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Abstract *Septoria tritici* blotch (STB), caused by
S. tritici, Stagonospora glume blotch (SGB), caused
by *S. nodorum*, and *Fusarium* head blight (FHB),
caused by *F. graminearum* and *F. culmorum*, are the
most important diseases of wheat (*Triticum aestivum*
L.) in temperate growing areas. The main goals of
this study were to detect (1) new quantitative trait loci
(QTL) for STB resistance in two adapted European
biparental populations (Arina/Forno, History/Rubens)
and (2) QTL regions for broad-spectrum resistance
(BSR) to the above-mentioned diseases during the
adult-plant stage in the field. The three resistances

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were phenotyped across 4–7 field environments and phenotypic data revealed significant ($P < 0.01$) genotypic differentiation in all cases. Entry-mean heritabilities (h^2) ranged from 0.73 to 0.93. For STB resistance, correlations between disease ratings and heading date were significant ($P < 0.01$), but moderate ($r = -0.23$ to -0.30) in both populations. Correlations between STB and plant height were higher in Arina/Forno ($r = -0.45$) and History/Rubens ($r = -0.55$), the latter population segregating at the *Rht-D1* locus. During the initial QTL analysis, 5 QTL were detected for STB resistance in each of the populations, amounting to an explained genotypic variance of 45–63%, thus, showing the same ranges as FHB and SGB resistances in Arina/Forno and FHB resistance in History/Rubens. In total, 7 BSR QTL were found in the meta-analysis with the raw data, including the QTL on chromosome 4D at the *Rht-D1* locus. A BSR QTL for all three diseases was not found but several BSR QTL for combinations with two diseases were detected. Combining the BSR QTL detected in the present breeding material by applying marker-assisted selection seems a promising approach.

Keywords Adult-plant resistance · Broad spectrum resistance · *Fusarium* head blight · Meta-analysis · *Mycosphaerella graminicola* · *Phaeosphaeria nodorum* · QTL · *Septoria tritici* blotch · Septoria glume blotch · *Triticum aestivum* · Wheat

Introduction

Wheat (*T. aestivum* L.) is one of the most important food crops worldwide, covering more land area than any other crop (FAOSTAT 2010). In Germany, arable land comprises about 12 million hectares including 3.2 million hectares of wheat. In comparison to other wheat-growing regions worldwide with an average yield of 1–3 t/hectare, Germany is one of the highest yielding areas with an average yield of 7–8 t/hectare (FAOSTAT 2010), with winter wheat being predominant. Several fungal diseases restrict maximum grain yield, among which *Septoria tritici* blotch (STB), caused by *S. tritici* (teleomorph *M. graminicola*), Stagonospora glume blotch (SGB), caused by *S. nodorum* (teleomorph *P. nodorum*), and *Fusarium* head blight (FHB), caused by

F. graminearum (teleomorph *G. zeae*) and *F. culmorum*, are the most important fungal pathogens. Each of them can cause considerable yield losses. FHB additionally affects grain quality and leads to mycotoxin contamination. Cultivation of resistant varieties is the most environmentally friendly and cost-effective strategy for controlling these diseases.

The main challenge in European wheat breeding is to combine maximum grain yield with good baking quality and acceptable levels of resistance to the most important pathogens. Resistances to STB, FHB, and SGB are of great relevance in wheat breeding; they are quantitatively inherited and controlled by multiple genes (Schnurbusch et al. 2003; Paillard et al. 2004; Semagn et al. 2007; Uphaus et al. 2007; Holzapfel et al. 2008; Shankar et al. 2008; Bonin and Kolb 2009). Additionally, race-specific resistances are mediated by several *Stb* resistance genes (*cf.* Goodwin 2007). Analyses of quantitative trait loci (QTL) were previously carried out for resistance to FHB (Paillard et al. 2004) and SGB (Schnurbusch et al. 2003; Abeysekara et al. 2009) in the Swiss population Arina/Forno and for resistance to FHB in History/Rubens (Holzapfel et al. 2008). The resistance donor Arina was mapped independently in other studies for STB resistance (Chartrain et al. 2004) and FHB resistance (Draeger et al. 2007; Semagn et al. 2007). A recently published QTL analysis of quantitative STB resistance in two other European wheat populations (Florett/Biscay, Tuareg/Biscay) revealed a high number of loci with two major QTL/population (Risser et al. 2011). Broad-spectrum resistance (BSR) is restricted in our study to loci conferring resistance to multiple taxa of pathogens within one crop (Poland et al. 2009). BSR loci have previously been described in *Arabidopsis*, maize and rice (Cao et al. 1998; Wisser et al. 2005; Wisser 2006). In *Arabidopsis* even individual genes effective against groups of pathogens, e.g., *NDR1*, *NPR1*, *esa1*, *WRKY33*, have been cloned (*cf.* Poland et al. 2009). There is also evidence for the existence of BSR loci in wheat, including the detection of non-adapted germplasm with resistances to multiple diseases (Gurung et al. 2009) and clustering of resistance genes in distinct genomic regions such as on chromosomes 3BS (*Sr2*, *Lr27*, *Yrns-B1*, *Rph7*-homologue, FHB, SNG; *cf.* Miedaner et al. 2008) or 3DL (seedling STB, tan spot, *Lr24*, *Sr24*; Zwart et al. 2010). Another prominent example is the *Lr34*/*Yr18*/

Pm38 locus in wheat which confers resistance to leaf rust, yellow (stripe) rust, powdery mildew, and barley yellow dwarf virus (*Bdv1*). This locus represents a single gene (Krattinger et al. 2009) and is the only example in wheat where the actual gene causing BSR has been cloned.

Meta-QTL analysis identifies consensus QTL across studies which allows validation of results from individual studies and refining of QTL positions on the consensus map by statistical methods (Goffinet and Gerber 2000). In contrast to meta-QTL studies for disease resistance using data from the literature (Liu et al. 2009; Löffler et al. 2009), we searched for BSR QTL within two elite populations across different quantitative resistances by combining the original data from several experiments. The objectives were to (1) analyze the Arina/Forno and History/Rubens populations for quantitative STB resistance QTL for the first time and (2) identify chromosomal locations, positions, support intervals, and genetic effects of BSR-QTL by combining STB data with FHB (History/Rubens) and FHB and SGB data (Arina/Forno) from previous mapping studies by meta-QTL analysis.

Materials and methods

Initial population development

In previous studies, mapping populations of 240 and 103 recombinant inbred lines (RILs) were developed from a cross between Arina and Forno and between History and Rubens, respectively (Paillard et al. 2003; Schnurbusch et al. 2003; Holzapfel et al. 2008). Arina and Forno are Swiss cultivars; History and Rubens have been bred in Germany. Arina is quantitatively resistant to STB, FHB, and SGB, History to FHB and STB; Forno and Rubens are the susceptible parents with the latter including the dwarfing allele *Rht-D1b*. For this study, 200 and 94 RILs were used from Arina/Forno and History/Rubens, respectively.

Field trials, inoculation, and resistance evaluation

All populations were evaluated for all disease resistances in independent series of field experiments. RILs of Arina/Forno have previously been evaluated

for FHB severity by spray inoculation in each of 6 environments (location–year combinations, Paillard et al. 2004) and for SGB severity by natural infections in 7 environments (Schnurbusch et al. 2003). RILs of History/Rubens were spray-inoculated for induction of FHB symptoms in 5 environments (Voss et al. 2008). All disease resistances were recorded by 3–5 visual ratings during epidemic development as the percentage of infected glume or leaf area, from 0%, i.e., no visible lesions, up to 100%. SGB and FHB in History/Rubens were rated on a plot basis, FHB in Arina/Forno on the basis of 10 randomly chosen ears from each plot.

In this study, RILs of Arina/Forno were for the first time evaluated for quantitative STB resistance in four locations (Hohenheim/Stuttgart, Oberer Lindenhof near Reutlingen, Freising near Munich, Wohlde near Celle) in 2009 and RILs of History/Rubens at each of three locations in 2008 and 2009 (Hohenheim, Oberer Lindenhof, Freising). The populations were grown together with 5 replicated entries of the parental lines in α-designs with two replications at each trial. Each entry was sown in two 1 m rows with approximately 40–60 kernels/row.

Inoculation and resistance evaluation for STB severity has previously been described in detail (Risser et al. 2011). In brief, we used one or two pretested isolates of *S. tritici* that infected all four parents in a seedling test to 90–100%: isolates BAZ 6/1/04 and BAZ 8/8/04 for Arina/Forno and BAZ 8/8/04 for History/Rubens. With this procedure, we wanted to exclude the effect of race-specific *Stb* resistance genes known to reside in Arina (*Stb6*, *Stb15*), Forno (*Stb6*), and Rubens (*Stb6*) and expressed in the seedling stage. We wanted to ensure that we detected quantitative adult-plant resistances only. Inoculum was produced at the Institute of Crop Science and Plant Breeding, Freising, and sprayed once with 5×10^6 spores/ml by a tractor-mounted sprayer in the period between full appearance of flag leaf and mid flowering after late genotypes' flag leaves had been fully unrolled. Inoculation was done after rainfall and during cloudy weather conditions so that moisture was retained on the leaf surface for several hours. At Freising 2009, mist irrigation was used after inoculation to enhance infection frequency.

In all field trials STB, heading date, and plant height were evaluated. STB severity was visually scored plotwise as percentage coverage of flag leaves

with lesions bearing pycnidia. Flag leaves were assessed three times at an interval of 4–7 days, starting from about 30 days after inoculation. The arithmetic mean of two scorings, representing middle and end of disease development, was used in the following analyses. Unfortunately, inoculation at Wohlde did not lead to any scorable STB. Heading date was scored as days after 1st January when 50% of spikes of a plot had emerged. Plant height was measured from the soil surface to the middle of the spike on the main tillers.

Phenotypic data analyses

Analyses of phenotypic data were based on single-plot values. Estimation of variance components was done using the programme package PLABSTAT (Utz 2001). Residuals of STB mapping populations did not follow a normal distribution. Logit and arcsin transformations were applied, but did not improve the normality of the data. Therefore, untransformed data were used for analysis of variance (ANOVA) and QTL analysis. Lattice-adjusted means were calculated for genotypes in each environment and used for ANOVA. Analysis across environments was performed using a general linear model with genotype and environment effects, considering genotype as random. Entry-mean heritability (h^2) was calculated by PLABSTAT as the ratio of genotypic to phenotypic variance (Knapp et al. 1985):

$$h^2 = \frac{\sigma_g^2}{\sigma_g^2 + \frac{\sigma_{ge}^2}{E} + \frac{\sigma^2}{ER}}.$$

where σ_g^2 denotes the genotypic variance, σ_{ge}^2 the genotype × environment interaction variance, and σ^2 the error variance. E and R are the numbers of environments and replications/environment, respectively.

Genetic maps

A detailed genetic map of Arina/Forno was published by Paillard et al. (2003) with 380 marker loci spanning a total of 3,086 cM. The map of History/Rubens originally included 394 loci across 2,187 cM (Holzapfel et al. 2008). For this study, additional 97 polymorphic Diversity Arrays Technology (DArT) loci were included in the latter map (Risser 2010).

Abbreviations of DArT markers were used as provided by Triticarte (Triticarte P/L, Yarralumla, Australia, http://www.triticarte.com.au) with wPt and tPt referring to markers documented in the wheat and triticale map, respectively. Amplified fragment length polymorphism (AFLP) loci were designated according to the standard list provided by the manufacturer (KeyGene Inc., Wageningen, The Netherlands). For example, marker locus P6451-190 refers to a fragment of about 190 base pairs (bp) amplified with primer combination P64/M51 using *Pst*I/*Mse*I restriction enzymes. All simple sequence repeat (SSR) loci mentioned are publicly available.

QTL analyses

QTL analyses were applied within History/Rubens and Arina/Forno across three diseases: FHB, SGB, and STB. Schnurbusch et al. (2003), Paillard et al. (2004), and Holzapfel et al. (2008) provided phenotypic raw data of SGB (percentage of the infected glume area/spike) and FHB (percentage of diseased spikelets). In order to compare QTL effects between different experiments, the area under the disease progress curve (AUDPC) was not used for SGB and FHB in Arina/Forno, but rather the arithmetic mean of two scorings.

Initial QTL analysis was performed using composite interval mapping (CIM) by a multiple regression procedure with the program PLABMQTL (Utz 2009). We applied a LOD threshold of 3.0 to declare the presence of a putative QTL according to the previous authors working with the same populations. The Bayesian Information Criterion (BIC) was used to select the final model from the initially detected putative QTL having a LOD above the critical threshold. The critical LOD threshold was determined empirically according to Churchill and Doerge (1994) using 1,000 permutation runs. The proportion of genotypic variance (σ_G^2) explained by the model was calculated from the whole dataset ($\hat{p}_{DS}$) as $\hat{p}_{DS} = \hat{R}_{adj}^2 \big/ \hat{h}^2$, where $\hat{R}_{adj}^2$ is the adjusted proportion of phenotypic variance explained by the model and $\hat{h}^2$ is the heritability of the trait (Schön et al. 2004). The proportion of σ_g^2 explained by individual QTL in the model ($\hat{p}_{part}$) was calculated as $\hat{p}_{part} = \hat{R}_{part}^2 \big/ \hat{h}^2$ where $\hat{R}_{part}^2$ is the proportion of the phenotypic variance

explained by individual QTL. Individual $\hat{p}_{part}$ values were normalized according to Zhu et al. (2004) to sum up to $\hat{p}_{DS}$. The 95% confidence interval (CI) of the detected QTL was calculated after Darvasi and Soller (1997). Jiang and Zeng (1995) have proposed a multiple trait version of the CIM technique. This method is based on maximum likelihood, and requires special programs for analysis. We used PLABMQTL, written by the senior author (Utz 2009). If the correlation between residual effects of the traits is zero, the joint test statistic is approximately the sum of the test statistics for the single traits $LR_j \sim LR_{S1} + LR_{S2}$ (van der Werf 2000). Equal QTL-mapping procedures were applied for all three diseases to obtain comparable LOD curves and estimations of QTL effects. Meta-analysis was done by adding LOD scores of disease traits within each population. History/Rubens was analyzed for resistance to FHB and STB, Arina/Forno for resistance to FHB, SGB, and STB. According to the procedure described above, $\hat{p}_{part}$ was calculated as the part of genotypic variance explained by each individual BSR QTL. Critical LOD scores were determined within the program PLABMQTL by 1,000 permutation runs.

Results

QTL mapping for resistance to STB

Field trials revealed differentiation for STB rating between the parents and within the two mapping populations (Electronic Supplementary Material Table S1). Variance components for STB were highly significant ($P < 0.01$) for genotype and G × E interaction. History/Rubens population had a larger genotypic variance and only a slightly higher G × E interaction than Arina/Forno. Accordingly, entry-mean heritability was higher for the former population. Correlations between STB and heading date were negative and low for Arina/Forno and History/Rubens ($r = -0.23$ and -0.30, respectively) although significant ($P < 0.01$). Correlations between STB and plant height were considerably higher for Arina/Forno and History/Rubens ($r = -0.45$ and -0.55, respectively).

Both mapping populations showed a continuous distribution of mean STB rating averaged across environments (Electronic Supplementary Material Figure S1). Parental lines were located at either end of the distribution.

For STB resistance, we detected 5 QTL in each of both populations located on 5 chromosomes in Arina/Forno and four chromosomes in History/Rubens with LOD > 3 in the combined analysis across environments (Table 1). Due to the small size of the latter population, only QTL 1 is significant according to the critical LOD score after permutations. Values of $\hat{p}_{part}$ ranged between 3.0 and 24.4, with four QTL exceeding 10%. About half of the QTL show significant ($P < 0.01$) QTL × environment interaction.

Meta-analysis for broad-spectrum resistance QTL

Initial QTL analyses across all disease resistances in Arina/Forno revealed 7 and 3 QTL for FHB and SGB resistance, respectively, explaining together 52 and 42% of the genotypic variance (Electronic Supplementary Material Table S2). In History/Rubens, 6 QTL for FHB resistance were mapped, explaining 62% of $\hat{p}_{DS}$.

For meta-QTL analysis, LOD scores for STB, FHB, and SGB resistance in Arina/Forno and for STB and FHB resistance in History/Rubens were added to determine chromosomal localization, position, flanking markers, support interval (SI), and LOD scores of meta-QTL. Additionally, heading date and plant height in this study were used in meta-analysis. Using PLABMQTL, 7 QTL were detected showing significant ($P < 0.01$) additive effects across at least two resistance traits, located on chromosomes 3B, 4B, 5B, and 6D in Arina/Forno, and on chromosomes 2B, 4D, and 7B in History/Rubens, and were declared as BSR QTL. Meta-LOD scores ranged from 6.1 to 22.7 and from 18.1 to 48.7, respectively. Five out of seven BSR QTL were still significant at $P < 0.01$ by their critical LOD thresholds after the permutation test.

The BSR QTL with the highest additive effects was on chromosome 4D in History/Rubens, closely linked to the dwarfing *Rht-D1* locus (Table 2). The tall allele from History reduced disease severity by 9.8% for STB and 6.3% for FHB. All resistance alleles at one BSR QTL were contributed by the same parent. In Arina/Forno, no BSR QTL was found to have a significant effect on all three disease

Table 1 Chromosomal localization, flanking markers, LOD scores, distance to the next flanking marker (Dis.) and confidence interval (CI) of QTL with LOD ≥ 3.0 for resistance to *S. tritici* blotch (% flag leaf area affected, means across environments); N = population size

No.	Chromosome	Position (cM)	Flanking marker[a]		LOD[b]	Dis. (cM)	CI[c]	$\hat{p}_{part}^{d}$	QTL × E[e]
			Right	Left			(cM)		
Arina/Forno (N = 200)									
1	2B	140	Xpsr540	**Xcfd276**	5.5	0	133–147	9.6	**
2	3B	112	Xcfab2134	**Xgwm131**	7.3	2	96–128	14.2	**
3	5B	44	Xwmc473	**Xpsr574**	4.7	0	37–51	6.7	
4	6D	106	Xcfd19	**Xgdm14**	6.6	2	99–113	9.5	
5	7B	148	XksuD2	**Xgwm146**	3.7	0	130–157	3.0	**
							$\hat{p}_{DS}(\%)^{f}$ **45.5**		
History/Rubens (N = 94)									
1	4D	4	***Rht-D1***	Xbarc105	16.6	0	0–9	24.4	**
2	5B.1	34	XwPt-4996	**Xgwm274**	11.5	0	29–39	12.0	
3	5B.2	68	Xbarc142	**XP7152-196**	4.5	0	57–79	11.3	**
4	6B	16	**XP7162-180**	XP7256-485	7.9	0	11–21	8.2	**
5	7B	24	Xgwm263	**XP6653-115**	10.4	0	19–29	7.1	
							$\hat{p}_{DS}(\%)^{f}$ **63.0**		

** F test significant at $P < 0.01$

[a] Closest marker in *bold*

[b] Critical LOD scores after 1,000 permutations (α = 10%) are 4.5 (Arina/Forno) and 15.8 (History/Rubens)

[c] 95% confidence interval after Darvasi and Soller (1997)

[d] Partial genotypic variance explained by detected QTL

[e] QTL × environment interaction tested for significance (sequentially rejective Bonferroni F test)

[f] Adjusted genotypic variance explained by detected QTL (final simultaneous fit) across environments

resistances. Two BSR QTL, however, revealed resistances to STB and FHB, one to STB and SGB and another one to FHB and SGB. In History/Rubens, three BSR QTL significantly reduced both FHB and STB severity.

The LOD curves were plotted to illustrate BSR QTL in Arina/Forno (Fig. 1) and History/Rubens (Fig. 2). Effects of heading date and plant height on BSR QTL are crucial on chromosome 5B in Arina/Forno and chromosomes 2B, 4D, and 7B in History/Rubens, indicated by high LOD scores of these agronomic traits within support intervals of BSR QTL. In addition, significant QTL from initial QTL mapping experiments are labeled at the LOD peak. Initial QTL of Arina/Forno for STB, SGB, and FHB resistance on chromosomes 3B, 4B, 5B, and 6D and of History/Rubens for STB and FHB resistance on chromosomes 4D, and 7B were confirmed. Three

BSR QTL, on chromosome 5B in Arina/Forno and on chromosomes 4D and 7B in History/Rubens, are based on two initial QTL already significant in the single-trait analysis. All other BSR QTL were significant because of one detected initial QTL and the second initial QTL being below the significance level.

Discussion

The objectives of this study were to map new STB QTL and to reveal common QTL regions for BSR within two mapping populations using meta-analysis. Meta-analysis is defined as the integration of individual experiments with a comparative map-based approach. We focused on two wheat populations analyzed for quantitative resistance with different

Table 2 Chromosomal localization, position (Pos.), flanking markers, distance to the next flanking marker (Dis.), support interval (SI), effects and donors (D) of BSR QTL for *S. tritici* blotch (STB), *Fusarium* head blight (FHB), and *Stagonospora* glume blotch (SGB) in wheat population Arina/Forno and for STB and FHB in History/Rubens (N = population size)

Chromosome	Pos. (cM)	Flanking marker[a]		Dis. (cM)	SI (cM)	MetaLOD	P[b]	STB		FHB		SGB	
		Left	Right					Effect[c]	D[d]	Effect[c]	D[d]	Effect[c]	D[d]
Arina/Forno (N = 200)													
3B	120	**Xgwm131**	Xgwm383	5	106–130	6.1	<0.25	−5.9**	A	−3.1**	A	−0.8	A
4B	16	**Xpsr914**	Xglk335	1	10–22	12.0	<0.01	−2.5**	F	−0.2	F	−2.1**	F
5B	70	Xgwm639	**Xpsr120**	1	68–72	22.7	<0.01	−1.2	F	−3.8**	F	−1.7**	F
6D	106	Xcfd19b	**Xgdm14**	2	100–110	10.9	<0.01	−3.3**	A	−3.0**	A	−0.8	A
History/Rubens (N = 94)													
2B	140	**XwPt-0694**	XP7056-648	2	138–144	18.1	<0.25	−3.7**	R	−1.7**	R	–	–
4D	4	***Rht-D1***	Xbarc0105	0	2–6	48.7	<0.01	−9.8**	H	−6.3**	H	–	–
7B	24	Xgwm263	**XP6653-115**	1	22–28	30.6	<0.01	−5.4**	H	−3.1**	H	–	–

** Significant at $P < 0.01$ (standard *t* test of multiple regression)

[a] Closest marker in *bold*

[b] Probability of significance of the BSR QTL analyzed by critical LOD thresholds after 1,000 permutations; thresholds in Arina/Forno are 5.76 ($P < 0.25$), 7.51 ($P < 0.05$), and 10.63 ($P < 0.01$), repectively, in History/Rubens 16.04 ($P < 0.25$), 24.62 ($P < 0.05$), and 30.45 ($P < 0.01$), repectively

[c] Additive effects (% leaf area affected) of BSR QTL for STB, FHB, and SGB; negative numbers reflect better resistance

[d] Donor of resistance allele is Arina (*A*), Forno (*F*), History (*H*) or Rubens (*R*), respectively

pathogens: Arina/Forno with *S. tritici*, *S. nodorum*, *F. culmorum* and History/Rubens with *S. tritici* and *F. culmorum*. To our knowledge, this is the first paper in which a meta-analysis has been performed with raw data from several studies with the same progeny in adult-plant stage. The maps for the resistance combinations are, therefore, exactly the same, and this offers a good chance to detect BSR QTL by adding the LOD values using markers with similar positions. This procedure also equalizes heterogeneous error variances caused, for example, by highly differing resistance levels at different locations and/or years. Results are more critical when comparing data from different maps or using consensus maps with only a limited number of common markers. For exactly this reason, a meta-analysis across the two populations was not possible.

For STB resistance, 5 QTL were found in each of the populations, explaining 3–24% of genotypic variance. In total, six of them reacted differently according to the environment, thus, supporting the high proportion of genotype–environment interaction variance relative to genotypic variance (Electronic Supplementary

Material Table S1). In a previous study with two other elite winter wheat populations, a similar number of QTL was found for STB resistance (Risser et al. 2011). It can be concluded that this disease resistance in the two populations follows a typical quantitative inheritance (Electronic Supplementary Material Fig. S1) with several loci each having only a small effect. The *Stb6* and *Stb15* loci were not detected in our analysis because the isolates used seem to be virulent for both genes in the adult-plant stage.

On chromosome 5B, a single QTL for STB resistance was located close to *Xgwm274* in Arina/Forno (*QStb.lsa_af-5B*) and History/Rubens (*QStb.lsa_hr-5B.1*), indicating a QTL effective across both populations. Because of too few common markers between the two mapping populations at the respective QTL intervals, it was not possible to generate a consensus map. STB QTL in similar genomic positions to ours were previously detected on chromosomes 2B and 3B (Florett/Biscay), on chromosome 6B (Tuareg/Biscay), and even three QTL on chromosome 7B in Tuareg/Biscay, History/Rubens, and Arina/Forno (Risser et al. 2011). Again, a critical comparison of these QTL for

Fig. 1 LOD curves of BSR QTL on chromosomes 3B, 4B, 5B, and 6D in Arina/Forno for *Fusarium* head blight (*FHB*), *Stagnaspora* glume blotch (*SGB*), and *S. tritici* blotch (*STB*) detected by meta-analysis; LOD *curves* for plant height (*PLH*) and heading date (*HED*) are given for comparison; *rectangles with line inside* show support intervals and positions of meta-QTL; initial QTL in *brackets*
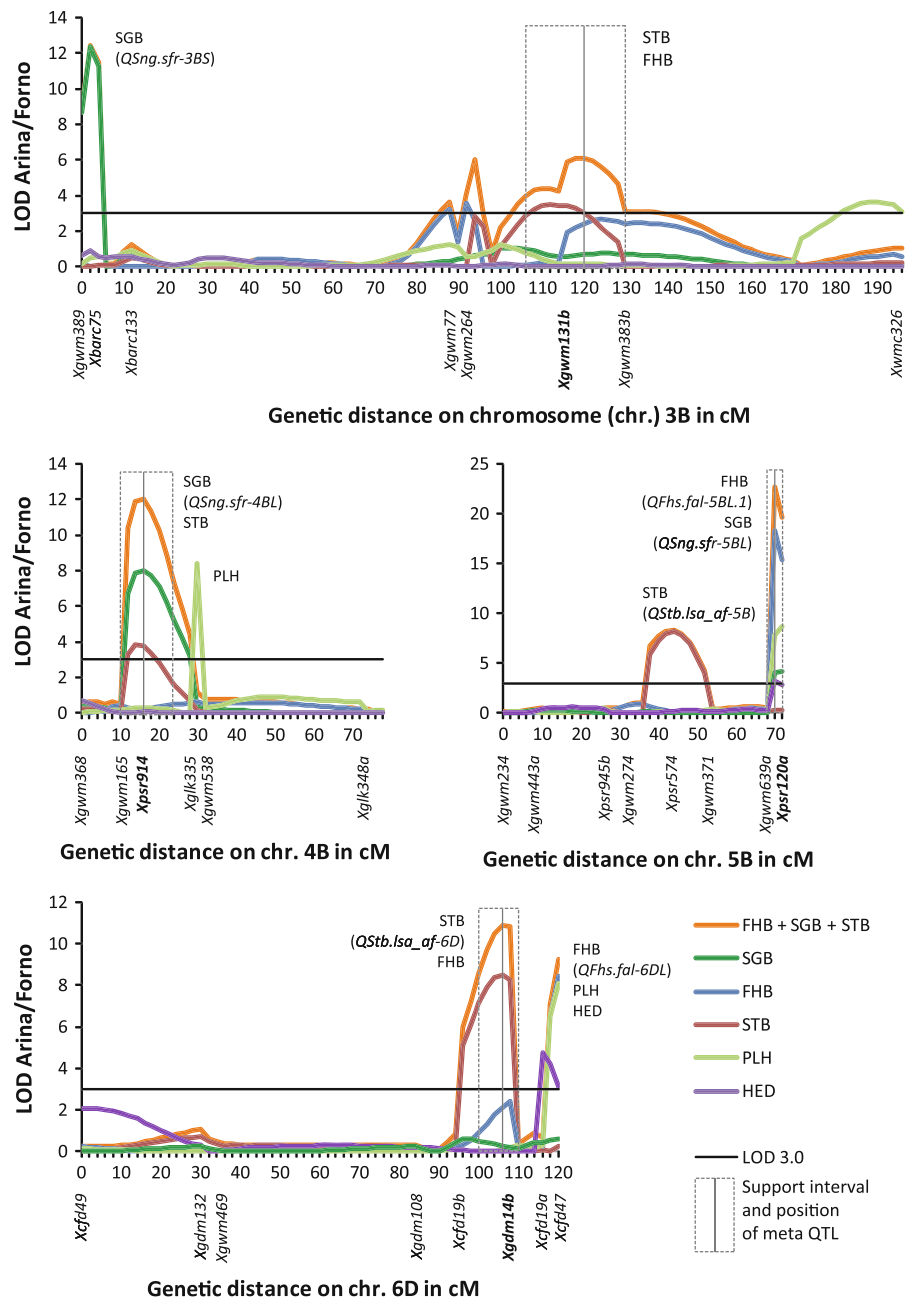
similarity was not possible due to missing common markers in the QTL regions. Eriksen et al. (2003) mapped QTL for adult-plant STB resistance in Savannah/Senat on chromosomes 2B, 3A, 6B, and 7B, altogether explaining 62–77% of phenotypic variance. SSR markers *Xwmc344* on chromosome 2B close to *QStb.lsa_fb-2B* and *Xwmc517* on chromosome 7B close to *QStb.lsa_tb-7B* were, however, the only

markers in accordance to the map of Savannah/Senat. Support intervals of resistance QTL did not include the common markers indicating independent QTL. Chartrain et al. (2004) detected one QTL (*QStb.psr-6B-1*) explaining 24% of phenotypic variance from the susceptible parent Riband in polytunnel trials at the adult-plant stage. The position of this QTL was close to SSR marker *Xgwm219*, which is also present in

Fig. 2 LOD curves of BSR QTL on chromosomes 2B, 4D, and 7B in History/Rubens for *Fusarium* head blight (*FHB*) and *S. tritici* blotch (*STB*) detected by meta-analysis; LOD *curves* for plant height (*PLH*) and heading date (*HED*) are given for comparison; *rectangles with line inside* show support intervals and positions of meta-QTL; initial QTL in *brackets*
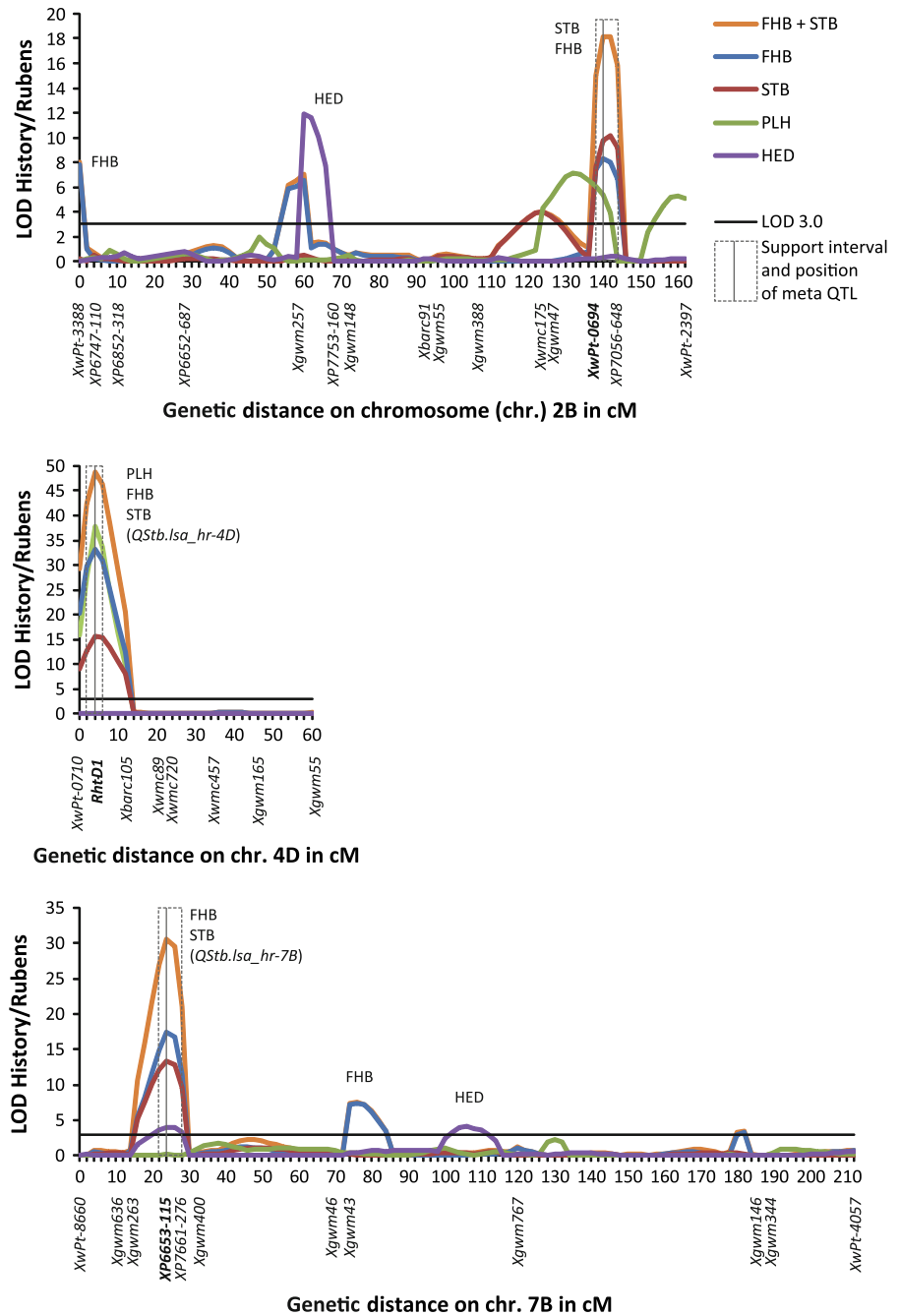
History/Rubens, but the peak of the QTL from the present study was about 25 cM distant from this marker. In conclusion, one and three major QTL were detected in Arina/Forno and History/Rubens, respectively, that are worth further validation for use in practical breeding.

The large effect on FHB resistance of the QTL on chromosome 4D of History/Rubens matching the dwarfing locus *Rht-D1* has been extensively described in recent years in several populations (e.g., Voss et al. 2008; Holzapfel et al. 2008; Srinivasachary et al. 2008). A similar effect of *Rht-D1* was previously detected in other wheat populations (Draeger et al. 2007; Gosman et al. 2007) and very recently retrieved by genome-wide association mapping (Miedaner et al. 2011). Until now, it was not possible to clarify whether

the combination of tallness and FHB resistance at this locus is due to pleiotropy or linkage. In this study, we first describe an effect of *Rht-D1* locus on STB resistance as being of the same magnitude as reported for FHB resistance. Of course, the locus was also detected as a meta-QTL with the tall allele causing "resistance" to both diseases. Comparing the additive effects, they were even larger for STB than for FHB resistance. This partly explains the phenotypic correlation between STB resistance and plant height in this population. Another QTL for plant height was located near the BSR QTL for STB and FHB resistances on chromosome 2B (see Fig. 2). It is, however, not recommended to use the tall allele of *Rht-D1* in practical breeding as a resistance donor, because the resistance of this allele may simply reflect the absence of the dwarfing allele and not inherit a resistance mechanism of its own. In a study with near-isogenic lines, however, Simón et al. (2004) did not find any effect of *Rht-D1b* from Norin 10 on STB severity. Only the extremely height-reducing genes *Rht-B1c* and *Rht12* had a significant ($P < 0.05$) effect in their study. Similarly, Chartrain et al. (2004) did not find this relationship in the Arina × Riband population.

Meta-analysis by the program PLABMQTL revealed a total of 7 BSR QTL with significant additive effects for each of two diseases, with 5 BSR QTL being highly significant ($P < 0.01$) due to critical LOD scores assessed by 1,000 permutations. The highly differing critical LOD scores between the two populations are mainly due to the different population sizes. The BSR QTL on chromosome 3B (Arina/Forno) showed a peak with similar map position for each of the two disease resistances in the initial QTL analysis, but LOD values were not significant in the primary analysis and were consequently detected only at $P < 0.25$ in the meta-analysis.

Four significant ($P < 0.01$) BSR QTL for different disease combinations were detected besides the *Rht-D1* locus: three on the B genome and one on the D genome. Initial QTL in Arina/Forno of Schnurbusch et al. (2003) for SGB and of Paillard et al. (2004) for FHB as well as in History/Rubens of Holzapfel et al. (2008) for FHB were confirmed by reanalyzing raw data with PLABMQTL. In Arina/Forno, BSR QTL were detected across each of two fungal pathogens, but not across all three pathogens. However, positive side effects of BSR QTL to the third pathogen exist, as shown for chromosome 5B, where STB infection is reduced by selecting the resistant allele effective for resistance to FHB and SGB (Table 2). Interestingly, the resistance locus *Sng.sfr-3BS* for *S. nodorum* resistance that is widespread in European elite winter wheat (Tommasini et al. 2007) resides in the same region as *Fhb1* of Asian origin for FHB resistance (Löffler et al. 2009) together with four race-specific resistances to biotrophic pathogens. In fact, there is increasing evidence for the existence of BSR QTL, because they were detected independently from the crop (e.g. *Arabidopsis*, maize, rice, wheat) and the pathosystem (e.g., STB, FHB, SNG, tan spot in wheat). The main question is whether this reflects just a random colocalization of resistance genes in gene-rich genomic regions or the action of the same gene on different pathogens. Poland et al. (2009) discussed several hypotheses for explaining the potential mechanisms underlying BSR QTL. The most probable hypothesis for the pathosystems analyzed in this study is their involvement in basal defense reaction or even non-host resistance and/or defense signal transduction. Because the pathogens analyzed are not genetically related and infect different plant organs (flag leaf vs. head) in different adult-plant stages (flag leaf extension vs. flowering), the hosts' resistance reaction might be triggered by widely conserved pathogen elicitors. Molecular studies in *Arabidopsis* support the hypothesis that pattern-recognition receptors can condition quantitative differences in resistance to several pathogens (Miya et al. 2007; Wan et al. 2008). In future analyses, synteny relations will promote detection of BSR QTL across crops also. Head blight in wheat and ear rot in maize, both caused by *F. graminearum*, might be a promising model.

In conclusion, meta-analysis is an useful tool for detecting common regions for quantitative disease resistances, especially when based on the same maps. By using original resistance scoring data, we detected a total of 5 significant ($P < 0.01$) BSR QTL across each of two pathogens in the adult-plant stage, a fact that has not been shown before in wheat. Closely linked markers are available, and small support intervals of some BSR QTL indicate high accuracy of the meta-QTL positions. Further research is required to validate STB QTL with major effects as well as BSR QTL in different genetic backgrounds. As all three resistances are mainly affected by additive effects, the combination of major QTL in

breeding material seems promising and breeders should concentrate on those QTL that reveal BSR.

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