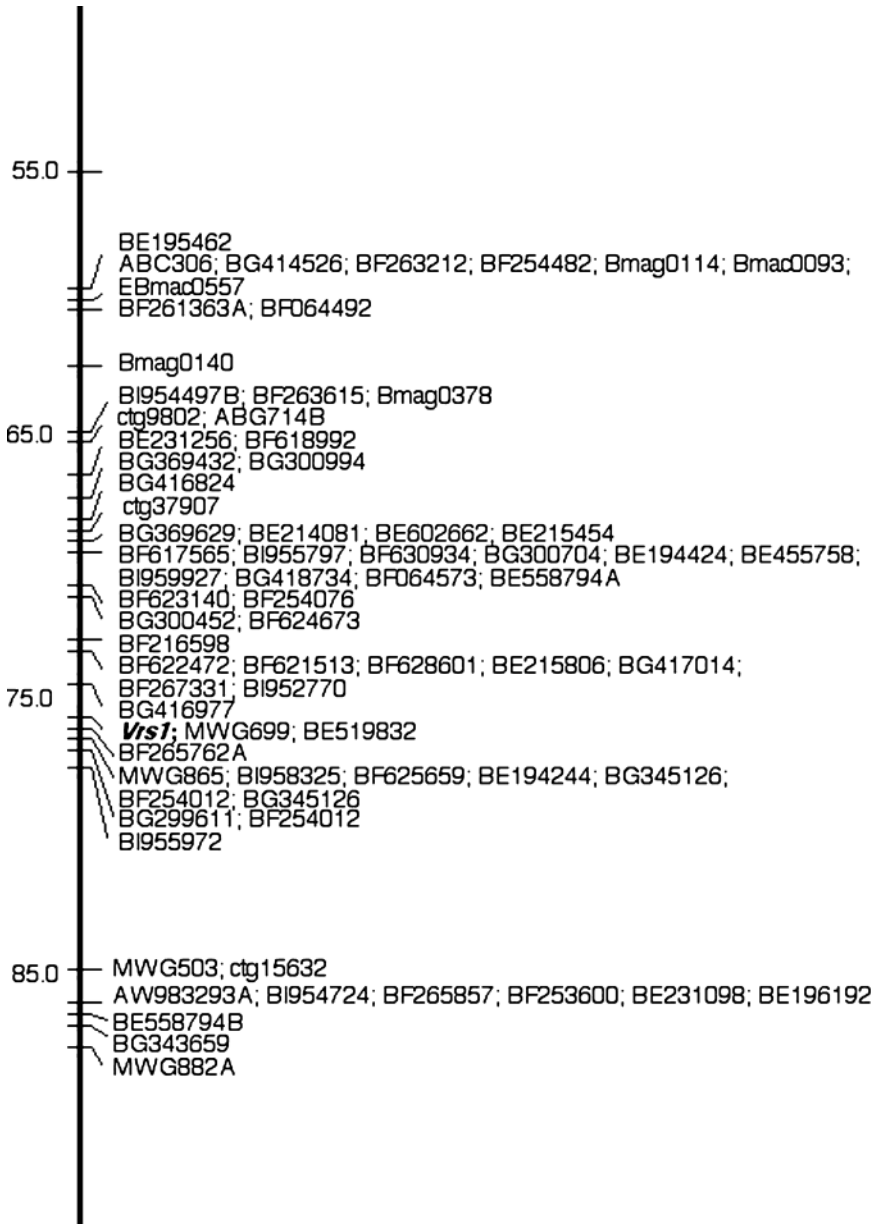

Fig. 2. Foster × CI4196 chromosome 2H ABC306–MWG882 region expanded map. Map was generated using 56 lines containing a recombination in the chromosome 2H region selected from the Foster × CI4196 recombinant inbred population. This map contains some markers not found in Fig. 1. The map distances were adjusted to correspond to the map distances based on the population used to generate the whole genome map in Fig. 1 and are approximately to scale.

rachis nodes in most environments where they were measured. This same region of chromosome 2HL was found to have QTLs associated with FHB severity and DON accumulation in all mapping papers for this trait published to date (Dahleen et al., 2003; Mesfin et al., 2003; Ma et al., 2000; de la Pena et al., 1999; Zhu et al., 1999). These papers also reported on other chromosome regions with QTLs controlling these traits. In a study validating MAS of QTLs for FHB resistance from Chevron-derived lines, Canci et al. (2004) confirmed the presence of QTLs for FHB resistance and days to heading in bin 8 of chromosome 2HL.

We identified QTLs for FHB severity in the region of chromosome 2H flanked by the markers ABG459 and ABG072 (bins 5–11) (Table 2). This region explained 11 to 60% of the variation in FHB severity, and CIho 4196 contributed the resistance allele. In eight of the 10 environments, the peak LRS value was located in the bin 10 region, and in the other two environments the peak LRS value was in bin 8. In the SIM analysis of 1999 Osnabrock and 2001 China, FHB QTLs were detected in bins 8 and 10. In these two environments, the bin 8 QTL explained 11 to 40% of the variation in FHB severity, and the peak LRS values were located in the region flanked by BE195462 and BF263615. The analysis based on means across all 10 environments detected a QTL in the region flanked by ABG459-ABG072 (bins 5–11) and the peak LRS value was closest to the *vrs1* locus. The QTL explained 71% of the variation.

Review of the QTL scans from the individual environ-

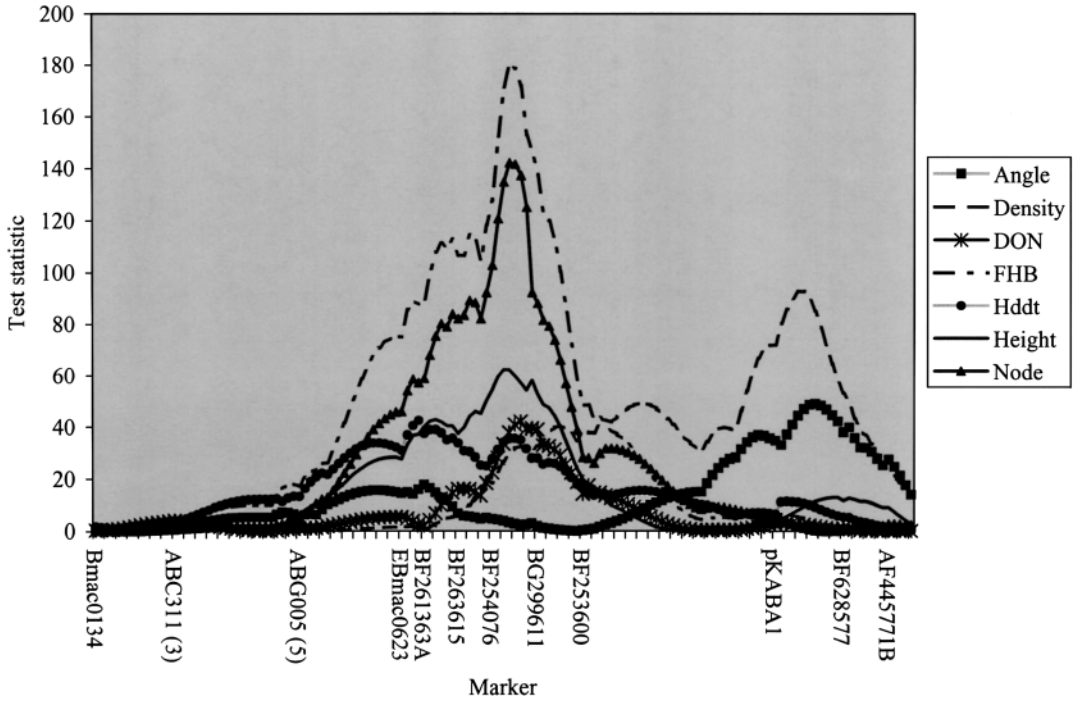

Fig. 3. Plot of the test statistics from the simple interval mapping analyses of the Foster/CIho 4196 mapping population for chromosome 2H for mean spike angle (Angle), relative spike density (Density), deoxynivalenol concentration (DON), Fusarium head blight severity (FHB), days to heading (Hddt), plant height (Height), and number of rachis nodes with fertile spikelets (Node) averaged across environments. The bin location appears in parentheses behind selected markers.

ment SIM analyses (not presented) and the analysis of means (Fig. 3) indicated there may be more than one QTL for FHB resistance in the region flanked by ABG459 and ABG072 controlling FHB severity. Composite interval mapping identified a second QTL of lesser effect in chromosome 2HL flanked by ABG461C and BF263615 (bins 6–8) in four of the 10 environments (Table 2). A QTL in this same region was detected in the SIM analysis at four environments as discussed earlier. Thus, the QTL in bin 8 was detected in 8 of the 10 environments. This strongly suggests the presence of two QTLs for FHB severity in chromosome 2HL. A QTL with a lesser effect is located in bin 8 and one with a much stronger effect is located in bin 10.

A QTL for reducing DON concentration was detected in the chromosome 2H region flanked by ABG005 and MWG882A (bins 5–10) in three of the four environments where DON was measured (Table 2). This region explained from 10 to 30% of the variation for DON accumulation in the progeny, with CIho 4196 contributing the resistance allele. The analysis based on means detected a QTL in the same region that explained 25% of the variation, with the peak LRS value located near the *vrs1* locus. Other studies found that the resistance allele at the QTL for reduced DON was contributed by Zhedar 2 (Dahleen et al., 2003), Fredrickson (Mesfin et al., 2003), Chevron (Ma et al., 2000; de la Pena, 1999), and Azafran (Zhu et al., 1999) in the same region of chromosome 2HL.

A QTL for days to heading was detected in the region of chromosome 2H flanked by ABG461C and MWG865 (bins 6–10) in all three environments where data were recorded (Table 2). This region explained 12 to 30% of the variation in days to heading with CIho 4196 contributing the allele for later heading. The peak LRS values were in the bin 8 region in all three analyses (Table 2). In the combined QTL analysis across environments, the QTL for days to heading was flanked by ABG461C and MWG503 (bins 6–11). This QTL explained 26% of the variation in days to heading and the peak LRS value was located nearest BF261363A in bin 8. The QTL detected may actually be the photoperiod responsive gene *Eam6* (early maturity 6) that maps to this region (Franckowiak and Konishi, 2002; Toho-oka et al., 2000) and is common in Midwestern six-rowed barley. Dahleen et al. (2003) came to the same conclusion using their mapping population for FHB resistance derived from the cross ND9712 × (Foster × Zhedar 2).

A QTL for plant height was detected in the region of chromosome 2H flanked by ABG461C and MWG503 (bins 6–11) in all seven environments where plant height was measured (Table 2). This QTL explained 11 to 31% of the variation in plant height of the population, with CIho 4196 contributing the allele for increased height. In the QTL analysis of mean plant height across environments, the QTL was again flanked by ABG461C and MWG503, and it explained up to 35% of the variation in the progeny. Review of the QTL scans from the individual analyses (not presented) and the analysis of means (Fig. 3) show two peaks near the markers BF254076 and BG299611 in the bin 10 region that flank the *vrs1* locus (Fig. 2); thus, there may actually be two linked QTL for plant height in this narrow region. The QTL above the *vrs1* locus may actually be the *hcm1* (short culm) gene that maps to this region and controls plant height (Franckowiak, 1997). Dahleen et al. (2003) also detected a QTL

Table 2. Marker region, closest marker to the peak and bin location in chromosome 2H, percentage of variation explained by the quantitative trait loci (QTLs), additive regression coefficient (Add), and parent contributing the allele for QTLs controlling Fusarium head blight severity (FHB), deoxynivalenol accumulation (DON), days to heading, plant height, spike density, spike angle, days to maturity, and number of rachis nodes with fertile spikelets at 10 environments, 1998 to 2001, and averaged across environments.

Trait	Environment	Region	Closest marker to peak	Bin†	Variation explained	Add	Parent contributing allele
					%		
FHB (SIM‡)	1997 Langdon	ABG461C-MWG882A	*vrs1*	10	52	−7.1	CIho 4196
	1998 Fargo	ABC454-MWG882A	BF254076	10	56	−11.8	CIho 4196
	1998 Langdon	ABG461C-MWG882A	*vrs1*	10	60	−15.4	CIho 4196
	1998 Osnabrock	ABG461C-ABG072	BF254076	10	33	−12.0	CIho 4196
	1999 Fargo	ABG461C-*vrs1*	Bmag0140	8	18	−3.7	CIho 4196
	1999 Langdon	ABG461C-MWG503	*vrs1*	10	39	−8.8	CIho 4196
	1999 Osnabrock	ABG459-BF263615	BE195462	8	40	−10.9	CIho 4196
		ABG714B-ABG072	BF254076	10	38	−11.2	CIho 4196
	2001 China	Bmag0140-ABG714B	BF263615	8	11	−1.9	CIho 4196
		ABG714B-BF254012	*vrs1*	10	17	−2.1	CIho 4196
	2001 Langdon	ABG461C-MWG882A	*vrs1*	10	58	−19.2	CIho 4196
	2001 Osnabrock	ABG461C-BF263615	Bmag0140	8	27	−7.0	CIho 4196
		BF263615-MWG503	BF254076	10	33	−7.8	CIho 4196
	Mean	ABG459-ABG072	*vrs1*	10	71	−10.3	CIho 4196
FHB (CIM§)	1997 Langdon	ABG461C-BE195462	EBmac0623	8	3	−1.8	CIho 4196
	1998 Fargo	ABG461C-BF263212	BF263212	8	3	−3.5	CIho 4196
	1998 Langdon	ABG461C-BF263615	BE195462	8	6	−5.1	CIho 4196
	1998 Osnabrock	–	–	–	–	–	
	1999 Fargo	BF254076-*vrs1*	*vrs1*	10	4	−2.0	CIho 4196
	1999 Langdon	ABG461C-Bmag0140	BF263212	8	9	−4.6	CIho 4196
	1999 Osnabrock	–	–	–	–	–	
	2001 China	–	–	–	–	–	
	2001 Langdon	–	–	–	–	–	
	2001 Osnabrock	–	–	–	–	–	
	Mean	ABG461C-ctg37907	BE195462	8	8	−3.8	CIho 4196
DON	1997 Langdon	BF261363A-MWG503	*vrs1*	10	30	−13.7	CIho 4196
	1998 Osnabrock	ABG005-MWG882A	*vrs1*	10	28	−10.5	CIho 4196
	2001 Langdon	BF254076-BG299611	*vrs1*	10	10	−11.8	CIho 4196
	2001 Osnabrock	BE195462-Bmag0140	BF261363A	8	11	10.1	Foster
	Mean	BF254076-MWG503	*vrs1*	10	25	−11.4	CIho 4196
Days to heading	1998 Fargo	ABG461C-BF263212	BF263212	8	18	1.3	CIho 4196
		BF261363A-ABG072	BF254076	10	20	1.5	CIho 4196
	1998 Langdon	ABG459-BG345126	BF261363A	8	30	1.5	CIho 4196
	1998 Osnabrock	ABG461C-Bmag0140	BF261363A	8	12	1.1	CIho 4196
	Mean	ABG461C-MWG503	BF261363A	8	26	1.2	CIho 4196
Plant height	1997 Langdon	BE195462-BF265762A	*vrs1*	10	28	6.8	CIho 4196
		BF265762A-MWG503	BG299611	10	28	6.4	CIho 4196
	1998 Fargo	BF254076-*vrs1*	BF254076	10	17	4.9	CIho 4196
		BF265762A-BG299611	BG345126	10	14	4.2	CIho 4196
	1998 Langdon	ABG714B-*vrs1*	*vrs1*	10	18	4.7	CIho 4196
		BF265762A-MWG503	BG299611	10	20	4.6	CIho 4196
	1998 Osnabrock	ABG714B-*vrs1*	BF254076	10	31	4.9	CIho 4196
		BF265762A-BF253600	BG345126	10	29	4.5	CIho 4196
	1999 Langdon	ABG461C-ctg37907	Bmag0140	10	26	6.5	CIho 4196
		BE558794A-MWG503	BF254076	10	23	6.1	CIho 4196
	1999 Osnabrock	EBmac0607-*vrs1*	*vrs1*	10	28	5.5	CIho 4196
		BF265762A	BG345126	10	27	5.1	CIho 4196
	2001 China	BF254076-*vrs1*	*vrs1*	10	11	3.5	CIho 4196
		BG345126-BG299611	BG345126	10	11	3.5	CIho 4196
	Mean	ABG461C-*vrs1*	BF254076	10	35	5.4	CIho 4196
		BF265762A-MWG503	BG299611	10	33	5.0	CIho 4196
Spike density	1999 Fargo	Hvm54-BF628577	Hvm54	13	20	−0.8	CIho 4196
	1999 Langdon	BF254076-CDO036B	Hvm54	13	37	−0.8	CIho 4196
	1999 Osnabrock	BF254076-CDO036B	Hvm54	13	37	−0.8	CIho 4196
	Mean	BF254076-AF445771B	Hvm54	13	47	−0.9	CIho 4196
Spike angle	1998 Osnabrock	BF216291-BF628577	Hvm54	13	7	−0.4	Foster
	1999 Fargo	BF216291-E6023232	Hvm54	13	17	−0.3	Foster
	1999 Langdon	Hvm54-BF628577	Hvm54	13	17	−0.5	Foster
	1999 Osnabrock	ABG461C-Bmag0140	EBmac0623	7	15	0.6	CIho 4196
		BF216291-BF628577	Hvm54	13	17	−0.5	Foster
	2001 China	ABG072-BE215515	BF628577	14	43	−0.5	Foster
	2001 Osnabrock	ABG072-CDO036B	BF628577	14	23	−0.2	Foster
	Mean	ABG072-AF445771B	BF628577	14	29	−0.4	Foster
Number of rachis nodes with fertile spikelets	1997 Langdon	ABG461C-ABG072	*vrs1*	10	63	2.2	CIho 4196
	1998 Fargo	ABG461C-MWG503	*vrs1*	10	47	2.5	CIho 4196
	1998 Langdon	ABG461C-ABG072	*vrs1*	10	60	1.8	CIho 4196
	1999 Fargo	EBmac0623-MWG503	*vrs1*	10	47	2.0	CIho 4196
	2001 Langdon	EBmac0623-*vrs1*	*vrs1*	10	28	2.4	CIho 4196
	2001 Osnabrock	ABG461C-MWG503	*vrs1*	10	42	2.5	CIho 4196
	Mean	ABG461C-ABG072	*vrs1*	10	63	2.2	CIho 4196

† Bin location is based on the work of Kleinhofs et al. (2005).
‡ SIM = simple interval mapping.
§ CIM = composite interval mapping.

for plant height in the same chromosomal region and concluded that it may actually be the *hcm1* gene. The possibility of two QTLs for plant height flanking the *vrs1* locus has not been reported previously. This arrangement of loci would explain the lack of FHB-resistant plants with acceptable plant height derived from crosses to CIho 4196 (Urrea et al., 2002). Subsequent to their research, F_2 populations with >15 000 plants were grown with the hope that FHB-resistant plants with acceptable plant height would be identified. However, no such plants were found (Horsley, 2002, unpublished data). To overcome this problem, we are crossing the tall resistant plants to susceptible semidwarf plants. It is our goal that the reduced height conferred by the *sdw1* gene in chromosome 3HL will allow us to identify at least one potential parent with acceptable plant height, FHB resistance, and DON accumulation that can be used for additional cycles of crossing.

Other explanations for the lack of recombinants between FHB resistance and plant height are pleiotropy, where a single locus is controlling FHB resistance and plant height, and an escape mechanism where being tall allows the spike of the plant to be further away from the inoculum located on the ground. The last explanation is unlikely true because it is our experience from screening over 8000 accessions from the USDA-ARS National Small Grains Collection that being tall does not lead to better resistance (Scholz et al., 1999). There were many tall accessions that were highly susceptible to FHB. Determining linkage vs. pleiotropy will be much more difficult to resolve and will necessitate fine mapping studies of the centromeric region of 2HL. This research is being conducted by at least two groups in the USA.

A QTL for spike angle was detected in the region of chromosome 2HL flanked by ABG072 and CDO036B (bins 11–15) in all six environments where it was measured (Table 2). The peak LRS values were in the region flanked by Hvm54 and BF628577 (bins 13–14), and the QTL explained 7 to 43% of the variation. Averaged across environments, the QTL for spike angle was flanked by ABG072 and AF445771B and the peak LRS value was near BF628577. This same region also had a QTL for spike density in all three environments where it was measured. The peak LRS values for these traits were in the region near Hvm54 (bin 13). Foster contributed the allele for reduced spike angle score (i.e., more erect spike) and CIho 4196 was responsible for the increased spike density. The correlation between spike angle score and relative spike density ranged from 0.17 to 0.33 ($P < 0.05$). Thus, it is possible that the same QTL may be affecting both traits since spikes with shorter distances between rachis nodes would be expected to have more upright spikes.

A QTL for the number of rachis nodes with fertile spikelets was detected in all six environments where this trait was measured (Table 2). The markers ABC461C and ABG072 flanked the QTL and the peak LRS values were located nearest the *vrs1* locus (bin 10) in all analyses. The QTL explained 28 to 63% of the variation in the progeny for this trait, with Foster contributing the allele for reduced rachis node number. The QTL analysis of mean rachis node number across environments provided similar results. The QTL detected may actually be the *lin1* locus. Previous research by Swenson and Wells (1944) determined that the *lin1* locus controls the number of rachis nodes and they mapped it to about 17 cM above the *vrs1* locus.

On the basis of the information obtained in this study, the region of chromosome 2HL near the centromere flanked by the markers ABG461C and MWG882A (bins 6–10) likely ($P < 0.001$) contains two QTLs each for FHB severity and plant height, and one QTL each for DON accumulation, days to heading, and number of rachis nodes. The importance of this region is reinforced by the fact that QTLs were identified using data obtained from $F_{4:5}$, $F_{4:6}$, and $F_{6:7}$ families, while the map was developed using DNA extracted from $F_{8:9}$ families. The size of this region in our map is about 46 cM, which is in close agreement with the 50 cM observed in the Steptoe × Morex bin map of chromosome 2H (Kleinhofs et al., 2005). The chromosome region chosen for map saturation covers about 30 cM.

A QTL for DON was found in the region of chromosome 4HS flanked by MWG077 and Bmag0375 in three of four environments (Table 3). This QTL explained 9 to 14% of the variation in DON concentration, with CIho 4196 contributing the resistance allele. The peak LRS value was located nearest Bmag0106 in bin 2. Quantitative trait loci for other traits were not found in this region. In previous mapping studies that measured DON concentration, no QTLs were identified in this region. The closest DON QTL reported was in the bin 4 region of 4H contributed by Gobernadora (Zhu et al., 1999).

Quantitative trait loci analyses in this study were done at $P < 0.001$ to reduce the probability of false positives (i.e., Type I error). Others have reported QTLs controlling FHB severity in chromosome regions other than 2H. We repeated the QTL analysis of our data again, this time at $P < 0.05$ to determine if QTLs for reduced FHB severity identified by others could be detected in our population. We identified no additional QTLs for FHB severity or DON concentration.

The putative order and location of QTLs controlling FHB severity, DON accumulation, days to heading, plant height, and number of rachis nodes in chromosome 2HL can be hypothesized from the results of our study. The bin 8 region appears to be the location of linked loci controlling FHB severity and days to heading. This region is also the location of the *Eam6* locus that controls photoperiod response (Franckowiak and Konishi, 2002). The QTL for FHB resistance in bin 8 is the one with the lesser effect as described previously. The plant height and rachis node number QTL, and the FHB resistance QTL with the larger effect are likely located in the bin 10 region of chromosome 2HL, the same region where the *vrs1* locus controlling row-type, the *hcm1* locus controlling plant height, and the *lin1* locus controlling rachis node number are located. A QTL for DON accumulation also mapped to this region; however, from our results we cannot conclude if the QTLs for FHB severity and DON accumulation are linked loci or the same

Table 3. Marker region, closest marker to the peak, and bin location in chromosome 4H, percentage of variation explained by the quantitative trait loci (QTLs), and additive regression coefficient (Add) for a QTL controlling deoxynivalenol accumulation (DON) in four environments, 1998 to 2001, and averaged across environments.

Trait	Environment	Region	Closest marker to peak	Bin†	% variation explained	Add
DON	1997 Langdon	MWG077-Bmag0375	Bmag0106	2	12	−9.5
	1998 Osnabrock	MWG077-Bmag0375	Bmag0106	2	14	−8.5
	2001 Langdon	–	–	–	–	–
	2001 Osnabrock	MWG077-Bmag0375	Bmag0106	2	9	−10.9
	Mean	ABG461E-ABC303	BF261363B	2	10	−9.0

† Bin location is based on the work of Kleinhofs et al. (2005).

locus. The fine mapping research of the bin 10 region being conducted in the U.S. should allow for a better understanding of the genetics of FHB resistance and its relationship with DON accumulation, plant height, rachis node number, and row type.

As mentioned earlier, other research groups also identified FHB resistance QTLs in the bin 8 to bin 10 region of chromosome 2HL (Canci et al., 2004; Dahleen et al., 2003; Mesfin et al., 2003; Ma et al., 2000; de la Pena et al., 1999; Zhu et al., 1999). It is tempting to conclude that the different resistant parents (i.e., CIho 4196, Fredrickson, Chevron, Zhedar 2, and Azafran) all have the same QTL controlling FHB severity; however, additional research must be done to confirm this hypothesis. Extensive mapping of populations derived from crosses of resistant × resistant parents or isolation and sequencing of the genes responsible for FHB resistance could answer this question. However, this would require extensive time and effort. At this time, it is probably best to focus on one or a few parents to determine if loci contributing to FHB resistance can be separated from the associated morphological traits and the genes for FHB resistance identified.

To simplify discussion of the two QTLs controlling FHB severity in chromosome 2HL, we provisionally named the QTL with the lesser effect in bin 8 *Qrgz-2H-8* and the QTL in bin 10 with the larger effect *Qrgz-2H-10*. The provisional name for the QTL for DON concentration in bin 2 of chromosome 4H is *QDON-4H-2*.

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