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ORIGINAL PAPER

QTL associated with Fusarium head blight resistance in the soft red winter wheat Ernie

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Abstract Fusarium head blight (FHB), mainly caused by *Fusarium graminearum* Schwabe [telomorph: *Gibberella zeae* Schw. (Petch)], is an increasingly important disease of wheat (*Triticum aestivum* L.). Host-plant resistance provides the best hope for reducing economic losses associated with FHB, but new sources of resistance are limited. The moderately resistant winter wheat cultivar, Ernie, may provide a source of resistance that differs from Sumai 3 but these genes have not been mapped. Also hindering resistance breeding may be associations of resistance with agronomic traits such as late maturity that may be undesirable in some production environments. This research was conducted to identify QTL associated with type II FHB resistance (FHB severity, FHBS), and to determine if they are associated with days to anthesis (DTA), number of spikelets (NOS), and the presence/absence of awns. Two hundred and forty-three F_8 recombinant inbred lines from a cross between the resistant cultivar, Ernie and susceptible parent, MO 94-317 were phenotyped for type II FHB resistance using point inoculation in the greenhouse during 2002 and 2003. Genetic linkage maps were constructed using 94 simple sequence repeat (SSR) and 146 amplified fragment length polymorphic (AFLP) markers. Over years four QTL regions on chromosomes 2B, 3B, 4BL and 5A were consistently associated with FHB resistance. These QTL explained 43.3% of the phenotypic variation in FHBS. Major QTL conditioning DTA and NOS were identified on chromosome 2D. Neither the QTL associated with DTA and NOS nor the presence/absence of awns were associated with FHB resistance in Ernie. Our results suggest that the FHB resistance in Ernie appears to differ from that in Sumai 3, thus pyramiding the QTL in Ernie with those from Sumai 3 could result in enhanced levels of FHB resistance in wheat.

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Introduction

Fusarium head blight (FHB), mainly caused by *Fusarium graminearum* Schwabe [telomorph: *Gibberella zeae* Schw. (Petch)], is a devastating disease of wheat (*Triticum aestivum* L. and *T. durum* L.) in warm and humid regions of the world (Parry et al. 1995). Although host-plant resistance has long been considered the most practical and effective means of control (Schroeder and Christensen 1963; Martin and Johnston 1982), breeding has been hindered by a lack of effective resistance genes and by the complexity of the resistance in identified sources (Mesterházy 1995). Effective use of available sources of resistance has been additionally hindered by the highly labor intensive disease evaluation procedures that must be done in the adult plant and by variability associated with field-based evaluation methods (Campbell and Lipps 1998). The identification of

quantitative trait loci (QTL) associated with genetically different sources of resistance and their incorporation into adapted wheat varieties may enable breeders to accelerate the development of wheat varieties with improved resistance *per se*, resistance under higher inoculum loads, and/or enhanced stability of resistance over broad geographical areas.

Sumai 3, an FHB-resistant wheat cultivar developed in China, is arguably the most widely used source of type II FHB resistance globally. It forms the basis of the FHB resistance in many US wheat breeding programs (Rudd et al. 2001). A major QTL on 3BS (*Qfhs.ndsu-3BS*) associated with the type II resistance in Sumai 3 (Anderson et al. 2001) and its derivatives has been well characterized (Bai et al. 1999; Zhou et al. 2002; Buerstmayr et al. 2002). Despite differing from Sumai 3 by descent, populations involving other Chinese FHB-resistant cultivars including Wuhan-1, Wangshuibai, and Ning 894037, have been shown to have a QTL in the same region (Somers et al. 2003; Mardi et al. 2005; Shen et al. 2002).

As more simple sequence repeats (SSRs) have become available in wheat (Röder et al. 1998; Somers et al. 2004; Song et al. 2005), additional QTL associated with FHB resistance have been identified in populations developed from wheat varieties from South America and Europe including Frontana, Arina, and Fundulea 201R (Mardi et al. 2006; Steiner et al. 2004; Paillard et al. 2004; Shen et al. 2003). Based on chromosome locations and linked markers these studies suggest that the sources of FHB resistance from South America and Europe differ from that in Chinese cultivars.

Anecdotal evidence from breeding programs suggests that resistance breeding may also be hindered by associations between FHB resistance and other important agronomic traits with linkages being hypothesized between resistance and the presence/absence of awns and flowering time. Examination of these associations in genetic studies of type II resistance are limited and those papers in which these traits have been studied, show mixed results. Snijders (1990) was among the first to report linkage of FHB resistance with the B_1 gene on 5AL for the presence of awns. Mesterházy (1995) reported that awned genotypes were more susceptible to FHB under natural epidemics but found that this effect was absent in artificially inoculated trials while Gervais et al. (2003) found a linkage between one resistance QTL and the presence of awns. Similar results have been reported for the relationship between FHB resistance and flowering date. Buerstmayr et al. (2000) and Steiner et al. (2004) found that in general, FHB symptoms were not correlated with flowering date while Gervais et al. (2003) found a negative correlation between FHB severity and flowering date. Somers et al. (2003) also reported a negative correlation between days to heading and infection under field spray inoculation. These different results have been hypothesized to result from differences in genotypes, experimental environments, inoculation methods, and/or *Fusarium* species.

Ernie, an early maturing awnless soft red winter wheat, released by the University of Missouri Agricultural Experiment Station in 1994 (McKendry et al. 1995) has a moderately-high level of type II resistance to FHB that, based on pedigree analysis, is not derived from Chinese or other known sources of resistance. It is widely used in US breeding programs as a complementary source of resistance to Sumai 3. Currently Ernie is a resistant check in both the Northern and Southern US Winter Wheat FHB Nurseries. Conventional genetic analyses indicated that the resistance in Ernie appears to be controlled by a minimum of four additive genetic factors (Liu et al. 2005); however, these resistance genes have not been mapped. The objectives of this research were: (a) to identify QTL associated with type II FHB resistance in Ernie (b) to determine if they differ from those QTL identified in Sumai 3 and its derivatives, (c) to map agronomic traits including days to anthesis, number of spikelets on the inoculated head, and (d) to determine if these agronomic traits and awnedness are associated with type II FHB resistance in Ernie.

Materials and methods

Disease evaluation

A set of F_3-derived F_8 recombinant inbred lines (RILs), developed at the University of Missouri from the cross Ernie/MO 94-317, was used for QTL analysis. Ernie is awnless and moderately resistant (20% FHBS), whereas MO 94-317 is awned and highly susceptible (80% FHBS). Ernie is early maturing with a shorter spike length while MO 94-317 is relatively late maturing with a longer spike. Development of these lines was described by Liu et al. (2005). Briefly, RILs used in the current study were F_3-derived from a field-grown F_2 plot, consisting of approximately 5,000 plants which was bulk-harvested and advanced to the F_3. The F_3 population was planted without selection as a 100-ft plot from which 1,000 F_3 heads were randomly taken. Single-seed-descent was used to advance those 1,000 plants to the F_7. We kept the larger population to maintain phenotypic variance representative of the original F_2. Theoretically some F_2 plants may have contributed more than once to the final population. If this was the case, this bias may have decreased the power to detect QTL and QTL effects. The genetic similarity between RILs was tested using JoinMap. It was determined with a 95% confidence limit, that no two lines were genetically similar; therefore, the probability of bias was low.

For QTL analyses, a random sub-set of 243 F_8 RILs was screened for type II FHB resistance, days to anthesis, number of spikelets on the inoculated head, and presence/absence of awns in the greenhouse environment. Although we acknowledge the need to verify these QTL in the field environment, we elected to initially conduct this experiment in the greenhouse because of the variability associated with type II reactions in the field. The reproducibility of type II phenotyping is essential to QTL analyses and the greenhouse environment provided us with precision in inoculation technique, a consistently high level of disease pressure, and the absence of confounding effects of other types of resistance that can plague field estimates.

The experiment was conducted with F_8 RILs in 2002 and was repeated with F_9 RILs in 2003. In 2002, the experiment was replicated three times while in 2003, four replications were used. In both years, eight plants per RIL per replication were planted and evaluated. The mean phenotypic value for each numeric trait of those eight plants was considered the experimental unit for statistical analyses. Within each replication, RILs were randomized by line in D40 Deepots (Hummert International, Earth City, MO, USA) to permit plants to be removed individually from the replication at first anthesis for inoculation and replaced into the design following disease initiation.

The procedures for inoculum preparation, inoculation and scoring were as described by Liu et al. (2005). Briefly, each plant was inoculated at first anthesis with a 10 µl suspension of *F. graminearum* macroconidia (concentrated to 50,000 spores ml^{-1}). The inoculum was placed in the basal floret of a central spikelet. Plants were incubated in a mist chamber at 100% relative humidity for 72 h post-inoculation to initiate disease development and then returned to the greenhouse bench to enable disease progression in the head. Disease levels were scored at 21 days post inoculation. Data collected included the total number of spikelets on the inoculated head (NOS) and the number of those spikelets showing disease symptoms. The FHB severity (FHBS) was computed as number of diseased spikelets/NOS, expressed as a percentage. Agronomic data collected included the presence/absence of awns and days to anthesis (DTA), the latter determined as the number of days between transplanting and anthesis.

Molecular marker analysis

Leaf tissue was harvested from tillers at Zadoks 30 (Zadoks et al. 1974), the erect stage of stem development. DNA was extracted using DNAzol (Invitrogen, Carlsbad, CA, USA), quantified using a spectrophotometer (Bio-tek, Winooski, Vermont) and diluted to 25 ng/µl. Both AFLP and SSR markers were used for genotyping.

Amplified fragment length polymorphism procedures followed the manufacturer's recommendation for the AFLP System I kit from Invitrogen (Carlsbad, CA, USA). Eight *Eco*RI and eight *Mse*I primers forming 64 primer combinations were used with one selective nucleotide for pre-amplification and three nucleotides for selective amplification. Standard nomenclature for *Eco*RI/*Mse*I primer combinations was used for naming AFLP markers (http://www.wheat.pw.usda.gov/ggpages/keygeneAFLPs.html) followed by an underscore and a number designating the polymorphic band (e.g. *Xe36m50_1*). The *Eco*RI selective primer was labeled using ^{33}P and 1 µl of the resulting polymerase chain reaction (PCR) reaction was run on 7% polyacrylamide gel at 55°C for 3.5 h. Gels were dried and exposed to film. Polymorphic markers were visually identified on the developed film and were used to genotype the mapping population. Polymorphic AFLP markers were visually scored as Ernie allele (A) and MO 94-317 allele (B) on the mapping population.

For SSR marker analysis, 260 Xgwm and Xwms primers (Röder et al. 1998; Plaschke et al. 1995) and 160 Xbarc primers (Song et al. 2005) were screened to identify polymorphic loci between Ernie and MO 94-317. The PCR contained 50 ng of each SSR primer, 50 ng of genomic DNA, 10 µl of Jumpstart Ready Mix REDTaq^{TM} PCR reaction mix (Sigma-Aldrich, St. Louis, MO, USA) plus sterile distilled water to a volume of 20 µl per reaction. Thermocycling was as follows: 94°C for 1 min, 65°C (or optimum annealing temperature plus 10°C) for 1 min, and 72°C for 1.5 min for the first ten cycles followed by a 1° decrease in the annealing temperature per cycle. The regime thereafter was 94°C for 1 min, 55°C (or the optimum annealing temperature of that specific primer) for 1 min, 72°C for 1.5 min, repeated for 30 cycles. Amplified products were resolved by electrophoresis using a 4.5% super fine resolution agarose gel (Amresco, Solon, OH, USA) of 200 ml volume made with 1× TBE buffer and 10 µl ethidium bromide (10 mg/ml) and run at 130 V for a duration long enough to separate polymorphic bands (1–2 h depending on the marker). The SSR gel pictures were taken under UV light and manually scored. Each marker was run with a 100 bp molecular marker ladder to estimate the band size. Polymorphic markers were used to genotype the mapping population.

Statistical analysis

Prior to analyses of variance (ANOVA), tests for normality were conducted using PROC UNIVARIATE NORMAL PLOT (SAS Institute Inc. 2005, Cary, NC, USA). Homogeneity of variance was tested using Bartlett's test to determine whether the data could be combined across years for analysis. ANOVA was conducted on the two individual

years of data and the combined data over years using PROC MIXED in SAS (SAS Institute Inc., Cary, NC, USA). The statistical model included RILs, year, RILs × year, replication and random experimental error terms. In the model, RILs were considered as fixed effects while other terms were considered as random effects. For the across year ANOVA, the Satterthwaite approximation was used to calculate degrees of freedom for rep (year) and pooled error terms due to differences in the number of replications between the 2 years. *F* tests for years and RILs were conducted against the RILs × year interaction if the interaction term was significant. The RILs × year interaction was tested against the experimental error. Broad-sense heritabilities were determined from the ANOVA for each trait both within and across years. Ninety-five percent confidence limits were calculated following the procedure of Knapp et al. (1985).

Map construction and QTL analyses

Linkage maps were constructed using MapMaker 3.0 (Lander et al. 1987). Initial chromosome locations of SSRs were determined based on the International Triticeae Mapping Initiative (ITMI) reference map (Röder et al. 1998; Song et al. 2005). The Kosambi mapping function was used to estimate the distance between markers (Kosambi 1944). Markers were grouped with a threshold log likelihood ratio (LOD) of 3.0 and distance < 37 cM. Linkages with larger distances were only allowed on chromosomes if they were consistent with the ITMI map. Marker order within groups was determined using the try, compare and ripple commands. Since the mapping population is comprised of RILs, additive and additive-by-additive interactions were evaluated in the QTL model as dominance and dominance associated epistasis should be negligible. QTL analysis was conducted with composite interval mapping (CIM) using windows QTL Cartographer (WinQTLCart 2.0), (Zeng 1994; Wang et al. 2004). Based on 1,000 permutations (Churchill and Doerge 1994), LOD scores of 4.0, 5.0, and 4.7 were determined to be the threshold values to declare significant QTL for FHBS, DTA, and NOS, respectively, on data combined across years. Co-factors were identified using forward and backward stepwise regression with $P(F_{in}) = P(F_{out}) = 0.01$. When multiple peaks were found within a single marker interval, the location with the highest LOD score was defined as the QTL peak. A one-LOD drop from the peak position was used as a confidence interval for each QTL location. If another QTL peak appeared at more than 20 cM away, it was claimed as a separate QTL. The R^2 values of significant QTL were obtained from multiple interval mapping (MIM) from WinQTLCart 2.0 using all significant QTL from CIM (Wang et al. 2004). Interactions between significant QTL were analyzed using MIM. All possible pairs of marker combinations were tested for significance using SAS PROC GLM and significant interaction terms with $R^2 > 5\%$ were included in the model. QTL maps were drawn using MapChart 2.1 (Voorrips 2002).

Phenotypic effects of major QTL alleles were estimated using closely linked markers. Recombinant inbred lines carrying the same QTL alleles were grouped. Mean FHBS data were averaged across RILs within group and mean comparisons were made using Fisher's protected least significant difference (LSD) at $P = 0.05$. Percent reduction in FHBS associated with QTL alleles was determined.

Results

Trait analyses

For individual year data and that combined over years, the frequency distribution for FHBS approximated a normal distribution confirming the quantitative nature of type II FHB resistance in Ernie (Liu et al. 2005). Error variances were homogeneous over years; therefore, individual year data were combined for analyses. Highly significant genotypic effects for FHBS were detected among RILs in each year as well as in the multi-year analysis (Table 1). The year effect was highly significant indicating environmental effects are important contributors to the phenotype. Replication effects were also highly significant. These latter effects were likely due in part to variation in temperature and photoperiod over the duration of the experiment. Despite environmental effects, FHBS data were highly correlated ($r = 0.75$; $P < 0.001$) between the 2 years of testing. Combined FHBS data for the resistant parent, Ernie (22%) and the susceptible parent, MO 94-317 (80%) illustrate the genetic distance between parents in this study (Table 2). Population means for FHBS in 2002 and 2003 were 66.2–57.3%, respectively, and ranged over the 2 years from 9.7 to 100% (Table 2). Broad sense heritabilities for FHBS were 91% in both 2002 and 2003, and 83% across 2 years (Table 2).

Analyses of variance for DTA and NOS indicated significant effects among RILs both for individual year data and in data combined over years (Table 1). Phenotypic differences observed between means of the two parents were small or negligible for both traits. However, RIL means for NOS ranged from 7.9 to 18 spikelets and for DTA ranged from 48.5 to 77.9 days over the 2 years, demonstrating the existence of allelic differences between the two parents for these traits. Heritability estimates from the combined data for NOS and DTA were 71 and 74%, respectively (Table 2). Pearson correlation analysis indicated that DTA and NOS were highly correlated ($r = 0.83$,

Table 1 Mean squares for Fusarium head blight severity (FHBS), days to anthesis (DTA) and number of spikelets on the inoculated head (NOS) from recombinant inbred lines (RILs) of the soft red winter wheat cross, Ernie/MO 94-317 inoculated with *Fusarium graminearum*

Year	Source[a]	*df*	FHBS[b]	DTA	NOS
2002	RILs	242	1,556***	310***	29***
	Replications	2	4,506***	93,141***	57***
	Error	484	138	11	1.9
2003	RILs	242	1,955***	77***	8***
	Replications	3	15,728***	7,045***	220***
	Error	726	174	11	1.5
Combined	Years	1	34,616***	843,808***	623***
	Rep(years)	5	11,163***	41,497***	154***
	RILs	242	2,995***	336***	32***
	RILs × years	242	512***	87***	9***
	Error	1,210	159	11	1.5

Experiments were conducted in the greenhouse at the University of Missouri-Columbia in 2002 and 2003

*** Significant at $P < 0.001$

[a] Years and RILs were tested against the RILs × year interaction. The RILs × year and rep(year) interactions were tested against the experimental error for combined ANOVA

[b] FHBS = number of diseased spikelets/number of spikelets on the inoculated head expressed as a percentage

$P < 0.0001$), however, neither was significantly correlated with FHBS.

In both years, the proportion of awned and awnless RILs in our mapping population was about equal. In 2002 FHBS means for awned and awnless RILS were 69.7 and 63.0%, respectively, whereas in 2003, FHBS means for the two respective groups were both 57%. These differences were not statistically significant (data not shown). Pearson correlation analysis indicated that awnedness was not correlated with FHBS ($r = -0.1$, $P > 0.2$).

Molecular map construction

Polymorphism between Ernie and MO 94-317 was assessed using 64 *Eco*RI/*Mse*I AFLP primer pairs and 420 SSR markers. Among AFLP markers, 52 primer pairs generated at least two polymorphic fragments between parents. Scored fragments ranged from 100 to 500 bp. The 14 most polymorphic primer pairs produced 1,023 fragments, 162 (16%) of which were polymorphic. Ninety-three of 420 SSR primers (22%) were polymorphic between Ernie and MO 94-317 producing 100 loci that were scored on the RILs.

In total, 162 AFLP and 100 SSR loci were used to construct the genetic linkage groups. Forty-six linkage groups were generated with 146 AFLP and 94 SSR loci while the remaining 22 loci were unlinked. Using SSRs as anchor markers, 139 markers, including 94 SSR and 45 AFLP loci from 29 linkage groups were mapped onto 19 chromosomes. The remaining 17 linkage groups containing only AFLPs could not be linked to chromosomes because of a lack of anchored SSRs. The 22 markers on these 17 linkage groups were tested and none was significantly associated with FHBS, therefore, these linkage groups were not included in this report.

The linkage map covered 2,086 cM of the wheat genome with an average of 15 cM between loci. Chromosomes 2A, 2B, 2D, 3A, 3B, 4A, 5A, 5B, 7A, and 7D had better coverage than other chromosomes. For example, 23 markers spanning 168.5 cM with an average of 7 cM per marker were mapped on chromosome 2B, while 18 markers covering

Table 2 Means, ranges, and broad sense heritabilities (H^2) for FHBS, days to anthesis (DTA), and number of spikelets on the inoculated heads (NOS)

Traits	Year	Ernie	MO 94-317	RILs	Range	H^2	95% CI [a]
FHBS[b] (%)	2002	23 ± 2	89 ± 7	66.2 ± 6.8	9.7–100	0.91	0.89–0.93
	2003	20 ± 2	72 ± 5	57.3 ± 8.6	10.2–92	0.91	0.89–0.92
	combined	22 ± 2	80 ± 2	61.0 ± 4.8	10.7–92	0.83	0.79–0.86
DTA (days)	2002	60 ± 1.3	59 ± 2.6	88.1 ± 1.9	70.3–110	0.96	0.95–0.97
	2003	39 ± 1.9	42 ± 2.5	43.0 ± 1.7	34.4–60	0.85	0.82–0.88
	Combined	50 ± 1.6	51 ± 2.5	62.2 ± 1.3	48.5–78	0.74	0.68–0.79
NOS	2002	11 ± 1.0	13 ± 1.0	13.1 ± 0.8	7.8–20.8	0.94	0.92–0.95
	2003	11 ± 0.3	12 ± 1.0	11.8 ± 0.6	7.9–16.8	0.83	0.79–0.85
	Combined	11 ± 0.7	12 ± 1.0	12.4 ± 0.5	7.9–18.0	0.71	0.65–0.77

Data are for parents and recombinant inbred lines (RILs) from the soft red winter wheat cross, Ernie/MO 94-317. Experiments were conducted in the greenhouse at the University of Missouri-Columbia in 2002 and 2003

[a] 95% confidence intervals (CI) for the heritability estimate

[b] FHBS = number of diseased spikelets/number of spikelets on the inoculated head expressed as a percentage

200 cM with an average of 11 cM per marker were mapped on chromosome 3B. On some chromosomes (1BL, 3DL, 4AL, 4BL, 5DL, and 7BS), markers were mapped to only one arm. Due to the scarcity of markers on some linkage groups, marker densities for each of the QTL regions differed which may have led to an under- or over-estimation of the magnitude of QTL.

QTL identification and their associations among traits

Four QTL regions, named as *Qfhs.umc-2B*, *Qfhs.umc-3B*, *Qfhs.umc-4BL*, and *Qfhs.umc-5A* on chromosomes 2B, 3B, 4BL and 5A were consistently associated with FHB resistance in Ernie in both years (Table 3, Fig. 1). The only exception was that the peak of the 2B QTL from 2003 mapped 15 cM away from the peak from 2002 and that for the combined data. The estimated proportions of the phenotypic variation explained by all four QTL were 39.7 and 35.1% from 2002 and 2003, respectively. For the combined data, QTL *Qfhs.umc-5A*, *Qfhs.umc-3B*, *Qfhs.umc-4BL*, and *Qfhs.umc-2B* accounted for 43.3% of the total phenotypic variation with individual QTL having R^2 values of 17.4, 12.9, 8.8 and 4.2%, respectively. Additive genetic effects of these QTL regions ranged from 6.1 to 8.8% and were associated with reduced FHBS (Table 3). Based on LOD scores, R^2 values, and additive effects, *Qfhs.umc-5A* and *Qfhs.umc-3B*, appeared to have larger effects than *Qfhs.umc-4BL*, and *Qfhs.umc-2B*.

Qfhs.umc-5A is flanked by *Xbarc56* and *Xbarc165* (Fig. 1). The distance between these two markers is about 20 cM and the QTL peak is 9 cM away from *Xbarc165*. The marker density of the *Qfhs.umc-4BL* region is better and the QTL is positioned exactly on the marker itself (*Xgwm495*). *Qfhs.umc-3B* is centromeric and located between an SSR marker, *Xgwm285* and an AFLP marker, *Xe41m50_6* with the QTL peak 8 cM away from *Xgwm285*. The minor QTL, *Qfhs.umc-2B*, is located on chromosome 2B and is closely linked to the SSR marker, *Xgwm276b*.

Two major QTL, *QNos.umc-2D* and *QDta.umc-2D* on chromosome 2D, were identified that were associated with NOS and DTA, respectively. They were co-localized on chromosome 2D (Table 3, Fig. 2) and, based on the combined data, explained 51.5 and 73.7% of phenotypic variation for these two traits, respectively. These two major QTL alleles were both from the susceptible parent, MO 94-317. No common QTL were detected between FHBS and either DTA or NOS. Finally, B_1, the morphological marker conditioning awnedness, which has been mapped distally on chromosome 5AL, is not associated with the FHBS QTL, *Qfhs.umc-5A*, identified in this study. This is consistent with data presented earlier which indicates that awnedness is not correlated with FHBS in this population.

Major QTL effects

The allelic effects of QTL combinations of the three major QTL are summarized (Table 4). The minor QTL on 2B was not included in this analysis. Forty-seven RILs carried none of the three major QTL alleles from Ernie ($M_1M_1M_2M_2M_3M_3$). Mean FHBS in this group of RILs was 73.2%. When FHBS was analyzed across genotypes that carried one, two and three Ernie alleles for the QTL on chromosomes 3B, 4BL, and 5A, three groups with significantly different FHBS means were identified. Eighty-eight

Table 3 Quantitative trait loci (QTL) associated with the Fusarium head blight severity (FHBS), days to anthesis (DTA) and number of spikelets on the inoculated head (NOS) of recombinant inbred lines from the soft red winter wheat cross, Ernie/MO 94-317

Trait[a]	QTL[b]	Marker[c]	2002			2003			Combined		
			LOD[d]	*A*[e]	R^2 (%)[f]	LOD	*A*	R^2 (%)	LOD	*A*	R^2 (%)
FHBS	*Qfhs.umc-2B*	*Xgwm276b*	3.9	−6.3	4.4	3.6	−6.1	4.9	4.7	−6.1	4.2
	Qfhs.umc-3B	*Xgwm285*	2.9	−5.7	7.6	5.1	−7.5	13.1	6.1	−7.7	12.9
	Qfhs.umc-4BL	*Xgwm495*	6.3	−7.3	8.1	4.9	−6.4	7.0	7.3	−7.0	8.8
	Qfhs.umc-5A	*Xbarc165*	7.6	−10.0	19.6	3.9	−6.6	10.1	6.9	−8.8	17.4
DTA	*Qdta.umc-2D*	*Xbarc95*	21.3	9.9	81.8	2.7	1.5	14.4	10.0	6.2	73.7
NOS	*Qnos.umc-2D*	*Xbarc95*	9.1	2.2	44.7	1.6	0.4	14.8	9.0	1.5	51.5

Analyses were conducted for individual experimental years (2002 and 2003) and for data combined over years

[a] FHBS = number of diseased spikelets/number of spikelets on the inoculated head expressed as a percentage

[b] Nomenclature for QTL is as follows: Q for QTL followed by a trait designator, a period, a laboratory designator, a hyphen (-) and the symbol for the chromosome on which the QTL is located

[c] Marker linked to the QTL peak

[d] The peak position had the highest LOD score in that marker interval along the chromosome scanning every 1 cM

[e] The additive effects from the marker at the peak LOD

[f] Amount of phenotypic variation explained by significant QTL based on multiple interval mapping (MIM) estimations

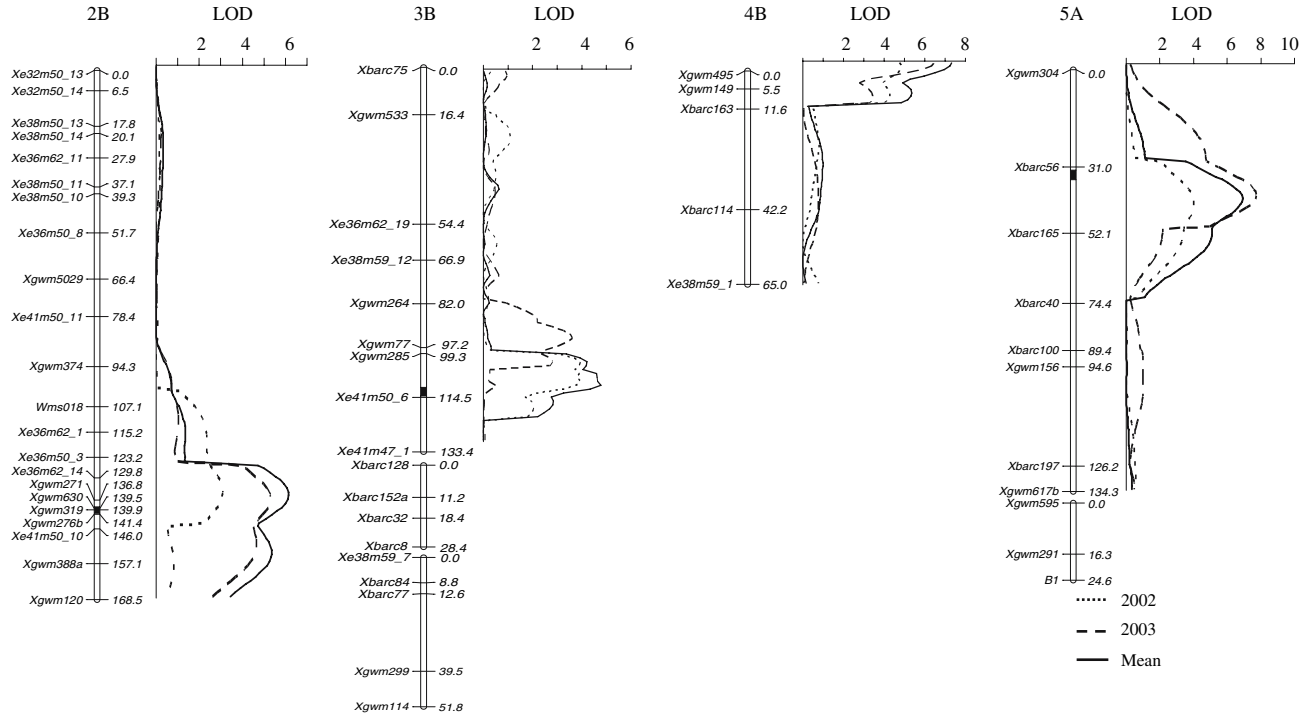

Fig. 1 QTL associated with Fusarium head blight severity (*FHBS*) in the soft red winter wheat cross Ernie/MO 94-317 detected by composite interval mapping. The LOD scores were plotted against centimorgans on the chromosome. Data are for individual years (2002 and 2003) and combined 2 years. The *black square* inside the chromosome indicates the position of the centromere. For chromosomes 3B and 5A, three and two unlinked segments, respectively, were obtained. Anchored SSR marker locations on chromosomes are based on the reference International Triticeae Mapping Initiative mapping population (Röder et al. 1998; Song et al. 2005)

lines carried one of the three Ernie QTL alleles. This group averaged 65.4% FHBS; 7.8% better than lines carrying MO-94-317 alleles across the three QTL. A second group of 69 RILs carried two of the three Ernie QTL alleles and one allele from MO 94-317 across the three QTL. This group averaged 53.1% FHBS, reflecting a 20.1% improvement in FHB resistance compared with RILS that carried three alleles from MO 94-317. A final group of 16 RILs carried alleles from Ernie at all three QTL (genotype $E_1E_1E_2E_2E_3E_3$) and belong to the most resistant group with a mean FHBS of 33%. Fusarium head blight resistance was improved by 40.2% in this group compared with RILs carrying all three alleles from MO 94-317. These data suggest that where alleles from the three major Ernie QTL are incorporated into a wheat line, FHB resistance should be significantly enhanced.

Discussion

Breeding for resistance to FHB has been slowed by the lack of genetically different sources of resistance. Among the types of resistance reported for FHB, type II resistance has proven valuable to breeders because it has good combining ability in crosses, can be precisely assessed using greenhouse techniques, and is repeatable. Arguably, this has been the most widely used type of resistance in US breeding programs to date (Bai and Shaner 1994; Mesterházy 1995; Rudd et al. 2001). Although most breeders agree that type II resistance alone is not sufficient to confer high levels of FHB resistance in the field, a variety with a moderate level of type I resistance and no type II resistance, will certainly fail under disease pressure in the field environment. The value of type II resistance is clearly evidenced by the wide-spread use and study of FHB resistance from the Chinese variety Sumai 3 and its derivatives. Sumai 3 has excellent levels of type II resistance and only a low level of type I resistance, yet its use in US breeding programs has led to a significant increase in the FHB resistance level of germplasm in many US programs. The identification of genetically different sources and types of resistance is a critical goal for enhancing the levels of FHB resistance in winter wheat and thus, study of the resistance in the soft red winter wheat cultivar Ernie, which differs from Sumai 3 by descent, was warranted.

Accurate, repeatable phenotypic data are critical to QTL analyses. The reproducibility of type II phenotypic data requires precision in inoculation technique, a consistently high level of disease pressure, minimization of the effects of the environment and the absence of the confounding effects of other types of resistance. In order to maximize control

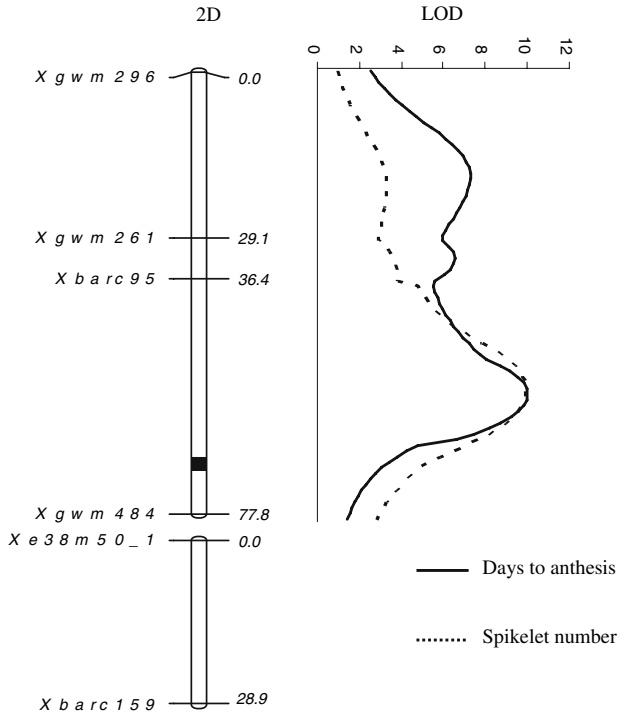

Fig. 2 QTL associated with mean days to anthesis (*DTA*) and number of spikelets on the inoculated head (*NOS*) in the soft red winter wheat cross Ernie/MO 94-317, identified using composite interval mapping. Data presented for each trait are combined data over two experimental years, 2002 and 2003. The LOD scores from each centimorgan of the chromosome were plotted against the chromosome. The *black square* inside the chromosome indicates the position of the centromere. Two segments were obtained for chromosome 2D

over these variables, this experiment was conducted in the greenhouse rather than in the field environment. Our success in control of these variables is evidenced by the high correlation ($r = 0.75$) of the RIL data over years.

Four QTL regions on chromosomes 2B, 3B, 4BL, and 5A were associated with increased type II FHB resistance. Each of these QTL alleles was derived from Ernie. These data were consistent with previously published results from conventional genetic analyses which indicated that resistance in Ernie is conditioned by a minimum of four additive genetic factors (Liu et al. 2005).

Qfhs.umc-5A, linked to *Xbarc165*, ($R^2 = 17.4\%$) has not been identified in Sumai 3. Gervais et al. (2003) identified three QTL on 5A in a population involving the European wheat Renan. *Qfhs.inra-5a1* was mapped to the short arm of 5A while both *Qfhs.inra-5a2* and *Qfhs.inra-5a3*, were on 5AL. *Qfhs.inra-5a3* is distally located between *Xgwm595* and B_1 and therefore, probably differs from *Qfhs.umc-5A*. *Qfhs.inra-5a2* linked to *Xgwm639b* was 23 cM distal to *Xgwm156*. Based on the common marker locations between the two studies, the *Qfhs.umc-5A* which is located close to the centromere appears to be between *Qfhs.inra-5a1* and *Qfhs.inra-5a2*. Steiner et al. (2004) also identified a 5A QTL in Frontana located in the *Xgwm129-Xbarc197* region. This 20 cM region spans 74.4 cM on the ITMI reference map and 126 cM on our map and includes the *Qfhs.umc-5A* region of Ernie. Without further study, it is unclear whether the 5A QTL in these two populations are the same as they were assessed through two different inoculation techniques and Frontana is known to have only an intermediate level of type II resistance. Since the mapping populations were derived from different parents, this may also lead to different genetic distances between markers. Finally, a 5A QTL with a minor effect (LOD 1.91; $R^2 = 2.1$) has also been identified in Fundulea 201R, linked to *Xgwm304* (Shen et al. 2003). Based on its linkage data *Qfhs.umc-5A* may differ from the 5A QTL in Fundulea 201R.

Table 4 Major QTL effects on Fusarium head blight severity (FHBS) in recombinant inbred lines (RILs) of the cross Ernie/MO 94-317

FHBS QTL	Markers linked to QTL[a]			Number of RILs with QTL alleles	FHBS of RILs with QTL alleles (%)[b]	Reduction in FHBS with QTL alleles (%)[c]
	Xgwm285	*Xgwm495*	*Xbarc165*			
–, –, –	M_1M_1	M_2M_2	M_3M_3	47	73.2 ± 1.2 a	–
3B, –, –	E_1E_1	M_2M_2	M_3M_3	27	66.4 ± 1.4 b	6.8
–, 4BL, –	M_1M_1	E_2E_2	M_3M_3	26	65.8 ± 1.7 b	7.4
–, –, 5A	M_1M_1	M_2M_2	E_3E_3	35	64.1 ± 1.5 b	9.1
3B, 4BL, –	E_1E_1	E_2E_2	M_3M_3	19	51.3 ± 2.0 c	21.9
3B, –, 5A	E_1E_1	M_2M_2	E_3E_3	25	55.1 ± 1.8 c	18.1
–, 4BL, 5A	M_1M_1	E_2E_2	E_3E_3	25	52.9 ± 1.8 c	20.3
3B, 4BL, 5A	E_1E_1	E_2E_2	E_3E_3	16	33.0 ± 2.2 d	40.2

Data are from experiments conducted in the greenhouse at the University of Missouri-Columbia in 2002 and 2003

[a] 'E' allele from Ernie and 'M' allele from MO 94-317 for all markers

[b] Statistical comparisons were made among lines across all combinations of the three major QTL (*Qfhs.umc-3B*, *Qfhs.umc-4BL*, *Qfhs.umc-5A*). Mean FHBS followed by the same letter were not significantly different at $P < 0.001$

[c] Reduction in FHBS compared with the mean FHBS of the 47RILs that carried only MO 94-317 alleles for the 3B, 4BL, and 5A QTL

Similarly, *Qfhs.umc-4BL*, linked to *Xgwm495*, which accounted for 8.8% of the variation has not been identified in Sumai 3. It is, however, in the same chromosome region as a QTL, linked to *Xwmc238* reported by Somers et al. (2003) in a cross of Wuhan1/Maringa. Both are proximal to the centromere. However, the 4BL QTL in the Somers study was identified using field spray inoculation assessed at 21 days post inoculation and therefore may be related to a complex of type I (disease incidence) and type II (disease severity) resistances rather than type II resistance which we evaluated. When type II resistance was assessed using a single floret inoculation technique similar to the one used in the current study, Somers et al. (2003) failed to detect a 4BL QTL. This may be related to the different environmental variances in the two research environments. Whether these QTL, which originate from different types of resistance, are different linked genes needs further study.

Qfhs.umc-2B, linked to *Xgwm276b*, is proximal to the centromere and accounted for 4.2% of the phenotypic variation over years. Two other QTL for FHB resistance have been reported on 2B, however, both were found on the long arm of this chromosome. Zhou et al. (2002) reported a 2B QTL linked to *Xgwm120* in a population with Ning 7840, a Sumai 3 derivative, as did Gervais et al. (2003) for a population involving the European wheat Renan. We believe that the 2B QTL in Ernie is novel.

Finally, a major QTL on 3BS has been identified and well studied in populations with Sumai 3 and its derivatives (Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002; Zhou et al. 2002), in Wangshuibai (Mardi et al. 2005; Lin et al. 2006) and the Chinese germplasm, W14 (Chen et al. 2006). This QTL, flanked by *Xgwm533* and *Xgwm493* (Anderson et al. 2001) is located near the telomeric region of 3BS. We identified a different QTL on 3BS linked to *Xgwm285* which is proximal to the centromere. This QTL is close to a centromeric QTL identified by Somers et al. (2003) in the cross Wuhan-1/Maringa. As with the 4BS QTL in these two genotypes, the centromeric 3BS QTL identified by Somers et al. (2003) was detected using field spray inoculation. When Somers et al. (2003) used point-inoculation, only the Sumai 3 telomeric QTL on 3BS was detected. Again, it is unclear whether the two centromeric 3BS QTL are due to the same gene conditioning different types of resistance or different linked genes.

It is important to note that in this study, all of the FHB resistance QTL alleles identified were from Ernie and none were from the susceptible parent, MO 94-317. Others have reported favorable contributions from the susceptible parent at minor resistance QTL. Waldron et al. (1999) found a QTL on 2AL where the moderately susceptible parent Stoa contributed the favorable allele, while Shen et al. (2002) identified one FHB resistance QTL where the susceptible parent Alondra contributed the favorable allele. Other studies have found transgressive segregation for resistance and the presence of resistance genes in susceptible and moderately susceptible cultivars would explain these findings (Singh et al. 1995). It is possible that resistance alleles in MO 94-317 were not detected due to low marker coverage or were minor QTL and this could account for the low level of transgressive segregation observed in this population.

Breeding agronomically competitive FHB-resistant wheat can also be hindered if FHB resistance QTL are linked to agronomic traits that may be undesirable. Anecdotal evidence from breeding programs as well as published reports have suggested that traits such as increased height and late maturity may be associated with resistance to FHB. Other reports suggest that the presence of awns may be associated with FHB susceptibility.

In winter wheat, anthesis is associated with response to both photoperiod and vernalization. A major photoperiod response gene, *Ppd1*, is located on chromosomes 2D (McIntosh et al. 1993) and a major QTL ($R^2 = 73.7\%$) on 2D was identified in this study that was associated with DTA (Table 3, Fig. 2). The susceptible parent MO 94-317 contributed the late maturing allele. A major QTL ($R^2 = 51.5\%$) for NOS was also co-localized in this region (Table 3, Fig. 2). As expected, DTA and NOS were highly correlated ($r = 0.83$, $P < 0.0001$), however, neither was significantly correlated with FHBS. These data indicate that it should be possible to use the type II source of resistance in Ernie without compromising earliness per se, an important agronomic trait in the Midwestern US. Our results differed from those of Gervais et al. (2003) who found that FHB resistance QTL on chromosome 2B overlapped with a QTL for flowering time in the cross Renan/Recital. One explanation for differences observed in this study versus the work of Gervais et al. (2003) is the phenotyping environment. We have found from our field work that associations with heading date are often artifacts of the field inoculation protocol rather than real effects. Greenhouse protocols enable us to inoculate at precisely the same stage of development, independent of when the crop heads. By doing so, we effectively control for the effect of heading date on phenotypic data and have found, more often than not, that resistances in our program are independent of heading date.

It is not clear from the literature whether the presence of awns influences FHB resistance or not. The awnedness gene, B_1, maps to the distal region of 5AL. Results from our work with Ernie indicate that despite having a major QTL on 5A, there was no association between FHBS and awnedness. Similarly, Buerstmayr et al. (2002) found a QTL on 5A for FHB resistance in CM-82036, a Sumai 3 derivative, that was not associated with the presence or absence of awns. Ban and Suenaga (2000), however, found that one of two major genes in Sumai 3 was linked in repulsion with the B_1 gene while Gervais et al. (2003) found that one of two 5A FHB-resistance QTL from Renan was linked to B_1 gene.

SSR polymorphism in this population was low (22%) compared to other mapping populations in which reported polymorphism has been as high as 40% (Zhou et al. 2002). Consequently, marker saturation on some chromosomes was less than desirable. This lack of marker density in some wheat chromosome areas has been previously reported (Liu and Anderson 2003) and has led to further efforts to design primer pairs from wheat ESTs thought to be near known QTL. This work is ongoing at Missouri but as yet, has not produced many new SSRs in critical chromosome regions. The lack of polymorphism in the population used in this investigation is probably also due to fact that both of the parents were from the same breeding program and therefore, the coefficients of parentage were greater than those in studies involving crosses between Chinese resistant cultivars and more adapted susceptible cultivars. We included AFLPs in this study to offset this problem; however, several of the AFLPs could not be linked to chromosomes due to the lack of anchored SSRs. Although AFLPs were more polymorphic, their unknown chromosome location was a major disadvantage. Despite these difficulties, we were able to identify four significant QTL associated with the FHB resistance in Ernie, two of which have linkages close enough for use, once validated, in marker-assisted-selection.

In summary, it appears that Ernie represents a source of FHB resistance that differs from that in Sumai 3. Addition of novel SSR markers to the map will be done as they are available to saturate regions where the number of markers is limited. Our next goals are to validate the QTL and to identify closely linked markers prior to their use for marker-assisted-selection. F_7 lines in several Ernie-derived backgrounds that are near-isogenic for FHB reaction are being genotyped to validate QTL identified in the study. These QTL in combination with those identified from the Sumai 3 population especially the 3BS major QTL could be combined to significantly enhance resistance in future cultivars. The lack of association among QTL for awnedness, DTA and NOS in Ernie suggest that utilization of this source of resistance for pyramiding FHB alleles can be accomplished without potentially adverse affects due to linkage with other agronomic traits.

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