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Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. II. Resistance to fungal penetration and spread

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Abstract Fusarium head blight (FHB, scab) causes severe yield and quality losses, but the most serious concern is the mycotoxin contamination of cereal food and feed. The cultivation of resistant varieties may contribute to integrated control of this fungal disease. Breeding for FHB resistance by conventional selection is feasible, but tedious and expensive. The aim of this work was to detect QTLs for combined type I and type II resistance against FHB and estimate their effects in comparison to the QTLs identified previously for type II resistance. A population of 364, F_1 derived doubled-haploid (DH) lines from the cross 'CM-82036' (resistant)/ 'Remus' (susceptible) was evaluated for components of FHB resistance during 2 years under field conditions. Plants were inoculated at anthesis with a conidial suspension of *Fusarium graminearum* or *Fusarium culmorum*. The crop was kept wet for 20 h after inoculation by mist-irrigation. Disease severity was assessed by visual scoring. Initial QTL analysis was performed on 239 randomly chosen DH lines and extended to 361 lines for putative QTL regions. Different marker types were applied, with an emphasis on PCR markers. Analysis of variance, as well as simple and composite interval mapping, revealed that two genomic regions were significantly associated with FHB resistance. The two QTLs on chromosomes 3B (*Qfhs.ndsu-3BS*) and 5A (*Qfhs.ifa-5A*) explained 29 and 20% of the phenotypic variance, respectively, for visual FHB severity. *Qfhs.ndsu-3BS* appeared to be associated mainly with resistance to fungal spread, and *Qfhs.ifa-5A* primarily with resistance to fungal penetration. Both QTL regions were tagged with flanking SSR markers. These results indicate that FHB resistance was under the control of two major QTLs operating together with unknown numbers of minor genes. Marker-assisted selection for these two major QTLs appears feasible and should accelerate the development of resistant and locally adapted wheat cultivars.

Keywords *Triticum aestivum* · Fusarium head blight · Resistance · Genetic mapping · QTLs

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Introduction

Fusarium head blight (FHB, scab) is a widespread and destructive disease of small grain cereals. Apart from losses in grain yield and quality, the most serious threat of FHB is the contamination of the harvested grain with mycotoxins. Chemical and agronomic control measures are either not available or not feasible. The cultivation of genetically resistant cultivars is the most cost-effective method to control the disease. A range of sources for FHB resistance have been identified (Snijders 1990; Buerstmayr et al. 1996; Rudd et al. 2001). However, the development of FHB resistant cultivars is still challenging because the resistance trait needs to be incorporated into locally adapted germplasm. The classical model proposed by Schroeder and Christensen (1963), who suggest two components of resistance (type I and type II), has been widely accepted by most authors. Type-I resistance operates against initial infection and type II against spread of the pathogen within the host. Further types or components of FHB resistance have been proposed by Mesterhazy (1995). Kolb et al. (2001) summarized the recent advances in molecular genetic analysis of FHB

resistance in wheat and barley. All reports published so far focused on resistance type II, where resistance evaluation was done by placing *Fusarium* inoculum directly into a floret (Bai et al. 1999; Waldron et al. 1999; Anderson et al. 2001; Buerstmayr et al. 2002). Under natural conditions conidia or ascospores land on the surface of the glumes and the fungus has to penetrate into the plant tissue. In a detailed study Kang and Buchenauer (2000) observed that fungal spores germinated on the plant surface and hyphal networks were usually formed within 2 days on the inner surfaces of lemma, palea and glume. Penetration of the host tissues occurred by infection hyphae on the inner surfaces of the floret. In the present study we evaluated a population segregating for FHB resistance by simulating a FHB epidemic in the field. The aim of this work was to detect QTLs for combined type I and type II resistance against FHB and estimate their effects in comparison to the QTLs identified previously for type II resistance (Anderson et al. 2001; Buerstmayr et al. 2002).

Materials and methods

Plant materials

The population of recombinant F_1 derived doubled-haploid (DH) lines which was described in Buerstmayr et al. (2002) was used for this research. In brief: the parental lines were 'CM-82036-1TP-10Y-OST-10Y-OM-OFC' (abbreviated to 'CM-82036' in the following text) and 'Remus'. In total 364 DH lines were evaluated for resistance and 361 DH lines were used for QTL mapping. 'CM-82036' originates from the cross 'Sumai#3'/'Thornbird-S'. It expresses a high level of FHB resistance both after spray (Buerstmayr et al. 1996) and single-floret inoculation (Buerstmayr et al. 2002). 'Remus' is a FHB susceptible German spring wheat cultivar. Doubled-haploid production was performed using wheat by maize pollination (Laurie and Bennett 1988).

Field experiments for evaluation of Fusarium head blight resistance

Experiments in Tulln 1999 and 2001

The DH population and the parental lines were evaluated during two seasons (1999 and 2001) at the experimental field of IFA-Tulln, 30 km west of Vienna, at 180 m above sea level. The average temperature and annual precipitation were 10.4 °C and 611 mm (1999), and 9.4 °C and 632 mm (2001). Trial location, seed treatment, plot size, sowing density and crop management were described in Buerstmayr et al. (2002). Sowing time was the first half of March in both years. The experiment was a split-plot design with three main plots: isolates (*Fusarium graminearum* and *Fusarium culmorum* and non-inoculated control) and 366 subplots within each main plot: the parents and the DH lines. The trial was sown in four or two replications in 1999 and 2001, respectively. The replications were purposely sown several days apart, resulting in a one to three days difference in anthesis between replications. For inoculation, macroconidia of the *F. culmorum* single-spore isolate 'IPO 39-01' were prepared as described by Buerstmayr et al. (2000) and of the *F. graminearum* single-spore isolate 'IFA-65' as described by Buerstmayr et al. (2002). The final spore concentration for inoculation was 5×10^4 spores ml^{-1}. Inoculations were performed individually on each plot when 50% of the plants had reached anthesis, and repeated 2 days later. Using a motor driven back-pack sprayer, 50 ml of inoculum was sprayed on the heads. Neighboring plots were protected by a plastic shield. Inoculations were carried out in the evenings on alternate days. An automated mist-irrigation system, switched by leaf-wetness measurement, maintained humidity and kept the plants wet for 20 h after inoculation. In each plot the percentage of visually infected spikelets was scored according to a linear 0 to 100% scale on a whole-plot basis. For scoring we assumed an average head-size of 20 spikelts per spike as the basis for estimating FHB severity; e.g. an average of one infected spikelet per spike was rated as 5% FHB severity. The percent FHB severity was recorded on days 10, 14, 18, 22 and 26 after inoculation. The area under the disease progress curve (AUDPC) was calculated for each plot and was used for further statistical analysis as described by Buerstmayr et al. (2000). After ripening, 30 heads were harvested manually from each plot. The weight of these 30 heads was recorded and used to calculate the yield of the inoculated plots relative to the non-inoculated control.

Verification experiment in Hohenheim 2001

The population was grown in 2001 at Hohenheim near Stuttgart, Germany (400 m above sea level, 10.1 °C average temperature, 821 mm precipitation). The trial was a randomized complete block design with three replications sown on April 5, 2001; plot size was the same as in Tulln. To avoid infection by unwanted fungal pathogens, all plots were sprayed once with 'Opus Top' (Epoxiconazol 126 g ha^{-1} + Fenpropimorph 375 g ha^{-1}, BASF, Ludwigshafen, Germany) shortly before heading.

Inoculum of the single-spore *F. culmorum* isolate 'IPO 39-01' was prepared according to Miedaner et al. (1996). For inoculation, a suspension of 5×10^5 conidia ml^{-1} was applied at a rate of 1,000 l ha^{-1} with a tractor-mounted sprayer in the evenings. To account for the variation in flowering time within the population, the whole experiment was inoculated four times (June 20, June 22, June 26, July 2). Inoculation dates were timed such that each genotype was inoculated at least once at its appropriate mid-flowering stage.

Plots were rated on a whole-plot basis at five successive dates in terms of the percentage infected spikelets per plot (0–100%). Head-blight rating started with the appearance of the first symptoms and continued according to disease progress. Early flowering genotypes displayed the disease symptoms most early, and disease severity scores of the 1st, 2nd and 3rd rating-date were used. Accordingly, for medium-flowering genotypes the 2nd, 3rd and 4th rating, and for late-flowering genotypes the 3rd, 4th, and 5th ratings, were taken for calculating an average disease severity for each line.

Genotyping of the DH population with molecular markers

Genotyping of 239 of the 361 DH lines was performed using 28 RFLPs, 267 AFLPs, 112 SSRs, three storage proteins and one morphological marker. The molecular data were the same as used by Buerstmayr et al. (2002) but extended for one RFLP and 23 SSRs (Roeder et al. 1998; Pestova et al. 2000; Cregan et al. 2001; Song et al. 2002). The other 122 DH lines were genotyped with the markers Barc75, Gwm389, Gwm1034, Gwm533, Barc133, Gwm493, Barc141, Barc40, Gwm304, Gwm293, Barc117, Barc186 and Barc1, which appeared to be close to one of the putative QTL regions, and included in the QTL mapping.

Statistical analysis

Visual disease data: the FHB severity data (percentage of diseased spikelets and AUDPC per plot) obtained in Tulln were analyzed by ANOVA over two isolates (*F. graminearum, F. culmorum*) and 2 years (1999, 2001) using SAS/STAT (SAS Institute 1989). Broad-sense heritabilities were estimated according to Nyquist (1991). The FHB severity data obtained in Hohenheim were analyzed as a single randomized complete block experiment by ANOVA.

Ear weight: the weight of 30 heads of each line was expressed in the percent of the non-inoculated control within every experiment (year by isolate combination) and then analyzed by ANOVA over 2 years.

Marker data: linkage maps were constructed using MAPMAKER 3.0 (Lander et al. 1987) assuming the Haldane (1919) mapping function. A logarithm of odds (LOD) threshold of 3 was set for grouping. Most-likely marker orders were determined using the MAPMAKER 'ripple' command. Linkage map construction was first carried out on 239 DH lines. The additional 122 DH lines were included for map construction and map verification for the linkage groups corresponding to putative QTL regions on chromosomes 3B and 5A

QTL analysis: QTL analysis was done by one- and multifactor-ANOVAs using the SAS GLM procedure (SAS Institute 1989) for DH-line mean values. Furthermore, simple interval mapping and composite interval mapping were carried out using PLABQTL (Utz and Melchinger 1996). QTL scanning was done with 411 markers on 239 DH lines. For putative QTL regions on chromosomes 3B and 5A the mapping population was extended to 361 DH lines. A QTL was declared significant with a LOD > 3. For composite interval mapping, cofactor selection was done automatically with an *F*-to-enter threshold of 15. Dependence of QTL estimation on sampling effects was estimated by a five-fold cross validation with the 'cross-validate' option of PLABQTL. With 4/5 of the individuals QTLs (positions and effects) were estimated and with the other 1/5 of the genotypes a validation was performed.

The FHB severity data from the independent experiment in Hohenheim 2001 were analyzed by interval mapping as described above, for verification of the QTLs.

Results

Fusarium head blight resistance

The DH lines showed continuous variation for the percentage of diseased spikelets 18–26 days after inoculation, for AUDPC and relative ear weight. As an example, Fig. 1 shows a histogram for the percentage of diseased spikelets on day 26 after inoculation (FHB-26). The resistant parent 'CM-82036' had an average of 9% disease severity on day 26 after inoculation, whereas 'Remus' had 77%.

Initial ANOVA calculations of the FHB data obtained in Tulln with all possible factors and interactions included in the model, revealed a non-significant effect of genotype by isolate, and genotype by isolate by year interaction, for all FHB related traits (data not shown). Therefore, the factor isolates was merged with replications in a simplified model.

ANOVAs and broad-sense heritabilities were calculated for the FHB severity observed at each of the five observation dates, AUDPC and relative ear weight.

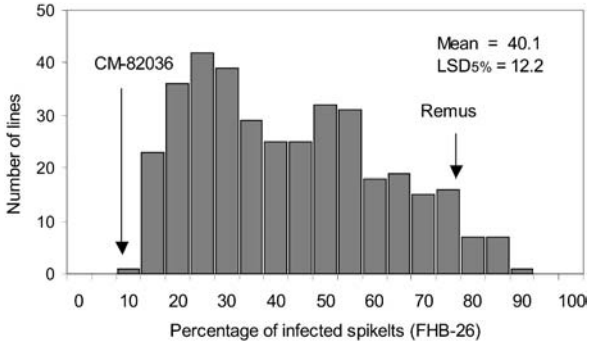

Fig. 1 Frequency distribution of 364 DH-lines for mean values of FHB severity on day 26 after inoculation (FHB-26). *Arrows* indicate values of the parental lines. The overall population mean and the least significant difference for comparison of line means ($\alpha = 0.05$) using the genotype by year interaction mean square as an error term are given as well

Heritabilities were highest for the assessments on days 22 and 26 after inoculation, and for AUDPC ($H^2 = 0.87$). The ANOVAs for FHB-26 and AUDPC are shown in Table 1. For both, FHB-related traits effects were significant for genotypes as well as genotype by year interaction. Genotype mean squares were therefore tested against genotype by year interaction mean squares.

Molecular map

Of the 411 polymorphic markers, 387 could be mapped and they formed 40 linkage groups, covering a total genetic distance of 2,404 cM. The chromosome identity for 28 linkage groups could be determined based on anchor microsatellite markers (Roeder et al. 1998). Partial maps were obtained for all wheat chromosomes apart from 4D. As shown in the linkage map corresponding to part of chromosome 5A (Fig. 2B) several markers appeared to be closely or completely linked (e.g. Gwm293, Gwm304a, Gwm1057, Barc186, Barc117, Barc186 and Barc56).

Quantitative trait mapping

Analysis of variance revealed that two genomic regions (located on chromosomes 3B and 5A) were significantly ($p < 0.0001$) associated with Fusarium head blight related traits (data not shown). Simple and composite interval

Table 1 Analysis of variance for the percentage of FHB infected spikelets on day 26 after inoculation (FHB-26) and the area under the disease progress curve over five scores (AUDPC) obtained in Tulln across 2 years

Source	*df*	FHB-26			AUDPC		
		MS	*F*-value	*P*	MS	*F*-value	*P*
Replications (in years)	12	8,201.71	35.15	<0.0001	1,002.59	48.15	<0.0001
Years	1	2,009.47	8.61	0.0034	88.72	4.26	0.0391
Genotypes	365	3,822.47	7.53	<0.0001	333.34	7.96	<0.0001
Genotypes × years	365	507.70	2.18	<0.0001	41.88	2.01	<0.0001
Error	3,599	233.32			20.82		

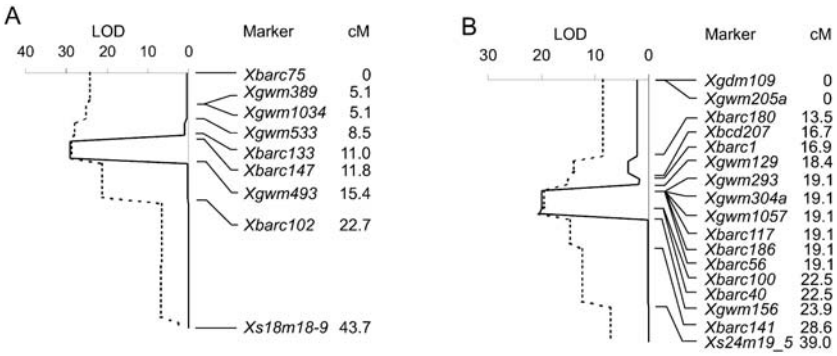

Fig. 2 Interval analysis of QTLs for the percentage of infected spikelets on day 26 after inoculation (FHB-26) on linkage groups corresponding to chromosomes 3B (**A**) and 5A (**B**). LOD values were calculated by composite interval mapping (*solid line*) and simple interval mapping (*dotted line*)

Table 2 QTL estimates for mean values of percentage of infected spikelets on day 26 after inoculation (FHB-26), the area under the disease progress curve (AUDPC) and the relative ear weight (REW) over 2 years of the experiments in Tulln, and for the mean percentage of diseased spikelets across three observations obtained in Hohenheim in 2001. QTLs are described by chromosome location, logarithm of odds (LOD) and percentage of explained phenotypic variance (VE). QTL analysis was carried out by composite interval mapping

Map interval	Chromosome	Tulln (means over 1999 and 2001)						Hohenheim (2001)	
		FHB-26		AUDPC		REW		FHB-mean	
		LOD	VE	LOD	VE	LOD	VE	LOD	VE
Xgwm533–Xgwm493	3B	29.1	31.6	17.6	20.4	36.5	37.8	25.8	28.6
Xgwm293–Xgwm156	5A	20.5	23.2	21.4	24.1	13.4	15.9	11.6	13.9
Simultaneous fit			46.9		40.3		47.6		37.9

mapping also confirmed the prominent QTL effects on chromosomes 3B and 5A. The two genomic regions were significantly associated with single FHB severity readings on day 18, 22 and 26 after inoculation, with AUDPC and relative ear weight. The QTL positions remained in the same regions regardless of testing the year or the Fusarium strain used (data not shown). As revealed by composite interval analysis the most likely position on chromosome 3B is in the *Xgwm533–Xgwm493* interval. The most likely QTL position on 5A is in the *Xgwm293–Xgwm156* interval (Table 2 and Fig. 2). In all tested models with combinations of QTLs, the additive effects were significant, while the two-way epistatic interactions were not significant. The QTL analysis by interval mapping of the mean FHB severity readings obtained in the experiment at Hohenheim revealed the same QTL positions, with a larger effect of 3B compared to 5A (Table 2). Five-fold cross-validation consistently detected both QTLs. As an example the cross validation result for the mean percentage of infected spikelets 26 days after inoculation is given in Table 3. The magnitude of the explained phenotypic variance was comparable in the calibration and in the validation set (Table 3), indicating

Table 3 Five-fold cross validation of QTLs for mean percentage of infected spikelets on day 26 after inoculation across 2 years (FHB-26). Data were split in five subsets of 289 lines used for QTL detection (calibration) and the remaining 72 lines were used for QTL validation. For description see Table 2

Cross validation split	Calibration		Validation VE
	QTL detected	VE	
1	3B 5A	44.7	58.1
2	3B 5A	45.8	49.9
3	3B 5A	44.6	55.5
4	3B 5A	51.7	26.6
5	3B 5A	47.5	43.7

Table 4 Effect of alternative alleles at two QTL regions for the mean percentage of infected spikelets 26 days after inoculation (FHB-26) and relative ear weight of inoculated heads in percent of the non-inoculated control (REW) for line means obtained in Tulln over 2 years

QTL[a]		Number of lines	FHB-26			REW		
Qfhs.ndsu-3BS	*Qfhs.ifa-5A*		Median	Mean	SE	Median	Mean	SE
CM-82036	CM-82036	87	19.8	21.9	8.5	92	92	7
CM-82036	Remus	74	34.1	34.8	13.6	86	86	8
Remus	CM-82036	73	37.5	39.6	13.1	78	80	7
Remus	Remus	110	58.3	57.7	16.1	70	71	8

[a] Only lines with non-recombined *Xgwm533–Xgwm493* (*Qfhs.ndsu-3BS*) and *Xgwm293–Xgwm156* (*Qfhs.ifa-5A*) intervals were included in these calculations

that the QTL positions and effects were estimated correctly.

For both QTLs the allele-conferring resistance originated from the resistant parent 'CM-82036'. The association of the two QTLs on 3B and 5A with the phenotype is shown in Table 4. Lines with the resistance allele (originating from 'CM-82036') at both QTL regions showed a mean FHB severity of 22%, whereas lines with both alleles from susceptible 'Remus' reached on average 58% bleached spikelets after 26 days.

Discussion

FHB resistance assessment

Precise phenotypic evaluation is a prerequisite for any QTL mapping. FHB resistance of wheat is a quantitative trait, confounded by environmental effects like temperature, humidity, plant development stage and abundance of inoculum (Parry et al. 1995). Therefore, artificial inoculation is indispensable for a reliable FHB resistance evaluation of wheat genotypes. Whereas previous studies were based on single floret inoculation and analysis of type II resistance, in this study we aimed to measure FHB resistance by simulating an epidemic under field conditions. The disease reaction should thus reflect all possible mechanisms, which may contribute to the resistant phenotype under epidemic conditions. In order to account for the environmental influence on FHB infection, resistance testing was done in several macro-environments (years and locations) and micro-environments (sowing of replications in staggered time intervals), as described in Buerstmayr et al. (2000). Despite the small but significant genotype by year interaction for the FHB related traits, heritabilities were high, indicating that we achieved a reproducible measurement of disease severity of the lines. As in our previous study (Buerstmayr et al. 2002) one *F. culmorum* and one *F. graminearum* isolate were applied on separate plots. We again found a non-significant genotype by isolate interaction underlying the non-specific or horizontal nature of FHB resistance in wheat (Van Eeuwijk et al. 1995). We found a quantitative distribution among the doubled-haploid lines for the FHB related traits, without significant transgressions. The resistant parent 'CM-82036' appears to have contributed nearly all of the resistance alleles compared to 'Remus'.

QTL mapping

The linkage map was mainly based on PCR markers. Anchor SSR markers allowed us to relate most linkage groups to wheat chromosomes without the need to use aneuploid stocks.

After an initial QTL analysis and based on previous results (Anderson et al. 2001; Buerstmayr et al. 2002) we continued to genotype our population with markers mapping to chromosomes 3B and 5A respectively and extended the mapping population to 361 lines. Marker order and distances on the linkage group corresponding to chromosome 3B were in agreement with other published maps (Roeder et al. 1998; Anderson et al. 2001), but this was not the case for 5A. On the obtained 5A group many markers appeared to be closely or completely linked around the centromere, in the region where the 5A QTL was identified (Fig. 2). This map is in disagreement with the SSR map of Roeder et al. (1998). We assume that the two parental genotypes may have structural differences in that region suppressing recombination.

Two genomic regions were significantly associated with FHB resistance in this population, mapping to chromosomes 3B (*Qfhs.ndsu-3BS*) and 5A (*Qfhs.ifa-5A*), respectively. The two QTLs together explained 40–48% of the phenotypic variance depending on the resistance trait. The peaks of the LOD profiles obtained by simple and by composite interval mapping were in the same regions. The two QTLs on 3B and 5A mapped to the same genomic regions as in our previous study for type II FHB resistance (Buerstmayr et al. 2002), with the exception that we did not find a QTL after spray inoculation on chromosome 1B. Our results concerning the *Qfhs.ndsu-3BS* locus are in full agreement with Waldron et al. (1999) and Anderson et al. (2001) who first reported type II resistance at this QTL, as well as Zhou et al. (2002). In the present study using spray inoculation, the effects of the two QTLs were in a comparable range. By contrast, after single floret inoculation, the 3B QTL had a much larger effect than the 5A QTL (Buerstmayr et al. 2002). This is an indication that *Qfhs.ifa-5A* may contribute more towards type I resistance and to a lesser extent to type II resistance, whereas *Qfhs.ndsu-3BS* appears to play a role primarily in type II resistance. Based on the maps and LOD profiles obtained by composite interval mapping we assume that *Qfhs.ndsu-3BS* is a single gene or a cluster of tightly linked genes. Because of the condensed map at *Qfhs.ifa-5A* it is very difficult to determine its genetic structure.

Both QTL regions are already well covered by SSR markers. Marker-assisted selection for the two major QTLs appears therefore feasible and should help breeders to select for improved lines with combined type I and type II resistance. We consider these markers especially useful to transfer FHB resistance from the spring wheat line 'CM-82036' into regionally adapted lines and cultivars. In addition, the transfer of the two QTLs into durum wheat appears feasible, since both are not on any D-genome chromosome.

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experiments conducted for this publication comply with the current laws of Austria and Germany.

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