

## QTL analysis of resistance to *Fusarium* head blight in wheat using a 'Frontana'-derived population

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### Abstract

*Fusarium* head blight (FHB or head scab) has become a major limiting factor for sustainable wheat (*Triticum aestivum* L.) production around the world. For quantitative trait loci (QTL) analysis of resistance to FHB, F<sub>3</sub> plants and F<sub>3:5</sub> lines, derived from a 'Frontana' (moderately resistant)/'Seri82' (susceptible) cross, were spray-inoculated in 2001 and 2002, respectively. Artificial inoculations were carried out under field conditions. Of 273 SSR and AFLP markers, 250 could be mapped and they yielded 42 linkage groups, covering a genetic distance of 1931 cM. QTL analysis was based on the constructed linkage map and area under the disease progress curve (AUDPC). The analyses revealed three consistent QTLs associated with FHB resistance on chromosomes 1BL, 3AL and 7AS explaining 7.9%, 7.7% and 7.6% of the phenotypic variation, respectively, above 2 years. The results confirmed the previously described resistance QTL of 'Frontana' on chromosome 3AL. A combination of 'Frontana' resistance with 'Sumai-3' resistance may lead to lines with augmented resistance expression.

**Key words:** *Triticum aestivum* — *Fusarium graminearum* — QTL mapping — AFLP — SSR

*Fusarium* head blight (FHB, scab), predominantly caused by *Fusarium graminearum* and *F. culmorum*, has become a major limiting factor for sustainable wheat (*Triticum aestivum* L.) production around the world. This destructive disease can cause serious grain yield losses. In addition, grain contaminated with mycotoxins, associated with FHB, reduces its marketability for use as a food or feed (Bai and Shaner 1994). FHB-resistant varieties are the best approach for controlling FHB (Parry et al. 1995). Schroeder and Christensen (1963) described two types of FHB resistance: type I (resistance against initial infection) and type II (resistance to the spread of the pathogen within a spike). Although diverse sources of resistance to FHB are available in wheat, breeding for FHB resistance is tedious due to the polygenic nature of FHB resistance, undesirable agronomic traits associated with resistance sources, and environmental influences on disease expression (Miedaner 1997, Buerstmayr et al. 2002).

Identifying DNA markers linked to genes governing resistance to FHB provides an additional tool for breeding programmes (Gupta et al. 1999). FHB-resistant Chinese germplasm and derivatives have been studied extensively using quantitative trait loci (QTL) mapping (Bai et al. 1999, Waldron et al. 1999, Anderson et al. 2001, Buerstmayr et al. 2002, 2003, Zhou et al. 2002, Bourdoncle and Ohm 2003, Shen et al.

2003, Lin et al. 2004, Mardi et al. 2005). Reports on QTL mapping of FHB resistance in wheat using other sources of resistance have also been published (Otto et al. 2002, Gervais et al. 2003, Shen et al. 2003, Somers et al. 2003, Steiner et al. 2004). QTL mapping results need to be independently validated before one can recommend their application in cultivar development. The major QTL, *Qfhs.ndsu-3BS*, from 'Sumai-3' or its derivatives was validated in several studies (Waldron et al. 1999, Anderson et al. 2001, Buerstmayr et al. 2002, 2003, Zhou et al. 2002). Steiner et al. (2004) performed an extensive QTL mapping study in a 'Frontana'/'Remus'-derived, doubled haploid population. The aim of this study was to map the QTLs in an independently derived 'Frontana'/'Seri82' population in order to assess the genetic background dependence and stability of 'Frontana'-derived FHB resistance. The ultimate goal was to identify molecular markers linked to Frontana's FHB resistance QTLs that have a potential for use in cultivar development.

### Materials and Methods

**Plant materials:** Studies were made on 171 F<sub>3</sub> plants of wheat, *Triticum aestivum* L., one from each F<sub>2</sub> individual, and 120 F<sub>3:5</sub> lines, derived from individual F<sub>3</sub> plants, from the cross 'Frontana'/'Seri82' (syn. 'Falat'). Seeds of the parental lines were obtained from CIMMYT. 'Frontana' has the pedigree 'Fronteira'/'Mentana' and is known for its moderate resistance to FHB (Schroeder and Christensen 1963, Singh et al. 1995, Buerstmayr et al. 1996, Van Ginkel et al. 1996, Steiner et al. 2004). The FHB-susceptible cultivar 'Seri82' originates from the CIMMYT spring wheat-breeding programme. 'Seri82' carries the T1BL.1RS translocation chromosome (Singh et al. 1998), its pedigree is 'Kavkaz'/'Buho-sib'/'Kalyansona'/'Bluebird'. Lines of the population, together with the parents, were evaluated for FHB severity in field experiments at the Department for Agrobiotechnology Tulln (IFA-Tulln), Austria, during 2001 and 2002. In 2001, 171 space-planted F<sub>3</sub> plants were evaluated in a non-replicated trial with 25 cm distances between the plants. Five-10 seeds from each single F<sub>3</sub> plant were multiplied in a greenhouse during winter 2001/2002. In 2002, 120 F<sub>3:5</sub> lines were sown in a randomized complete block design with two blocks. Each plot consisted of two rows (1 m/row) planted with 2 g seeds/row and 17 cm row spacing.

**Inoculation procedures and disease assessment:** The inoculation of plants with *F. graminearum* was carried out using the spray inoculation method. IFA65, a pathogenic strain of *F. graminearum* was used for

artificial inoculation as described by Mardi et al. (2005). To provide humidity for the disease development, the sprayed heads were bagged with polyethylene bags or irrigated with a mist irrigation system, in the 2001 and 2002 trials, respectively. Disease symptoms were assessed on three heads of each F<sub>3</sub> plant (2001) and by visually averaging whole plots (2002), respectively. The percentage of infected spikelets was visually estimated 14, 18, 22, and 26 days after inoculation on single heads in 2001 and on a whole plot basis in 2002. The area under the disease progress curve (AUDPC) was calculated for each inoculated spike in 2001 and each plot in 2002 using the following formula:

$$\text{AUDPC} = \sum_{i=1}^n \left( \frac{y_i + y_{i-1}}{2} \right) (x_i - x_{i-1})$$

where  $y_i$  is the percentage of visibly infected spikelets ( $y_i/100$ ) at the  $i$ th observation and  $x_i$  is the day of the  $i$ th observation, and  $n$  indicates the total number of observations (modified from Shaner and Finney 1977).

**Genomic DNA extraction:** Healthy leaves harvested from the parents and 171 F<sub>3</sub> plants were used for DNA extraction. Genomic DNA was isolated using the CTAB method (Saghai-Marouf et al. 1984). DNA quantity and quality were measured with a UV-photometer.

**AFLP and SSR markers:** The AFLP analysis was performed as described by Vos et al. (1995) using the enzyme combination *EcoRI* and *MseI*. Twenty-five *EcoRI* + NNN/*MseI* + NNN primer combinations with three selective nucleotides on the 3'-end of either primer were used for selective PCR amplification. The AFLP loci names were abbreviated according to the standard nomenclature of AFLPs (<http://wheat.pw.usda.gov/ggpages/keygeneAFLPs.html>). The SSR markers were used as landmarks to locate AFLP markers in the genetic map. One hundred and seventy SSR primer pairs (Röder et al. 1998) were used to assay parental polymorphism. PCRs were performed on a Bio-Rad thermocycler (Bio-Rad Laboratories Inc., Hercules, CA, USA). Amplification reaction products were separated on 6% denaturing polyacrylamide gels using a Sequi-Gen GT Sequencing Cell 30 cm gel apparatus (Bio-Rad Laboratories Inc.). The amplified fragments were detected by the silver staining method as described by Bassam et al. (1991). The resulting gels were scored visually and independently by two people.

**Analysis of phenotypic variation:** The analysis of variance was performed for data obtained from the 120 F<sub>3:5</sub> lines evaluated in 2002 using the general liner model (GLM) procedure of the SAS/STAT software (SAS Institute Inc 1990). In order to estimate the reproducibility of the disease evaluations, the Pearson correlation coefficient was calculated for mean FHB severity (AUDPC) between single F<sub>3</sub> plants evaluated in 2001 and their F<sub>3:5</sub> lines evaluated in 2002.

**Construction of the linkage map:** Segregating AFLP markers were scored for each individual of the mapping population with reference to parental genotypes. Scoring for polymorphic SSR markers was carried out as homozygous or heterozygous loci. A linkage map was constructed using the 'group', 'order', 'ripple' and 'try' commands of MAPMAKER 3.0b (Lander et al. 1987). Polymorphic AFLP markers were assigned to linkage groups by reference to linked SSR markers with known chromosomal locations (Röder et al. 1998). A minimum logarithm of the odds ratio (LOD) score of three and a maximum genetic distance of 30 cM were used for pair-wise linkage analysis. The Kosambi mapping function (Kosambi 1944) was used to convert recombination frequencies into genetic distances.

**QTL analysis:** The QTL analysis was performed using genotypic data from the 171 F<sub>3</sub> plants and the phenotypic data obtained from the same F<sub>3</sub> plants in 2001, the 120 F<sub>3:5</sub> lines evaluated in 2002 and for the mean values of 120 entries analysed in both years. Interval mapping was conducted with the PLABQTL software (Utz and Melchinger 1996). Simple interval mapping (SIM) was performed using a stepwise regression analysis with the additive genetic model

(Zeng 1994). The location of a QTL is defined as the position where the LOD score value exceeds two.

## Results

### FHB resistance

The difference in FHB severity between the parental genotypes at the end of the observation period ranged from 42% in 'Frontana' to 80% in 'Seri82'. A highly significant correlation coefficient was observed between the 2 years for AUDPC means ( $r = 0.70$ ;  $P < 0.0001$ ). The distribution of AUDPC mean values of the 120 lines evaluated in both years are shown in Fig. 1. The analysis of variance revealed a highly significant influence of the factor genotypes on the variation for AUDPC (Table 1).

### Genetic linkage map

Sixty-five SSR primer pairs produced polymorphic bands and were used for genotyping the population. Analysis of the mapping population using polymorphic AFLP primer combinations resulted in 208 polymorphic bands with an average of 8.3-bands/primer pair. The genetic linkage map was constructed using 65 SSR and 208 AFLP markers. Of 273 markers, 250 could be mapped and yielded in 42 linkage groups, covering a genetic distance of 1931 cM and providing partial linkage groups for all chromosomes except 6A and 7B.

### Quantitative trait mapping

Simple interval mapping analysis detected three QTLs on chromosomes 1BL, 3AL and 7AS (Table 2). The LOD scores ranged from 2.10 to 3.40 and the corresponding  $r^2$  ranged from 5.4 to 9.7 (Table 2). The QTLs on 1BL, 3AL, and 7AS were significant in the individual experiments and the combined analysis above 2 years. For the QTLs on chromosomes 3A and 7A, the resistance was conferred by alleles of 'Frontana',

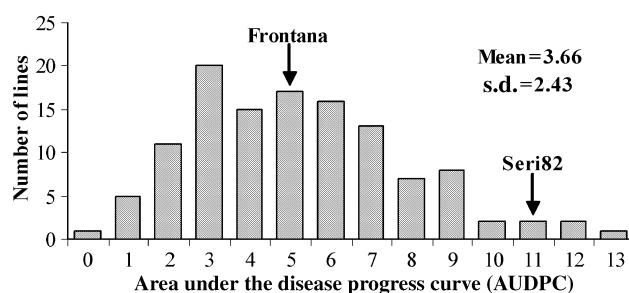


Fig. 1: Frequency distribution of 120 lines from 'Frontana'/'Seri82' for area under the disease progress curve (AUDPC) based on combined data across 2 years. Information included in the figure are means and standard deviations

Table 1: Analysis of variance for area under the disease progress curve (AUDPC) obtained from 120 F<sub>3:5</sub> lines derived from 'Frontana'/'Seri82' evaluated in 2002

Source	DF	MS	P-value
Rep.	1	0.20	0.2925
Genotype	119	0.41	0.0001
Error	119	0.18	

Table 2: The map intervals, chromosomal locations, source of resistance allele, logarithm of odds (LOD) and the percentage of explained phenotypic variance (VE) of quantitative trait loci (QTL) detected for area under the disease progress curve (AUDPC) using  $F_3$  and  $F_5$  generations derived from 'Frontana'/'Seri82'

Map interval	Chromosome	Source of resistance allele	$F_3$ (2001)		$F_{3:5}$ (2002)		Combined analysis	
			LOD	VE	LOD	VE	LOD	VE
<i>Xe38m50_10</i> – <i>Xe32m65_10</i>	1BL	Seri82	2.18	5.8	2.35	6.6	2.81	7.9
<i>Xgwm720</i> – <i>Xgwm1121</i>	3AL	Frontana	2.94	9.1	2.10	5.4	2.77	7.7
<i>Xe77m47_22</i> – <i>Xgwm233</i>	7AS	Frontana	2.75	7.9	3.40	9.7	2.70	7.6

QTL analysis was carried out by simple interval mapping (SIM).

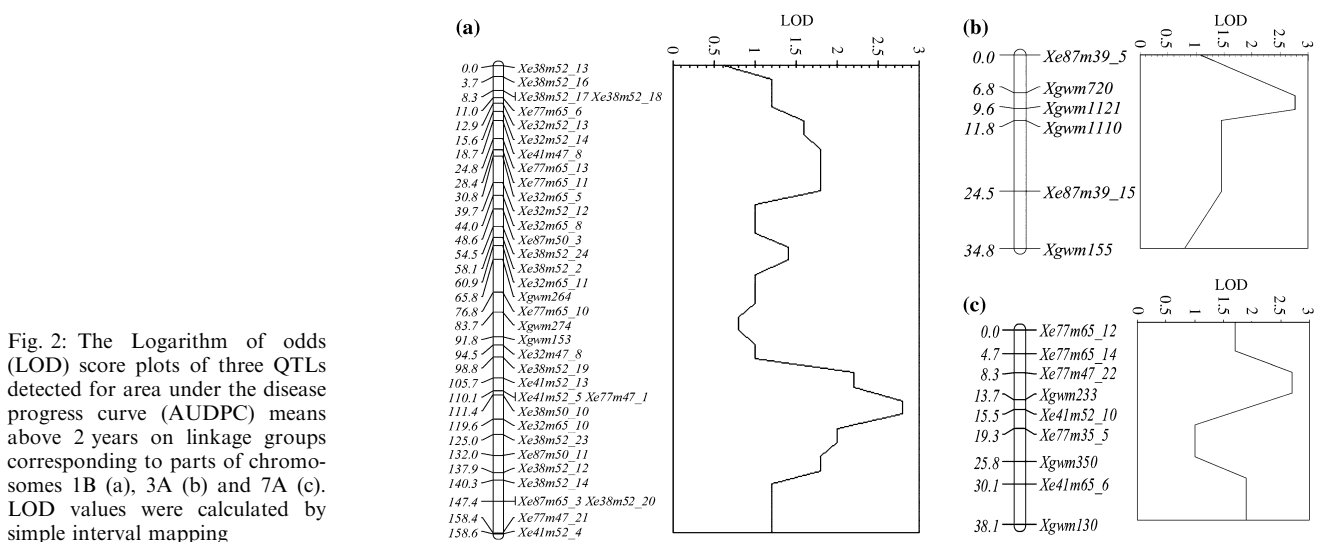


Fig. 2: The Logarithm of odds (LOD) score plots of three QTLs detected for area under the disease progress curve (AUDPC) means above 2 years on linkage groups corresponding to parts of chromosomes 1B (a), 3A (b) and 7A (c). LOD values were calculated by simple interval mapping

whereas the QTL on chromosome 1BL was contributed by 'Seri82'. The linkage groups and LOD profiles for mean AUDPC values around the QTL regions on chromosomes 1B, 3A and 7A and are presented in Fig. 2.

## Discussion

### FHB evaluation

Field screening of FHB resistance in wheat is difficult because of variations in abundance of inoculum, plant development stage, humidity and temperature (Parry et al. 1995). The resistance tests were therefore carried out in two seasons. Because of the non-specific nature of FHB resistance in wheat (Van Eeuwijk et al. 1995), one well-defined and highly aggressive *Fusarium graminearum* isolate (IFA65) was used for artificial inoculation (Lemmens et al. 1993, Buerstmayr et al. 2000). The percentage of infected spikelets after spray inoculation (reflecting all possible FHB resistance mechanisms) was used for estimation of AUDPC (Steiner et al. 2004, Mardi et al. 2005). The severe and uniform infection of the susceptible parent 'Seri82' and the significant correlation coefficient between AUDPC means from the 2001 and 2002 trials indicated accuracy and reproducibility of the field inoculation and FHB scoring methods, both here and in previous studies (Mardi et al. 2004, 2005). The distribution of AUDPC values revealed the quantitative nature of FHB resistance in the current 'Frontana'-derived population. Similar findings were reported in other 'Frontana' crosses (Singh et al. 1995, Van Ginkel et al. 1996, Steiner et al. 2004) and other spring wheat (Bai et al. 1999, Waldron et al. 1999, Anderson et al. 2001,

Buerstmayr et al. 2002, 2003, Zhou et al. 2002, Mardi et al. 2005).

### QTL mapping

By combining the results above 2 years, three genomic regions associated with resistance to FHB were identified. The QTLs on chromosomes 1BL, 3AL and 7AS were consistently detected and seem to be essential for FHB resistance expression in 'Frontana'/'Seri82'. The consistently detected QTL on chromosome 1B derived from 'Seri 82' in this study is possibly the same QTL located on 1BL close to the centromere reported in 'Fundulea 201R' (Shen et al. 2003), 'Alondra' (Zhang et al. 2004) and 'Lynx' (Schmolke et al. 2005), which all carry the T1BL.1RS translocation. In the map, this QTL was detected with an estimated distance of 45.6 and 19.6 cM from the *Xgwm264* and *Xgwm153* loci, respectively. *Xgwm264* and *Xgwm153* loci were reported to map at the short- and long-arm of 1B, respectively, by Röder et al. (1998) using the ITMI mapping population for genetic mapping. More recently, Sourdille et al. (2004) located the SSR locus *Xgwm264* on the long arm of 1B in the deletion bin C-1BL6–0.32 and the locus *Xgwm153* in the deletion bin 1BL2–0.69–1.00 by applying physical mapping using 'Chinese Spring' deletion lines. Several resistance QTLs mapping on different parts of chromosome 3A were reported in different mapping populations (Otto et al. 2002, Bourdoncle and Ohm 2003, Gervais et al. 2003, Shen et al. 2003). The consistently detected QTL on chromosome 3AL in the present study is the same as that reported by Steiner et al. (2004), who evaluated a 'Remus'/'Frontana'

doubled haploid population above 3 years. After spray inoculation they detected consistent QTLs for FHB severity on chromosomes 3A and 5A and smaller QTL effects on 2B, 4B and 6B, but found no QTL on 7A. The consistent QTL detected on 7AS is likely to be different from that reported by Bjornstad et al. (2004) in 'Arina' detected on 7AL.

Current results agree with those of Steiner et al. (2004), indicating that chromosome 3AL is predominantly involved in the FHB resistance of 'Frontana'. In addition, Steiner et al. (2004) detected a consistent QTL on chromosome 5A of 'Frontana', whereas the present study indicates a consistent QTL on 7A. This discrepancy may be due to incomplete map coverage or to different experimental conditions in the two mapping experiments. The results agree with Steiner et al. (2004) in indicating that the QTLs of 'Frontana' are different from the major FHB resistance QTL *Qfhs.ndsu-3BS* derived from Chinese wheat 'Sumai-3', 'Ning7840' and 'CM-82036' (Waldron et al. 1999, Anderson et al. 2001, Buerstmayr et al. 2002, 2003, Zhou et al. 2002). Because FHB resistance genes act mainly additively (Buerstmayr et al. 1999, Mardi et al. 2004), a combination of 'Frontana' resistance with 'Sumai-3' resistance QTLs may lead to lines with augmented and stable resistance expression. Microsatellite markers linked to the stable QTLs on 3BS and 5A of 'Sumai-3' or its derivatives and 3AL of 'Frontana' should be used to select for the desired genotypes.

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