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ORIGINAL PAPER

B. Steiner · M. Lemmens · M. Griesser · U. Scholz · J. Schondelmaier · H. Buerstmayr

Molecular mapping of resistance to *Fusarium* head blight in the spring wheat cultivar Frontana

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Abstract *Fusarium* head blight (FHB) is a destructive disease of wheat. The objective of this study was to characterise the FHB resistance of the Brazilian spring wheat cultivar Frontana through molecular mapping. A population of 210 doubled-haploid lines from a cross of Frontana (partially resistant) and Remus (susceptible) was evaluated for FHB resistance during three seasons. Spray and single-spikelet inoculations were applied. The severity, incidence and spread of the disease were assessed by visual scoring. The population was genotyped with 566 DNA markers. The major QTL effect associated with FHB resistance mapped to chromosome 3A near the centromere, explaining 16% of the phenotypic variation for disease severity over 3 years. The most likely position is in the *Xgwm720–Xdupw227* interval. The genomic region on 3A was significantly associated with FHB severity and incidence in all years evaluated, but not with FHB spread, indicating the prominent contribution of this QTL to resistance against initial infection. The map interval *Xgwm129–Xbarc197* on chromosome 5A also showed consistent association with FHB severity and accounted for 9% of the phenotypic variation. In addition, smaller effects for FHB severity were identified on chromosomes 1B, 2A, 2B, 4B, 5A and 6B in single years. Individual QTLs for resistance to FHB spread accounted for less than 10% of the variation in trait expression. The present study indicates that FHB resistance of Frontana primarily inhibits fungal penetration (type I resistance), but has a minor effect on fungal spread after infection (type II resistance).

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B. Steiner · M. Lemmens · M. Griesser · U. Scholz · H. Buerstmayr (✉)
Department of Biotechnology in Plant Production, IFA-Tulln, Institute for Agrobiotechnology, Konrad Lorenz Strasse 20, 3430 Tulln, Austria
e-mail: hermann.buerstmayr@boku.ac.at
Fax: +43-2272-66280203

J. Schondelmaier
Saaten-Union Resistenzlabor GmbH, Hovedisser Strasse 92, 33818 Leopoldshoehe, Germany

Introduction

Fusarium head blight (FHB) is a devastating disease of wheat (*Triticum aestivum* L.) world-wide. FHB reduces wheat grain yield and quality. The mycotoxins produced by *Fusarium* spp. are harmful to livestock and are also a safety concern in food. The most effective strategy for controlling FHB in wheat is through the development of resistant cultivars. Resistance to FHB exhibits quantitative variation and its inheritance involves several loci on different chromosomes (Kolb et al. 2001). Genotype × environment interaction complicates the phenotypic evaluation of FHB resistance and makes screening of FHB resistance laborious, time-consuming and costly (Rudd et al. 2001). Identifying resistance genes and understanding the complex genetic structure of FHB resistance will greatly enhance breeding for FHB resistance. FHB resistance of Sumai-3, a wheat line from China, has been well characterised through molecular mapping, resulting in the detection of a major QTL on chromosome 3B that explains up to 60% of the phenotypic variation for fungal spread within the spike. This QTL, *Qfhs.ndsu-3BS*, has been verified by several research groups and is consistent across a wide range of genetic backgrounds and environments (Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002, 2003; Zhou et al. 2002; Bourdoncle and Ohm 2003; Shen et al. 2003a). Other QTLs conferring FHB resistance of Sumai-3 and derivatives have been reported on chromosomes 6B (Waldron et al. 1999; Anderson et al. 2001), 5A (Buerstmayr et al. 2002, 2003) and 2A and 2B (Zhou et al. 2002).

Only limited molecular genetic information is available on other sources of resistance. In the *T. dicoccoides* line LDN(DIC-3A), Otto et al. (2002) detected a QTL for FHB resistance on chromosome 3A. In the European winter wheat cultivar Renan, Gervais et al. (2003) reported nine QTLs for FHB resistance, of which three

were stable over years. Bulked segregant analysis of the FHB resistance of the Romanian winter wheat cultivar Fundulea 201R detected resistance QTLs on chromosomes 1B and 3A (Shen et al. 2003b). In the two moderately resistant cultivars Wuhan-1 and Maringa, QTLs for FHB resistance and QTLs controlling the accumulation of deoxynivalenol (DON) were identified on chromosomes 2DL, 3BS, and 4B, and on 2DS and 5AS, respectively (Somers et al. 2003).

The Brazilian spring wheat cultivar Frontana is a widely used FHB resistance source. Its resistance has been analysed through classic genetic studies, which indicated a minimum of two to three additive genes (Singh et al. 1995; Van Ginkel et al. 1996).

As early as 1963, Schroeder and Christensen (1963) analysed FHB resistance of Frontana and other wheat cultivars. They proposed that at least two components contribute to FHB resistance: resistance to initial infection (type I) and resistance to fungal spread of the pathogen within the spike (type II). This classic model is widely accepted. Additional components of resistance in Frontana appear to be its ability to degrade and tolerate higher DON levels than other genotypes (Miller and Arnison 1986; Wang and Miller 1988). Furthermore, various morphological and agronomic traits may affect the development of FHB on plants in the field. These traits, termed passive resistance mechanisms (Mesterhazy 1995), can interfere with measurement of resistance and may result in apparent resistance by increasing the probability that the host escapes infection by the pathogen rather than reducing disease by a defence response in the host (Kolb et al. 2001).

The objective of this study was to identify loci involved in resistance to initial infection and fungal spread of FHB in the wheat cultivar Frontana through molecular mapping and to study the relationship between FHB resistance traits and some developmental and morphological traits, such as plant height, date of anthesis and spike compactness.

Materials and methods

Plant materials

A population of 210 recombinant F_1-derived doubled-haploid (DH) lines was developed from a cross of Frontana and Remus. Frontana (Fronteira/Mentana) is a Brazilian spring wheat cultivar known for its resistance to FHB (Schroeder and Christensen 1963; Singh et al. 1995; Buerstmayr et al. 1996; Van Ginkel et al. 1996). Remus (Sappo/Mex//Famos) is a spring wheat cultivar released by the Bavarian State Institute for Agronomy in Freising, Germany. It is agronomically well adapted for cultivation in central Europe, but is susceptible to FHB. The DH lines were developed from greenhouse-grown F_1 plants, using the wheat by the maize pollination technique (Laurie and Bennett 1988).

Field experiments

The 210 DH lines and the parents were tested during 3 years (1999, 2001, 2002) at the experimental field of IFA-Tulln, 30 km west of Vienna, at 180 m above sea level. The soil type is a meadow-czernosem. The average temperature and annual precipitation were: 10.4 °C and 611 mm (1999), 9.9 °C and 640 mm (2001) and 10.2 °C and 672 mm (2002).

In each year, three adjacent experiments were sown: experiment 1 was spray-inoculated with *F. graminearum*, experiment 2 with *F. culmorum* and experiment 3 was not spray-inoculated and used as control. In 2001 and 2002, ten heads per plot in experiment 3 were chosen for point inoculations. Each experiment had a randomised complete block design with three (1999) or two blocks (2001, 2002). The blocks were purposely sown several days apart, resulting in a 1-day to 3-days difference in anthesis between the blocks. Plots consisted of double rows with a row spacing of 17 cm and 1 m in length. Seed treatment, sowing density and crop management were the same as described by Buerstmayr et al. (2002).

Inoculation technique and disease assessment

FHB severity

In 1999, 2001 and 2002, the DH population and the parental lines were evaluated for FHB resistance after spray inoculation as described by Buerstmayr et al. (2003). Two single-spore *Fusarium* isolates were applied. Macroconidia of the *F. culmorum* isolate IPO 39-01 and the *F. graminearum* isolate IFA 65 were prepared as described by Buerstmayr et al. (2000, 2002). Spray inoculation was performed individually on each plot when 50% of the plants had reached anthesis and was repeated 2 days later. Using a motor driven back-pack sprayer, 100 ml conidial suspension with a spore concentration of 5×10^4 ml^{-1} was sprayed onto the heads. An automated mist-irrigation system maintained high humidity for 20 h after inoculation. Disease symptoms were recorded on days 10, 14, 18, 22 and 26 after the first spray inoculation. In each plot, the percentage of visually infected spikelets was estimated on a whole-plot basis, according to a linear scale from 0 (no disease) to 4 (100% infected spikelets), as described by Buerstmayr et al. (2000). The area under the disease progress curve (AUDPC) was calculated for each entry as an integrated measure for disease severity, i.e. combined resistance to fungal penetration and spread.

FHB incidence

In 2002, the percentage of diseased heads was assessed in the spray-inoculated experiments by classifying a random sample of 20 heads per plot as diseased or healthy. A head was classified as diseased when at least one spikelet was bleached. The number of diseased heads was counted on days 10, 14, 18, 22 and 26 after the first spray inoculation. An AUDPC was calculated for each plot, presenting disease incidence as a measure for resistance to fungal penetration (type I resistance).

FHB spread

In 2001 and 2002, the DH lines were evaluated for FHB spread within the spike, using the single-spikelet inoculation technique. A needle was dipped in a macroconidial suspension of the *F. graminearum* isolate IFA 65 with a spore concentration of 5×10^5 ml^{-1} and then penetrated through a spikelet slightly above the middle of the spike. Ten heads per plot in experiment 3 were inoculated at anthesis and labelled. On day 22 after inoculation, the number of bleached spikelets was recorded as a measure for disease spread within the head (type II resistance).

Other traits

In each year, plant height was measured in the control plots as the distance from the soil surface to the top of the heads, excluding awns. Date of anthesis was recorded for each plot in the inoculated

experiments and used to calculate the number of days from 1 May to anthesis as a measure of earliness. In 1999 and 2001, the length of the head and the number of spikelets per head were recorded for ten heads of the non-inoculated control and used to calculate the average distance between spikelets, in millimetres, as a measure for spike compactness.

Molecular markers

Variation on the DNA level was estimated using three molecular marker techniques: microsatellites, AFLPs, and RFLPs. RFLPs and a first set of SSRs were carried out on 120 lines, 60 FHB-resistant and 60 FHB-susceptible lines, based on the phenotypic data for FHB in 1999. AFLPs and a second set of SSRs were mapped on 180 lines. Genomic DNA was isolated from young leaves using the cetyltrimethylammonium bromide extraction method described by Hoisington et al. (1994).

Microsatellite analysis was performed using fluorescent fragment detection on a LI-COR 4200 DNA dual-dye sequencing system. For this method, either the forward SSR primer was directly labelled with a fluorochrome (IRD700 or IRD800) or had a M13 tail. In the latter case, as a third primer, a fluorochrome-labelled M13-30 oligonucleotide (5′ CCC AGT CAC GAC GTT G 3′) was added to the PCR reaction. PCR for directly labelled SSRs was done according to Roeder et al. (1998). The reaction mix for M13-tailed SSRs contained: 0.02 μM forward primer (M13-30 sequence at the 5′ end), 0.18 μM M13-30 oligonucleotide (IRD700- or IRD800-labelled), 0.2 μM reverse primer, 0.2 mM each dNTP, 1.5 mM $MgCl_2$, 0.5 units *Taq* polymerase, 1× PCR buffer and 25 ng template DNA for a 10-μl reaction. The PCR programme for M13-tailed primers was: 94 °C for 2 min and then 30 cycles of 94 °C for 1 min, 0.5 °C s^{-1} to 51 °C, 51 °C for 30 s, 0.5 °C s^{-1} to 72 °C and 72 °C for 1 min, followed by 72 °C for 5 min. PCRs were performed on a Primus 384-well thermocycler (MWG Biotech, Germany). More than 200 SSRs were tested for polymorphism between the parental lines, based on the marker information of Roeder et al. (1998), Pestova et al. (2000), Eujayl et al. (2002), Shi et al. (2002) and Song et al. (2002). In addition, the proprietary SSRs Gwm720, Gwm779, Gwm1110 and Gwm1121 mapping to chromosome3A were kindly provided by M. Roeder (IPK Gaters-leben, Germany). The primer sequences are available upon request from IPK Gatersleben.

The AFLP analysis (Vos et al. 1995) was conducted as described by Hartl et al. (1999) and Buerstmayr et al. (2002) using *Mse*I and *Sse*8387I enzymes. In total, 36 AFLP primer combinations with two selective nucleotides on the 3′ end of either primer were applied. The standard list for AFLP primer nomenclature (http://wheat.pw.usda.gov/ggpages/keygeneAFLPs.html) was employed. The AFLP loci were named based on the primer combination followed by a number which referred to the specific polymorphisms. For example, s24m17_2 refers to the second polymorphic band (bands numbered from low to high molecular weight) amplified with the primer combination of *Sse*8387I-TC and *Mse*I-CG. The detection of the AFLP fragments was done on a LI-COR 4200 DNA dual-dye sequencing system.

The RFLP work was performed as described by Hoisington et al. (1994) and Sourdille et al. (1996). Plant genomic DNA of the DH population and the parents was digested using five restriction enzymes: *Hin*dIII, *Eco*RI, *Eco*RV, *Bam*HI and *Xba*I. Fragment detection was done with the non-radioactive digoxigenin system. Sources of the RFLP probes were oat (*Avena sativa* L.) cDNA clones (*cdo*), barley (*Hordeum vulgare* L.) cDNA clones (*bcd*) and wheat genomic DNA clones (*wg*, *ksu*).

Statistical analysis

Field data

The phenotypic data were analysed using the ANOVA, GLM and CORR procedures of SAS/STAT ver. 8.02(SAS Institute 1989). Broad-sense heritabilities were estimated according to Nyquist (1991).

Marker data

Linkage maps were constructed using MapMaker ver. 3.0b for MS-DOS (Lander et al. 1987), assuming the Haldane (1919) mapping function. A logarithm of odds (LOD) threshold of 3 was set for grouping. The most likely marker orders were determined using the MapMaker 'group', 'compare', 'try' and 'ripple' commands.

QTL analysis

QTL analysis was done by one- and multi-factor ANOVAs for line mean values. Furthermore, simple interval mapping and composite interval mapping were carried out using the PLABQTL software (Utz and Melchinger 1996). A QTL was declared significant with LOD $\geq$2.5. For composite interval mapping, cofactor selection was done automatically with a F-to-enter threshold of 12.

Results

FHB resistance

Means of the parental lines and the means and ranges of the DH lines for FHB severity, FHB incidence and FHB spread across the different environments and the broad-sense heritabilities (*H*) are given in Table 1.

Table 1 Means, ranges and broad-sense heritability estimates (*H*) for *Fusarium* head blight (FHB) severity, FHB incidence, FHB spread, plant height, date of anthesis and spike compactness for the parental lines and the doubled-haploid (DH) population. *AUDPC* Area under the disease progress curve

Trait	Year	Frontana	Remus	Population mean	Population range	*H*
FHB severity (AUDPC)	1999	7.6	17.6	13.3	3.1–30.4	
	2001	7.1	25.4	17.2	4.6–46.4	
	2002	12.1	39.8	25.9	8.6–64.5	
	Mean	8.8	26.1	18.0	5.5–36.6	0.77
FHB incidence (AUDPC)	2002	452.5	1237.5	895.4	170.0–1,600.0	
FHB spread[a]	2001	6.3	11.4	9.1	1.9–17.4	
	2002	4.3	9.5	7.6	1.6–14.9	
	Mean	5.3	10.5	8.4	1.8–15.3	0.75
Plant height (cm)	Mean	105.7	84.3	94.7	78.7–110.7	0.92
Date of anthesis[b]	Mean	33.3	34.9	35.1	27.4–45.4	0.94
Spike compactness (mm)[c]	Mean	5.8	4.9	5.4	4.1–6.8	0.90

[a] Number of bleached spikelets on day 22 after inoculation
[b] Number of days from 1 May to anthesis
[c] Distance between the spikelets

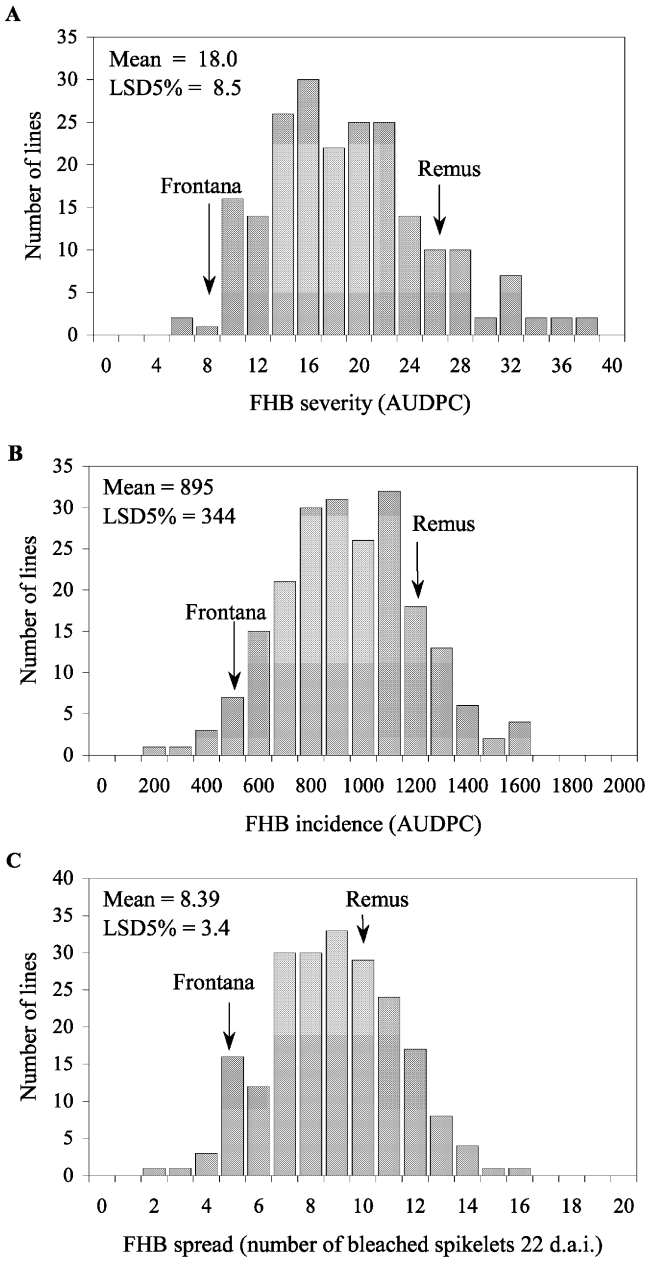

Fig. 1 Histogram of 210 doubled-haploid lines for 3-year mean values of *Fusarium* head blight (FHB) severity measured by the area under the disease progress curve (AUDPC; **A**), FHB incidence in 2002 measured by AUDPC (**B**), and for 2-year mean values for FHB spread 22 days after point inoculation (**C**). Values of the parental lines are indicated by *arrows*. The overall population mean and the least significant difference (*LSD*) are given for comparison of line means (α=0.05), using the genotype-by-year interaction mean square as an error term. *d.a.i.* Days after inoculation

FHB severity

The DH lines showed continuous variation for percentage of diseased spikelets 18–26 days after spray inoculation and for AUDPC. An example frequency distribution for FHB severity measured by AUDPC is illustrated in Fig. 1A. The partially resistant Frontana exhibited an average AUDPC of 8.8 units (25% diseased spikelets 26 days after inoculation), whereas Remus had an AUDPC mean of 26.1 (80% diseased spikelets 26 days after inoculation). ANOVAs were calculated for percentage of infected spikelets observed at each observation date and AUDPC. ANOVA calculations with all possible factors and interactions included in the model revealed no significant genotype-by-isolate and genotype-by-isolate-by-year interactions for any FHB-related trait (data not shown). Therefore, the factor isolates was merged with replications in a simplified model. ANOVA for AUDPC is shown in Table 2. For all FHB-related traits, genotype effects and genotype-by-year interactions were significant. Heritabilities for FHB severity ranged from $H = 0.84$ on day 26 after inoculation to H=0.77 for AUDPC.

FHB incidence

The DH lines showed continuous variation for percentage of diseased spikes after spray inoculation assessed in 2002 (Fig. 1B). Frontana and Remus displayed an average disease incidence of 52% (AUDPC mean of 452) and 100% (AUDPC mean of 1,238) on day 26 after inoculation, respectively (Table 1). ANOVA revealed significant effects for genotypes but no significant genotype-by-isolate interaction (data not shown).

FHB spread

The frequency distribution of the number of infected spikelets 22 days after point inoculation over 2 years is presented in Fig. 1C. ANOVA revealed a significant variation for FHB spread within the head among DH lines (Table 2).

Trait correlations

Correlations between FHB severity, FHB incidence and FHB spread were highly significant for single years and means over years (Table 3). A high coefficient of correlation was revealed for FHB severity and FHB incidence (r=0.73). FHB spread showed weaker associations with FHB severity (r=0.48) and FHB incidence (r=0.35).

Association of resistance to FHB with other traits

Genetic variation within the DH population was highly significant for plant height, date of anthesis and spike compactness. Means, ranges and H values of the parental lines and the mapping population for these traits are given in Table 1. Correlation coefficients between FHB severity, FHB incidence, FHB spread, plant height, date of anthesis and spike compactness are shown in Table 3. A significant negative correlation was found between plant height and FHB-related traits after spray inoculation for means across 3 years. Short genotypes tended to be more diseased. However, the r values were relatively low. FHB spread

Table 2 Analysis of variance for FHB severity and FHB spread

Source	FHB severity[a]				FHB spread[b]			
	df	MS	*F*-value	*P*	*df*	MS	*F*-value	*P*
Replications (years)	11	1,168.84	30.53	<0.0001	2	29.86	7.41	0.0007
Years	2	40,971.04	1,070.33	<0.0001	1	413.70	102.72	<0.0001
Lines	209	554.40	14.48	<0.0001	209	22.95	5.70	<0.0001
Lines × years	417	127.72	3.34	<0.0001	208	5.78	1.43	0.0011
Error	2,296	38.28			417	4.03		

[a] AUDPC across 3 years
[b] Number of infected spikelets on day 22 after point inoculation, across 2 years

Table 3 Phenotypic correlation coefficients among FHB severity, FHB incidence, FHB spread, plant height, date of anthesis and spike compactness. Correlations are for mean values. Consistent associations in all single years under investigation are given in italics. *ns* Not significantly different from zero at P=0.05. Levels of significance: ** P<0.0001, * P< 0.05

Means	FHB incidence	FHB spread	Plant height	Date of anthesis	Spike compactness
	(2002)	(2 years)	(3 years)	(3 years)	(2 years)
FHB severity (3 years)	*0.73***	*0.48***	–0.24*	0.21*	*–0.10*^{ns}
FHB incidence (2002)		*0.35***	–0.19*	0.24*	–0.14*
FHB spread (2 years)			*0.10*^{ns}	*0.50***	–0.14*
Plant height (3 years)				*0.48***	*0.45***
Date of anthesis (3 years)					*–0.06*^{ns}

Table 4 QTL estimates for FHB severity and FHB incidence. QTL analysis was carried out by simple interval mapping. QTL was declared significant at logarithm of odds (LOD)≥2.5. In regions where such loci were also detected, QTLs with LOD≥2 are indicated. Chromosomal location, LOD, percentage of explained phenotypic variance (VE) and the parent contributing to resistance in 1999, 2001, 2002 and across 3 years (measured by AUDPC) are given

Marker, map interval	Chromosome	Source of resistance allele	FHB severity (3 years)		FHB severity (1999)		FHB severity (2001)		FHB severity (2002)		FHB incidence (2002)	
			LOD	VE	LOD	VE	LOD	VE	LOD	VE	LOD	VE
Xs12m25_14–Xs24m17_2	1B	Remus	2.1	5.5					2.5	6.5	2.8	7.2
Xs13m26_4	2A	Remus	2.9	7.9	3.1	8.4	3.0	8.2				
Xs13m25_8–Xs24m15_6	2B	Frontana	2.3	6.1	3.2	8.7					2.3	5.9
Xdupw227–Xgwm720	3A	Frontana	6.6	16.2	4.8	12.0	4.9	12.1	5.2	12.9	4.6	11.5
Xs23m15_3	3B	Remus					2.8	7.2				
Xs13m25_9	4B	Frontana									2.8	7.4
Xgwm129–Xbarc197	5A	Frontana	3.5	8.8	3.6	9	2.1	5.4	3.1	7.8		
Xs23m20_8	5A	Frontana			2.5	6.5						
Xs23m14_4	6B	Frontana	2.5	6.7	2.5	6.6			2.7	5.8	2.0	5.5
Simultaneous fit				43.4		37.2		28.4		27.5		27.3

within the head showed no significant association with plant height. All FHB-related traits displayed positive correlation with date of anthesis; and late flowering date was associated with higher FHB susceptibility. Correlation analysis for FHB severity and spike compactness revealed no significant associations between these traits. FHB incidence and FHB spread displayed significantly negative but low correlations with spike compactness.

Molecular map

The genetic linkage map was constructed using 568 markers composed as follows: two morphological markers (awnedness, glume colour), 119 SSRs, 415 AFLPs and 32 RFLPs. Of these, 535 markers could be mapped to 42 linkage groups, covering a genetic distance of 2,840 cM. For 27 linkage groups, their chromosomal identity could be determined by comparison with the microsatellite map of wheat published by Roeder et al. (1998). For all wheat chromosomes, apart from 4D and 6D, at least partial maps were obtained.

Quantitative trait mapping of FHB

FHB severity

Simple interval mapping with PLABQTL revealed associations of several genomic regions with low FHB severity measured by AUDPC (LOD ≥2.5; Table 4). QTLs were detected on chromosomes 1B, 2A, 2B, 3A, 3B, 5A and 6B. QTLs on chromosomes 3A and 5A were consistent over all 3 years, with resistance conferred by

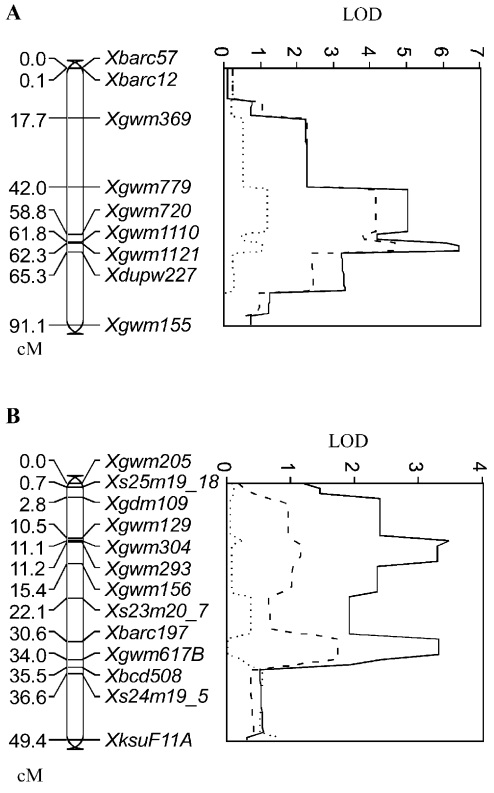

Fig. 2 Interval analysis of QTLs for FHB severity (AUDPC, means over 3 years, *solid line*), FHB incidence (AUDPC, 2002, *dashed line*) and FHB spread (number of infected spikelets on day 22 after inoculation, means over 2 years, *dotted line*) on linkage groups corresponding to parts of chromosomes 3A (**A**) and 5A (**B**). Logarithm of odds (*LOD*) values were calculated by simple interval mapping

alleles of Frontana. The major effect associated with FHB severity mapped to chromosome 3A near the centromere, explaining 16% of the phenotypic variation for FHB severity mean values over 3 years. In single years, this QTL accounted for 12–13% of the variation. The most likely position is in the marker interval *Xgwm720–Xdupw227* (Fig. 2A). The QTL was estimated to be in the same region with both simple and composite interval mapping (data not shown). The map interval *Xgwm129–Xbarc197* on chromosome 5A also showed an association with FHB severity over 3 years and accounted for 9% of the phenotypic variation. The LOD curve calculated by simple interval mapping across this region indicated two closely linked resistance QTLs (Fig. 2B), whereas with composite interval mapping just one QTL linked to *Xgwm129* was detected, suggesting a single QTL for FHB severity in this chromosomal region.

Further but smaller QTLs for FHB severity were detected on chromosomes 1B, 2A, 2B, 3B, 5AL and 6B in single years (Table 4). For the QTLs on chromosomes 1B, 2A and 3B, Remus contributed the alleles for resistance. The total phenotypic variance explained ranged from 28% to 37%, depending on the year. Means over three years showed an adjusted R^2 of 43% (Table 4).

Table 5 Effect of alternative alleles at two QTL regions for FHB severity, measured by AUDPC for line means over 3 years. Mean values followed by the same letter are not significantly different at $P<0.05$

QTL		Number of lines	FHB severity mean
3A *Xdupw227*	5A *Xgwm129*		
Frontana	Frontana	53	14.3a
Frontana	Remus	53	18.6b
Remus	Frontana	32	17.4b
Remus	Remus	36	23.1c

The association of the two most prominent QTLs on chromosomes 3A and 5A with the phenotype is illustrated in Table 5. Lines with the 'resistant' alleles from Frontana at both QTL regions had an AUDPC mean of 14.3. The lines with the alleles from the susceptible Remus exhibited an AUDPC mean of 23.1. Lines with both Frontana alleles at the 3A and 5A QTL regions had on average 40% less disease severity than lines with Remus alleles at both QTL positions.

FHB incidence

QTL analysis of FHB incidence detected five QTLs on chromosomes 1B, 2B, 3A, 4B and 6B (Table 4). The major QTL associated with low FHB incidence was located on chromosome 3A, overlapping with the most prominent QTL for FHB severity (Fig. 2A). This QTL explained 11.5% of the phenotypic variance. Other QTLs on chromosomes 1B, 2B and 6B also coincided with QTLs for FHB severity. QTL analysis for FHB incidence was based on a single year and the total phenotypic variance explained was 27.3% (Table 4).

FHB spread

QTL detection with simple interval mapping of FHB spread resulted in six minor QTLs. Two distinct QTLs derived from Frontana mapped to chromosome 2B and four QTLs were derived from Remus on chromosomes 2A, 5A, 6A and one unassigned linkage group (Table 6). Only two QTLs, one 'Frontana effect' on chromosome 2B and one 'Remus effect' on an unassigned linkage group were consistently detected in both years. Total phenotypic variance explained was 25.1% in 2001 and 20.7% in 2002 (Table 6). The major QTL for FHB severity and FHB incidence on chromosome 3A was not associated with FHB spread within the spike (Fig. 2A). Composite interval mapping with PLABQTL gave similar results, with no additional QTLs detected (data not shown).

Table 6 QTL estimates for FHB spread. QTL analysis was carried out by simple interval mapping. QTL was declared significant at LOD≥2.5. In regions where such loci were also detected, QTLs at LOD≥2 are indicated. Chromosomal location, LOD, VE and the parent contributing to resistance in 2001, 2002 and across 2 years for number of infected spikelets on day 22 are indicated. *ND* Chromosomal location not determined

Marker, map interval	Chromosome	Source of resistance allele	FHB spread (2 years)		FHB spread (2001)		FHB spread (2002)	
			LOD	VE	LOD	VE	LOD	VE
Xs13m26_4–Xs12m15_7	2A	Remus	2.5	6.5			2.4	6.1
Xs25m15_2–Xs24m19_11	2B	Frontana	3.0	8.1	2.8	7.4	3.2	8.7
Xs13m25_8–Xs24m15_6	2B	Frontana					3.0	7.9
Xs12m25_9	5A	Remus			2.7	7.3		
Xgwm169	6A	Remus	2.9	7.6	2.9	7.5		
Xs18m15_6–Xs12m15_5	ND	Remus	3.4	9.4	3.2	9.0	2.7	6.9
Simultaneous fit				23.1		25.1		20.7

Table 7 QTL estimates for plant height, date of anthesis and spike compactness. Chromosomal locations, LOD, VE and the estimated additive effect (*Add*) of Frontana alleles in conferring greater plant height, earlier anthesis or a laxer spike across 2 or 3 years. QTLs with consistent effects over all years investigated are shown in italics. *ND* Chromosomal location not determined

Trait	Chromosome	Linked marker	LOD	VE	Add
Plant height (cm; 3 years)	2B	*Xgwm972*	2.9	7.4	3.7
	4B	*Xs13m25_9*	*6.1*	*15.2*	*5.1*
	5A	*Xbarc197*	5.0	12.3	4.8
	7D	*Xgwm44*	*7.4*	*18.3*	*5.7*
	Simultaneous fit			48.0	
Date of anthesis (number of days from 1 May; 3 years)	2D	*Xs25m19_16*	*3.8*	*14.7*	*−2.6*
	2D	*Xgwm261*	*5.1*	*13.0*	*−2.7*
	7D	*Xs24m20_10*	*7.6*	*18.5*	*3.2*
	Simultaneous fit			36.8	
Spike compactness (mm; 2 years)	1A	*Xs12m19_15*	*4.2*	*11.2*	*−0.3*
	7A	*Xs12m19_1*	*2.7*	*6.8*	*0.3*
	ND	*Xs18m15_6*	3.6	10.0	−0.3
	Simultaneous fit			19.5	

Quantitative trait mapping of other traits

QTLs with simple interval mapping with LOD ≥2.5 for plant height, date of anthesis and spike compactness for means over years are given in Table 7. Plant height was controlled by four QTLs located on chromosomes 2B, 4B, 5A and 7D. For all loci, Frontana alleles contributed to higher plant height. QTL detection for date of anthesis revealed effects on chromosomes 2D and 7D. Distance between spikelets was controlled by three loci: two QTLs on chromosome 1A and 7A were detected in both years and the other QTL was located on an unassigned linkage group.

Co-localization of QTLs for FHB severity and plant height was found on chromosome 5A near to the marker *Xbarc197*. A FHB incidence QTL on chromosome 4B was overlapping with a QTL for plant height. Further, the AFLP marker *Xs18m15_6* (on an unassigned linkage group) showed an association with resistance to disease spread within the spike and spike compactness.

Discussion

FHB resistance assessment

The detection of QTLs that affect any trait depends largely on precise phenotypic assessments. Evaluation of FHB resistance is time-consuming, laborious and costly, because of the quantitative nature of the resistance and because phenotypic expression is affected by environmental factors. In this study, artificial inoculations conducted individually for each genotype at anthesis with two single-spore macroconidial suspensions led to the development of FHB on all genotypes under investigation. A mist irrigation system and repeated disease assessment for each entry within 3 years with several replications each contributed to precise estimation of the resistance level of all lines. Two inoculation techniques were applied to distinguish between FHB-resistance components. FHB severity was estimated by spray inoculation and assessment of the percentage of infected spikelets on a whole-plot basis. This approach accounts for most of the relevant factors that may contribute to reduced disease symptoms in a natural epidemic. To separate between resistance to fungal penetration and resistance to fungal spread, the percentage of diseased spikes was evaluated as a measure for type I resistance. Type II resistance was assessed by inoculating a single central spikelet of a head and measuring the progression of disease symptoms.

Variation of FHB symptoms was quantitative for both inoculation techniques and all FHB-related traits; and more phenotypic variation was observed after spray inoculation than after point inoculation. Significant transgressive segregation towards susceptibility was found for FHB severity and FHB spread. Despite the significant

genotype-by-year interaction for FHB-related traits, broad-sense heritabilities were high (H=0.75–0.84), indicating a reproducible assessment of the FHB resistance level. FHB incidence and FHB severity were highly correlated, indicating that low FHB incidence and low FHB severity are under similar genetic control, which is in agreement with Groth et al. (1999). However, FHB spread showed moderate to low association with other FHB-related traits.

The non-significance of the genotype-by-isolate interaction supports the horizontal nature of FHB resistance in wheat (Van Eeuwijk et al. 1995).

QTL mapping for FHB resistance

Simple interval mapping revealed associations of several genomic regions with resistance to FHB. The major QTL associated with FHB resistance mapped to chromosome 3A near the centromere, explaining 16% of the phenotypic variation for FHB severity. The genomic region on 3A was significantly linked with all FHB-related traits after spray inoculation (percentage of infected spikelets, percentage of infected spikes), but not with FHB spread within the spike, indicating the prominent contribution of this chromosome region to type I resistance (resistance to initial infection). The map interval *Xgwm129–Xbarc197* on chromosome 5A was also consistently associated with FHB severity only. Whether there are two closely linked QTLs or a single resistance gene located on this chromosome could not be determined.

Wheat chromosomes 3A and 5A have been reported to carry QTLs for FHB resistance. Otto et al. (2002) detected in *T. dicoccoides* a QTL near the centromere of 3AS; and the SSR marker *Xgwm2* was closely associated with the resistance QTL, *Qfhs.ndsu-3AS*. Using bulked segregant analysis, Shen et al. (2003b) identified a QTL in the same chromosomal region in the Romanian winter wheat cultivar Fundulea 201R, linked to *Xgwm674*. Furthermore, Bourdoncle and Ohm (2003) detected a minor resistance QTL associated with *Xgwm5* close to the centromere of chromosome 3A in the Chinese cultivar Huapei 57-2. Unfortunately, none of these SSR markers revealed polymorphisms in our mapping population. Our results indicate that the 3A QTL of Frontana is positioned in the proximal region of 3AL, whereas *Qfhs.ndsu-3AS* maps to 3AS. In addition, *Qfhs.ndsu-3AS* was detected by point inoculation and the 3A QTL of Frontana was not. We therefore hypothesise that the 3A QTL of Frontana is not allelic to *Qfhs.ndsu-3AS*. QTLs in the *Xgwm129–Xbarc197* interval on chromosome 5A were reported by Buerstmayr et al. (2002, 2003) and Shen et al. (2003b). Furthermore, Somers et al. (2003) detected in the wheat cultivar Maringa (Frontana/Kenya58//Pgi) a QTL controlling DON accumulation in the same 5A chromosomal region. The minor QTL on chromosome 3B detected in our study in 2001 only cannot be *Qfhs.ndsu-3BS* (Anderson et al. 2001), because the allele conferring reduced FHB severity originates from the susceptible parent Remus, which is not related to Sumai-3 (Buerstmayr et al. 2002).

Individual resistance QTLs for spread within the spike accounted for less than 10% of the variation in trait expression. Both parents contributed QTLs for type II resistance. These results suggest that Frontana does not carry any QTL alleles with major effects for resistance to fungal spread. In contrast, in genotypes of Asian origin, resistance to fungal spread seems to be of major importance (Anderson et al. 2001; Buerstmayr et al. 2002, 2003; Zhou et al. 2002; Bourdoncle and Ohm 2003; Shen et al. 2003a).

In the present work, QTL stability over years was moderate. Despite the use of DH progeny and replicated experiments at the same location, out of the 13 QTLs detected, just four QTLs were consistently identified in all years evaluated. The best multiple regression model accounted for 43% of the observed phenotypic variation. The unexplained variation probably results from a combination of undetected genes, epistasis and experimental error.

In this study, two-way interactions between loci revealed significant marker interactions for all FHB-related traits. Additive-by-additive epistatic QTL interactions are probably important for FHB resistance in this population. However, precise estimation of these interactions would require a larger population size, which could also increase the power of QTL main-effect detection.

QTL detection of other traits and association with FHB resistance

Most of the agronomic and morphological traits associated with FHB resistance potentially limit the number of ascospores/conidia reaching infection sites on heads, or otherwise reduce the likelihood of successful entry into head tissues. Generally, short-statured, awned genotypes with a short peduncle and a compact spike are more susceptible to FHB than tall lines that are awnless and have a long peduncle and a lax head (Mesterhazy 1995; Parry et al. 1995; Hilton et al. 1999; Rudd et al. 2001). If a trait correlated with resistance is undesirable (tall, lodging-susceptible plants), then it is necessary to determine whether the correlation is due to pleiotropy or linkage. Co-localisations between QTLs of resistance to FHB, plant height and earliness were found in wheat (Gervais et al. 2003) and barley (De la Pena et al. 1999; Zhu et al. 1999; Ma et al. 2000; Mesfin et al. 2003). Furthermore, in barley, FHB QTLs coincident with characters of the spike architecture were identified (Zhu et al. 1999; Ma et al. 2000; Mesfin et al. 2003).

In this study, the possible contributions of plant height, date of anthesis and spike compactness to FHB were investigated.

Plant height and FHB resistance

Plant height was significantly correlated with FHB incidence and severity. Taller lines tended to be less diseased than shorter ones. This phenomenon appears to be a common feature reported in several studies (Mesterhazy 1995; Hilton et al. 1999; Buerstmayr et al. 2000). QTL detection for plant height in this population revealed four QTLs, located on chromosomes 2B, 4B, 5A and 7D. A FHB incidence QTL on chromosome 4B was coincident with a QTL for plant height possibly corresponding to the *Rht-B1* gene (McIntosh et al. 1998). Furthermore, the QTL for plant height on chromosome 5A near to *Xbarc197* overlapped with a QTL for FHB severity. Similar results were obtained by Gervais et al. (2003), who identified co-localisation between QTLs for FHB resistance and plant height on chromosome 5A. These coincident QTLs in the Frontana mapping population on chromosomes 4B and 5A may be a contributing factor to the moderate correlation between these traits but there are mostly independent genes affecting FHB resistance and plant height.

Date of anthesis and FHB resistance

Since Arthur (1891), many authors have reported the apparent resistance of early-maturing cultivars. To minimise the confounding effect of flowering date, the lines were individually inoculated at anthesis. Nevertheless, early-flowering lines exhibited significantly less FHB than late-flowering lines.

QTLs for flowering time were detected in regions of the genome known to carry major genes for photoperiod and vernalisation response in wheat (McIntosh et al. 1998). In this study, QTL mapping revealed no co-localisation of QTLs for FHB resistance and date of anthesis. Mainly environmental effects were involved in the trait associations of FHB resistance and earliness.

Spike compactness and FHB resistance

Generally, plants having a dense head tend to be more susceptible to spike diseases because of micro-climatic conditions (Mesterhazy 1995). Within the mapping population evaluated here, FHB spread within the spike and FHB incidence displayed moderately negative correlations with spike compactness. The distance between the spikelets was controlled by several loci with small effects; and mainly unlinked genes affected FHB resistance and spike compactness.

Conclusions

Resistance to FHB of Frontana is complex, as indicated by a considerable number of genomic regions significantly associated with FHB severity, FHB incidence and FHB spread. The results indicate that the FHB resistance of Frontana is primarily based on resistance to initial infection, whereas resistance to fungal spread within the spike seems to be of minor importance. The types of resistance are controlled partly by independent loci. QTLs on chromosomes 3A and 5A appear to be essential for the expression of FHB resistance in Frontana. Part of the variation in FHB reaction may also be due to extended plant height, earliness, or a lax spike, which could be passive resistance factors. However, the correlations were low and co-localisation of QTLs was observed for minor QTLs only. There are independent genes affecting FHB resistance that should allow plant breeders to select resistant lines of any height, flowering date and spike morphology within a Frontana cross. The major resistance gene in Frontana on chromosome 3A revealed no association with plant height, earliness and spike compactness. SSR markers around the 3A and 5A QTLs were identified and can be used in marker-assisted selection, e.g. for combining the FHB resistance QTLs of Frontana and Asian wheats.

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References

Anderson JA, Stack RW, Liu S, Waldron BL, Fjeld AD, Coyne C, Moreno-Sevilla B, Mitchell Fetch J, Song QJ, Cregan PB, Frohberg RC (2001) DNA markers for *Fusarium* head blight resistance QTLs in two wheat populations. Theor Appl Genet 102:1164–1168

Arthur JC (1891) Wheat scab. Indiana Agric Exp St Bull 36:129–132

Bourdoncle W, Ohm HW (2003) Quantitative trait loci for resistance to *Fusarium* head blight in recombinant inbred wheat lines from the cross Huapei 57-2/Patterson. Euphytica 131:131–136

Buerstmayr H, Lemmens M, Grausgruber H, Ruckenbauer P (1996) Scab resistance of international wheat germplasm. Cereal Res Commun 24:195–202

Buerstmayr H, Steiner B, Lemmens M, Ruckenbauer P (2000) Resistance to *Fusarium* head blight in two winter wheat crosses: heritability and trait associations. Crop Sci 40:1012–1018

Buerstmayr H, Lemmens M, Hartl L, Doldi L, Steiner B, Stierschneider M, Ruckenbauer P (2002) Molecular mapping of QTLs for *Fusarium* head blight resistance in spring wheat. I. Resistance to fungal spread (type II resistance). Theor Appl Genet 104:84–91

Buerstmayr H, Steiner B, Hartl L, Griesser M, Angerer N, Lengauer D, Miedaner T, Schneider B, Lemmens M (2003) Molecular mapping of QTLs for *Fusarium* head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor Appl Genet 107:503–508

De La Pena RC, Smith KP, Capettini F, Muehlbauer GJ, Gallo-Meagher M, Dill-Macky R, Somers DA, Rasmusson DC (1999) Quantitative trait loci association with resistance to *Fusarium* head blight and kernel discoloration in barley. Theor Appl Genet 99:561–569

Eujayl I, Sorrells ME, Baum M, Wolters P, Powell W (2002) Isolation of EST-derived microsatellite markers for genotyping the A and B genome of wheat. Theor Appl Genet 104:399–407

Gervais L, Dedryver F, Morlais JY, Bodusseau V, Negre S, Bilous M, Groos C, Trottet M (2003) Mapping of quantitative trait loci for field resistance to *Fusarium* head blight in an European winter wheat. Theor Appl Genet 106:961–970

Groth JV, Ozmon EA, Busch RH (1999) Repeatability and relationship of incidence and severity measures of scab of wheat caused by *Fusarium graminearum* in inoculated nurseries. Plant Dis 83:1033–1038

Haldane JBS (1919) The combination of linkage values, and the calculation of distances between loci of linked factors. J Genet 8:299–309

Hartl L, Mohler V, Zeller FJ, Hsam SLK, Schweizer G (1999) Identification of AFLP markers closely linked to the powdery mildew resistance genes *Pm1c* and *Pm4a* in common wheat. Genome 42:322–329

Hilton AJ, Jenkinson P, Hollins TW, Parry DW (1999) Relationship between cultivar height and severity of *Fusarium* ear blight in wheat. Plant Pathol 48:202–208

Hoisington D, Khairallah M, Gonzalez-de-Leon D (1994) Laboratory protocols: CIMMYT applied molecular genetics laboratory, 2nd edn. CIMMYT, Mexico City

Kolb FL, Bai GH, Muehlbauer GJ, Anderson JA, Smith KP, Fedak G (2001) Host and plant resistance genes for *Fusarium* head blight: mapping and manipulation with molecular markers. Crop Sci: 41:611–619

Lander ES, Green P, Abrahamson J, Barlow A, Daly MJ, Lincoln SE, Newburg L (1987) MAPMAKER: An interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics 1:174–181

Laurie DA, Bennett MD (1988) The production of haploid wheat plants from wheat x maize crosses. Theor Appl Genet 76:393–397

Ma Z, Steffenson BJ, Prom LK, Lapitan NLV (2000) Mapping of quantitative trait loci for *Fusarium* head blight resistance in barley. Phytopathology 90:1079–1088

McIntosh RA, Hart GE, Devos KM, Gale MD, Rogers WJ (1998) Catalogue of gene symbols for wheat. In: Slinkard AE (ed) Proc 9th Int wheat Genet Symp, vol 5. University Extension Press, Saskatchewan

Mesfin A, Smith KP, Dill-Macky R, Evans CK, Waugh R, Gustus CD, Muehlbauer GJ (2003) Quantitative trait loci for *Fusarium* head blight resistance in barley detected in a two-rowed by six-rowed population. Crop Sci 43:307–318

Mesterhazy A (1995) Types and components of resistance to *Fusarium* head blight of wheat. Plant Breed 114:377–386

Miller JD, Arnison PG (1986) Degradation of deoxynivalenol by suspension cultures of the *Fusarium* head blight resistant wheat cultivar Frontana. Can J Plant Pathol 8:147–150

Nyquist WE (1991) Estimation of heritability and prediction of selection response in plant populations. Crit Rev Plant Sci 10:235–322

Otto CD, Kianian SF, Elias EM, Stack RW, Joppa LR (2002) Genetic dissection of a major *Fusarium* head blight QTL in tetraploid wheat. Plant Mol Biol 48:625–632

Parry DW, Jenkinson P, McLeod L (1995) *Fusarium* ear blight (scab) in small grain cereals—a review. Plant Pathol 44:207–238

Pestova E, Ganal MW, Roeder MS (2000) Isolation and mapping of microsatellite markers specific for the D genome of bread wheat. Genome 43:689–697

Roeder MS, Korzun V, Wendehake K, Plaschke J, Tixier MH, Leroy P, Ganal MW (1998) A microsatellite map of wheat. Genetics 149:2007–2023

Rudd JC, Horsley RD, McKendry AL, Elias EM (2001) Host plant resistance genes for *Fusarium* head blight: sources, mechanisms and utility in conventional breeding systems. Crop Sci 41:620–627

SAS Institute (1989) SAS/STAT user's guide, ver. 8.02. SAS Institute, Cary, N.C.

Schroeder HW, Christensen JJ (1963) Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831–838

Shen X, Zhou M, Lu W, Ohm H (2003a) Detection of *Fusarium* head blight resistance QTL in a wheat population using bulked segregant analysis. Theor Appl Genet 106:1041–1047

Shen X, Ittu M, Ohm H (2003b) Quatitative trait loci conditioning resistance to *Fusarium* head blight in wheat line F201R. Crop Sci 43:850–857

Shi JR, Song QJ, Singh S, Ward RW, Cregan PB, Gill BS (2002) Microsatellite genetic map in wheat. (National *Fusarium* head blight forum, 7–9 Dec. 2002) USDA, Cincinnati http://wheat.pw.usda.gov/ggpages/genomics.shtml

Singh RP, Ma H, Rajaram S (1995) Genetic analysis of resistance to scab in spring wheat cultivar Frontana. Plant Dis 79:238–240

Somers DJ, Fedak G, Savard M (2003) Molecular mapping of novel genes controlling *Fusarium* head blight resistance and deoxynivalenol accumulation in spring wheat. Genome 46:555–564

Song QJ, Fickus EW, Cregan PB (2002) Characterization of trinucleotide SSR motifs in wheat. Theor Appl Genet 104:286–293

Sourdille P, Perretant MR, Charmet G, Leroy P, Gautier MF, Joudrier P, Nelson JC, Sorrells ME, Bernard M (1996) Linkage between RFLP markers and genes affecting kernel hardness in wheat. Theor Appl Genet 93:580–586

Utz HF, Melchinger AE (1996) PLABQTL: a program for composite interval mapping of QTL. J Agric Genomics http://www.ncgr.org/research/jag/papers96/paper196/indexp196.html

Van Eeuwijk FA, Mesterhazy A, Kling CI, Ruckenbauer P, Saur L, Buerstmayr H, Lemmens M, Keizer LCP, Maurin N, Snijders CHA (1995) Assessing non-specificity of resistance in wheat to head blight caused by inoculation with European strains of *Fusarium culmorum*, *F. graminearum* and *F. nivale* using a multiplicative model for interaction. Theor Appl Genet 90:221–228

Van Ginkel M, Van Der Schaar W, Zhuping Y, Rajaram S (1996) Inheritance of resistance to scab in two wheat cultivars from Brazil and China. Plant Dis 80:863–867

Vos P, Hogers R, Bleeker M, Reijans M, van de Lee T, Horens M, Frijters A, Pot J, Peleman J, Kuiper M, Zabeau M (1995) AFLP: a new technique for DNA fingerprinting. Nucleic Acids Res 23:4407–4414

Waldron BL, Moreno-Sevilla B, Anderson JA, Stack RW, Frohberg RC (1999) RFLP mapping of QTL for *Fusarium* head blight resistance in wheat. Crop Sci 39:805–811

Wang YZ, Miller JD (1988) Effects of *Fusarium graminearum* metabolites on wheat tissue in relation to *Fusarium* head blight resistance. J Phytopathol 122:118–125

Zhou WC, Kolb FL, Bai GH, Shaner G, Domier LL (2002) Genetic analysis of scab resistance QTL in wheat with microsatellite and AFLP markers. Genome 45:719–727

Zhu H, Gilchrist L, Hayes P, Kleinhofs A, Kundra D, Liu Z, Prom L, Steffenson B, Toojinda T, Vivar H (1999) Does function follow form? Principal QTLs for *Fusarium* head blight (FHB) resistance are coincident with QTLs for inflorescence traits and plant height in a doubled-haploid population of barley. Theor Appl Genet 99:1221–1232