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Genetic architecture of resistance to *Septoria tritici* blotch (*Mycosphaerella graminicola*) in European winter wheat

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Abstract Genome-wide marker–trait associations (MTA) were established in a population of 358 European winter wheat cultivars and 14 spring wheat cultivars (*Triticum aestivum* L.) for resistance to *Septoria tritici* blotch caused by the fungal pathogen *Mycosphaerella graminicola*. The MTA were based on field data in two consecutive years and genotypic data on 732 microsatellite markers. Best linear unbiased estimations (BLUEs) for resistance were calculated across the trials and ranged from 0.67 (most resistant) to 19.63 (most susceptible) with an average value of 4.93. A total of 115 MTA relating to 68 molecular markers was discovered for the two trials and BLUEs by using a mixed linear model corrected by a kinship matrix. In addition, two candidate genes, *Ppd-D1* for photoperiodism and the dwarfing gene *Rht-D1*, were significantly associated with resistance to *Septoria tritici* blotch. Several MTA co-located with known resistance genes, e.g. *Stb1, 3, 4, 6* and *8*, while multiple additional MTA were discovered on several chromosomes, such as 2A, 2D, 3A, 5B, 7A and 7D. The results provide proof of concept for the method of genome-wide association analysis and indicate the presence of further *Stb* resistance genes in the European winter wheat pool.

Keywords *Septoria tritici* blotch · *Triticum aestivum* · Association mapping · *Stb* genes · *Mycosphaerella graminicola*

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Introduction

Septoria tritici blotch (STB) caused by *Mycosphaerella graminicola* (anamorph *Septoria tritici*) is one of the most important foliar diseases of winter wheat (*Triticum aestivum* L.) in Europe and in wheat-growing areas worldwide. Strobilurin fungicides or quinone outside inhibitors (QoIs) have been successfully used to control *Septoria* leaf blotch, but natural QoI-resistant variants caused by a point mutation in the cytochrome *b* allele of *M. graminicola* have

evolved in field populations (Fraaije et al. 2005). The breeding of genetically resistant cultivars is therefore still a major task in controlling the disease.

During recent years a number of major *Stb* resistance genes have been mapped in various bi-parental mapping populations (for review see Goodwin 2007). The *Stb6* resistance gene was associated with a significant reduction in STB in 226 wheat lines, while disease escape mechanisms, like plant height, leaf spacing, leaf morphology and heading date, have also played a role in avoidance of the disease (Arraiano et al. 2009). As well as the reports on mapping single *Stb* genes (Arraiano et al. 2001, 2007; Adhikari et al. 2004a, b, c; Brading et al. 2002; Chartrain et al. 2005a, b, 2009; McCartney et al. 2003; Ghaffary et al. 2012), several reports on mapping quantitative trait loci (QTL) for STB resistance in bi-parental mapping populations have emerged recently (Chartrain et al. 2004; Simón et al. 2004b; Risser et al. 2011; Kelm et al. 2012; Miedaner et al. 2012).

In this report we describe the application of a genome-wide association study (GWAS) for 358 recent European winter wheat varieties and 14 spring wheat cultivars in order to assess the genetic architecture of STB resistance in the spectrum of cultivars. While QTL mapping in bi-parental populations only represents the genetic repertoire of two accessions, GWAS is suitable for monitoring a broad spectrum of cultivars or accessions (Zhu et al. 2008). Additionally, more meiotic events that have taken place during evolution or cultivar development are taken into account compared to bi-parental populations, resulting in an increased genetic resolution and significance of the linkage disequilibrium (LD) of the species under investigation (Hamblin et al. 2011). There are a number of reports on GWAS in wheat for various traits including yield and agronomic traits (Neumann et al. 2011; Reif et al. 2011a; Wang et al. 2012), baking and milling quality (Breseghello and Sorrells 2006; Reif et al. 2011b; Bordes et al. 2011), ear emergence (Le Gouis et al. 2012), pre-harvest sprouting (Kulwal et al. 2012) and resistance to pathogens (Crossa et al. 2007; Maccaferri et al. 2010; Miedaner et al. 2011; Yu et al. 2011, 2013). Our set of cultivars was investigated in a parallel study on resistance to *Fusarium* head blight (Kollers et al. 2013).

The goal of the current study was (1) to assess a selection of 372 wheat cultivars for field resistance to STB in two environments, (2) to establish marker–trait associations for resistance to STB based on genome-wide coverage using 732 simple sequence repeat (SSR) markers plus markers for candidate genes, and (3) to compare the results obtained with the chromosomal locations of known *Stb* resistance genes and QTL, in order to test the hypothesis that association mapping is suitable for providing a comprehensive overview of the genetic architecture of STB resistance in recent European winter wheat cultivars and for detecting markers linked to resistance loci.

Materials and methods

Plant material, field trials and disease evaluation

A total of 358 European winter wheat cultivars and 14 spring wheat cultivars was evaluated in this study. Spring and winter wheat cultivars were sown at the same time. All varieties were grown in Cecilienkoog, Germany, in the years 2009 and 2010 using an incomplete alpha-block design with three replications per year. Each year was regarded as one environment.

The spray inoculation was performed with a spore suspension of 5×10^6 pycnidio spores/ml using a water application rate of 600 L/ha. The inoculation was performed twice at an interval of 10 days. The first inoculation was made at growth stage GS 39/41 (emerged flag leaf). To increase the risk of infestation, in plant stage GS 31/32 *Septoria*-infected grains were distributed on each plot at a density of 25 g/m^2. Visual assessments of first leaves and flag leaves were performed 32 and 48 days after spray inoculation. The arithmetic means for three replications, two assessments and two kinds of leaves were calculated as the phenotypic resistance score to STB for each environment.

The cultivars were also evaluated for heading date and plant height in a companion study in eight locations over the same two seasons.

Molecular data analysis

The 372 cultivars were genotyped with 732 microsatellite markers using standard protocols on capillary sequencing machines. Of the microsatellite markers tested, 48 amplified more than one locus, resulting in 782 loci spread across the 21 chromosomes. Heterozygotes comprised 2.6 % of the data. With 4.8 %

missing data points, 276,844 datapoints (95 %) were available for the association analysis.

Map positions on the ITMI mapping population (International Triticeae Mapping Initiative) were determined with the programme MAPMAKER v.3.0 using the Kosambi mapping function with a LOD score of 3.0 as the threshold for linkage. With this, 620 markers were placed on the ITMI map with 19 markers mapping to more than one position in the genome, resulting in a map of length 4,470 cM. This resulted in an average marker distance of 7.2 cM (ranging from 4.6 cM on chromosome 4B to 10.2 cM on chromosome 6A).

All the cultivars were additionally genotyped for candidate genes *Rht-B1* and *Rht-D1* (Ellis et al. 2002) and the *Ppd-D1a* allele of the photoperiod response locus *Ppd-D1* (Beales et al. 2007). The candidate genes were not scored in the ITMI population and are therefore not included in the ITMI map. Their approximate location is found in Pestsova and Röder (2002) for gene *Ppd-D1* on chromosome 2DS and in Börner et al. (1997) for genes *Rht-B1* and *Rht-D1* on chromosomes 4BS and 4DS.

Population structure was inferred with a principal coordinate analysis based on modified Rogers' distance (Wright 1978) which was calculated with a subset of 155 loci. Principal coordinate analysis was performed in R software using the function cmdscale. The 155 markers were chosen to be distributed across the genome and based on reliability as having the lowest number of missing data points, no null alleles and lowest number of heterozygotes. A kinship matrix was calculated using the software SPAGeDi (Hardy and Vekemans 2002) based on the aforementioned 155 markers. Negative values were set to 0.

Results concerning population structure and linkage disequilibrium of this mapping population have been described earlier (Kollers et al. 2013).

Statistical analysis and association mapping

Best linear unbiased estimations (BLUEs) were calculated across the phenotypic data of both environments using the "Mixed models REML" module and the "Linear mixed models" of the software package GenStat 14th edition (VSN International Ltd, UK).

Marker–trait associations were calculated separately for each environment and the BLUEs. In GenStat the "QTL analysis" module and the "Single trait association analysis" function were utilized and the kinship matrix was chosen as the relationship model. For the calculation of genotype–phenotype associations, microsatellite data were converted into a bi-allelic data format resembling single nucleotide polymorphism data. A minor allele frequency threshold of 3 % (equalling 11 varieties) was set and alleles with a lower frequency were excluded from the analysis. After the filtering process, 3,176 alleles remained and were employed for the association mapping approach. MTA were considered as significant with $-\log_{10}(P \text{ value}) > 3.0$ and the Bonferroni correction resulted in a significance threshold of $-\log_{10}(P \text{ value}) > 4.82$.

Results

Description of phenotypic data

Resistance scores to *Septoria tritici* for 358 European winter wheat cultivars plus 14 spring wheat cultivars were based on field trials in 2 years. The resulting BLUEs ranged from 0.67 to 19.63 with an average value of 4.93 (Fig. 1). A total of eight cultivars had BLUEs ≤ 1.0; the two cultivars Julius and Solitär were the most resistant with BLUEs of 0.67 and 0.83, respectively (Supplemental file 1). The correlation coefficient of resistance scores between the two environments was moderate with $R = 0.659$, while the correlations with the BLUEs were higher with 0.859 and 0.943 for 2009 and 2010, respectively. The ANOVA indicated significant genotype as well as environmental effects (Supplemental file 2).

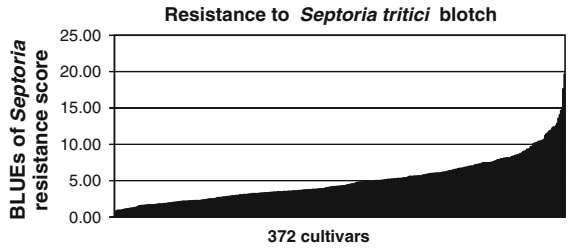


Fig. 1 Phenotypic distribution of resistance scores to *Septoria tritici* blotch in 372 cultivars. The best linear unbiased estimations (BLUEs) were based on resistance tests in two environments. A low score indicates high resistance

Marker–trait associations

Marker–trait associations (MTA) were calculated for each environment and the BLUEs based on the data of 782 microsatellite loci covering the whole genome by using a mixed linear model including the kinship matrix. Additionally, the genotyping data for the photoperiodism gene *Ppd-D1* and the dwarfing genes *Rht-D1* (formerly called *Rht2*) and *Rht-B1* (formerly called *Rht1*) were tested for association with *Septoria tritici* resistance scores. In total, 115 association events with $-\log_{10}(P \text{ value}) > 3.0$ were detected for the microsatellite loci and the candidate genes (Table 1; Supplemental file 3); 44 MTA were based on the BLUEs. A decreasing mean additive effect (resulting in an increased resistance) was detected for 36 of the individual MTA, while an increasing additive effect (resulting in decreased resistance) was found for 79 MTA. The MTA detected related to 68 individual microsatellite loci, of which 48 loci were integrated into the ITMI map. For the BLUEs, 39 microsatellite loci were significant. Most microsatellite loci detected MTA in only one of the two environments including BLUEs (Supplemental file 4); however, multiple MTA were detected with some loci, such as five MTA with GWM391 on chromosome 3AL, four MTA for GWM1391 on chromosome 6DS and six MTA for marker BARC182 on chromosome arm 7BL (Fig. 2). In these cases more than one allele of the multi-allelic microsatellite markers was significant with sometimes contrasting positive or negative additive effects.

Marker GWM 369 was reported to be closely linked to resistance gene *Stb6* (Brading et al. 2002). We had included this marker in our analysis, but of 22 alleles detected only three were above the MAF of 3 %. None of these alleles was significant; a significant effect was observed for allele GWM369_247 bp which was only present in one cultivar (Supplemental file 5).

In addition to the microsatellite loci, the *Ppd-D1* marker was significant in both environments and the BLUEs. In all cases a positive additive effect was found for the *Ppd*-insensitive allele resulting in decreased resistance, while for the *Ppd*-sensitive allele a negative additive effect was found resulting in increased resistance (Supplemental file 3). Three MTA were significant for the dwarfing gene *Rht-D1*; here the mutant allele had a positive additive effect, while the wild type showed negative additive effect. These data are in agreement with negative Spearman rank correlations for plant height (−0.269) and heading date (−0.241) with the scores for BLUEs of *Septoria* resistance. This means taller and later flowering cultivars had less disease at the time scored.

Table 1 Statistics of detected marker–trait associations

Environments	$-\log_{10}(P \text{ value}) > 3.0$	$-\log_{10}(P \text{ value}) > 4.82$
2009.CEC	36	6
2010.CEC	35	4
BLUEs	44	4
Sum	115	14
Total of different marker loci	68	9

Additive effects of favourable and unfavourable alleles

Each cultivar carries a combination of favourable and unfavourable alleles. The number of favourable alleles per cultivar (excluding the tested candidate genes) ranged from two to 18, with the highest number of observed events at six favourable alleles per cultivar (Fig. 3a). The number of unfavourable alleles per cultivar ranged from one to 30, with the highest number of observed events at seven (Fig. 3b). The two most resistant cultivars, Julius and Solitär, carried 13 and 14 favourable alleles, respectively, and each of them had two unfavourable alleles. The Spearman rank correlation between the number of favourable alleles per cultivar and its BLUEs for resistance to STB was −0.614, while the correlation of BLUES with the number of unfavourable alleles per cultivar was +0.56. This means cultivars with more favourable and fewer unfavourable alleles were more resistant.

Linear regression showed a dependence of BLUEs for resistance to STB on number of favourable alleles per cultivar with $R^2 = 0.325$ and $Y = 9.45 - 0.583X$ (Fig. 4a), while the relationship between the number of unfavourable alleles per cultivar and BLUEs was $Y = 2.03 + 0.271X$ with $R^2 = 0.334$ (Fig. 4b). These results indicated that, to a certain degree, the effects of favourable or unfavourable alleles are additive. Pyramiding of favourable alleles and avoidance of unfavourable alleles during the breeding process may therefore lead to more resistant cultivars.

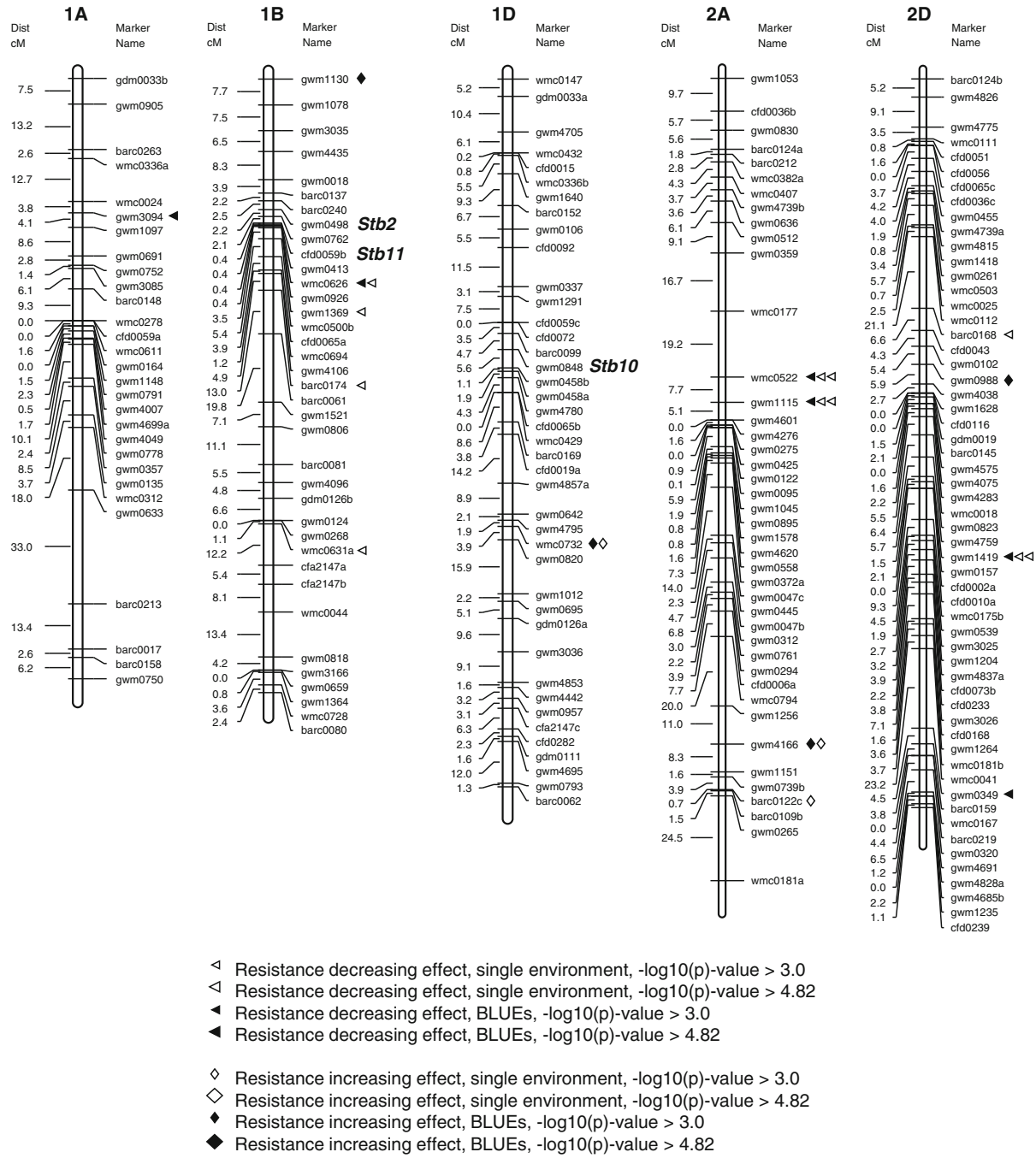
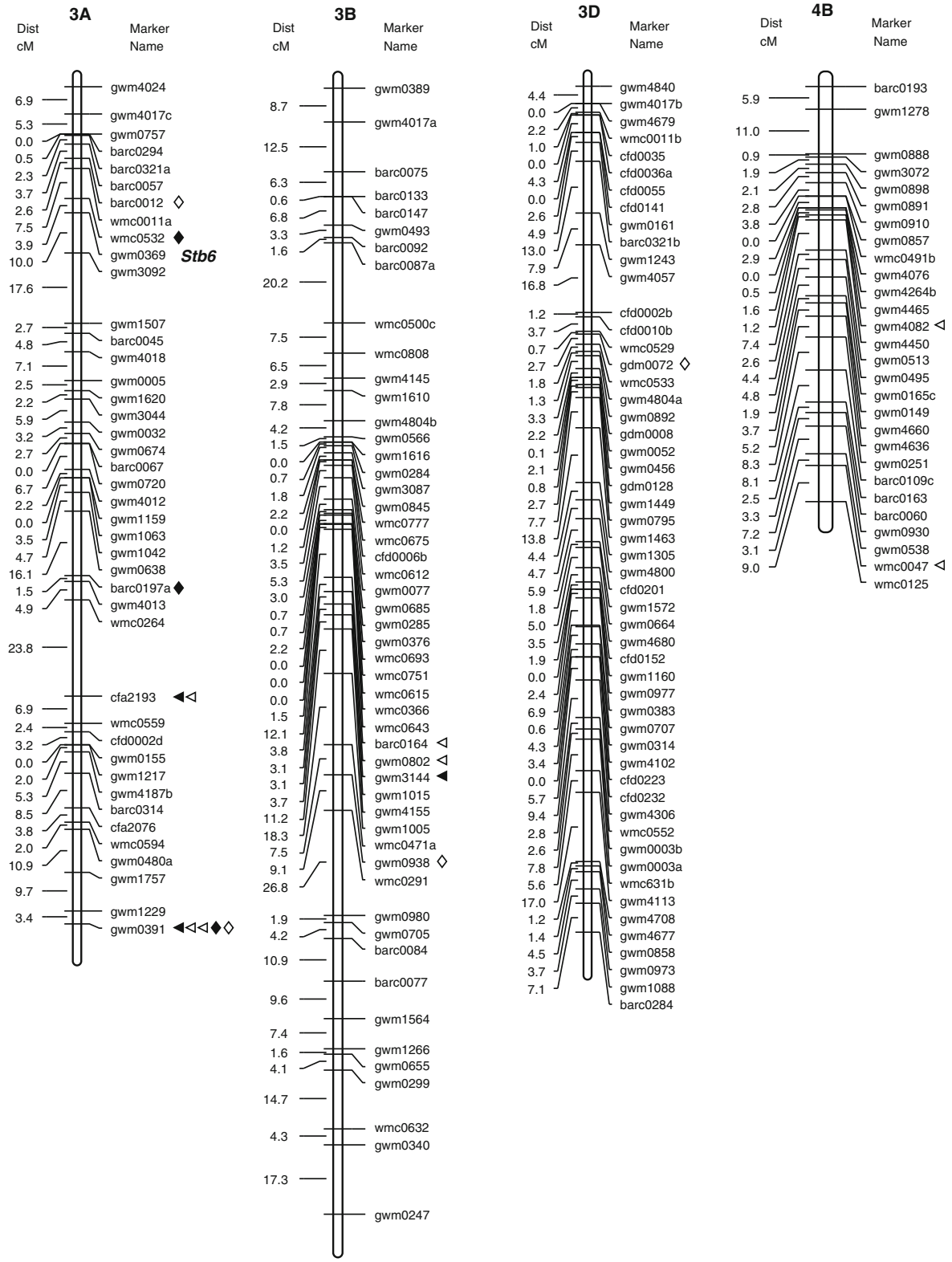


Fig. 2 Chromosomal location of significant marker–trait associations for *Septoria tritici* blotch. The locations of known *Stb* resistance genes are indicated

Discussion

Our strategy and experimental setup allowed the detection of numerous marker–trait associations for resistance to *Septoria tritici* blotch. In total, 68 microsatellite loci detected significant marker–trait associations and indicated a quantitative pattern of inheritance for resistance to STB, though several



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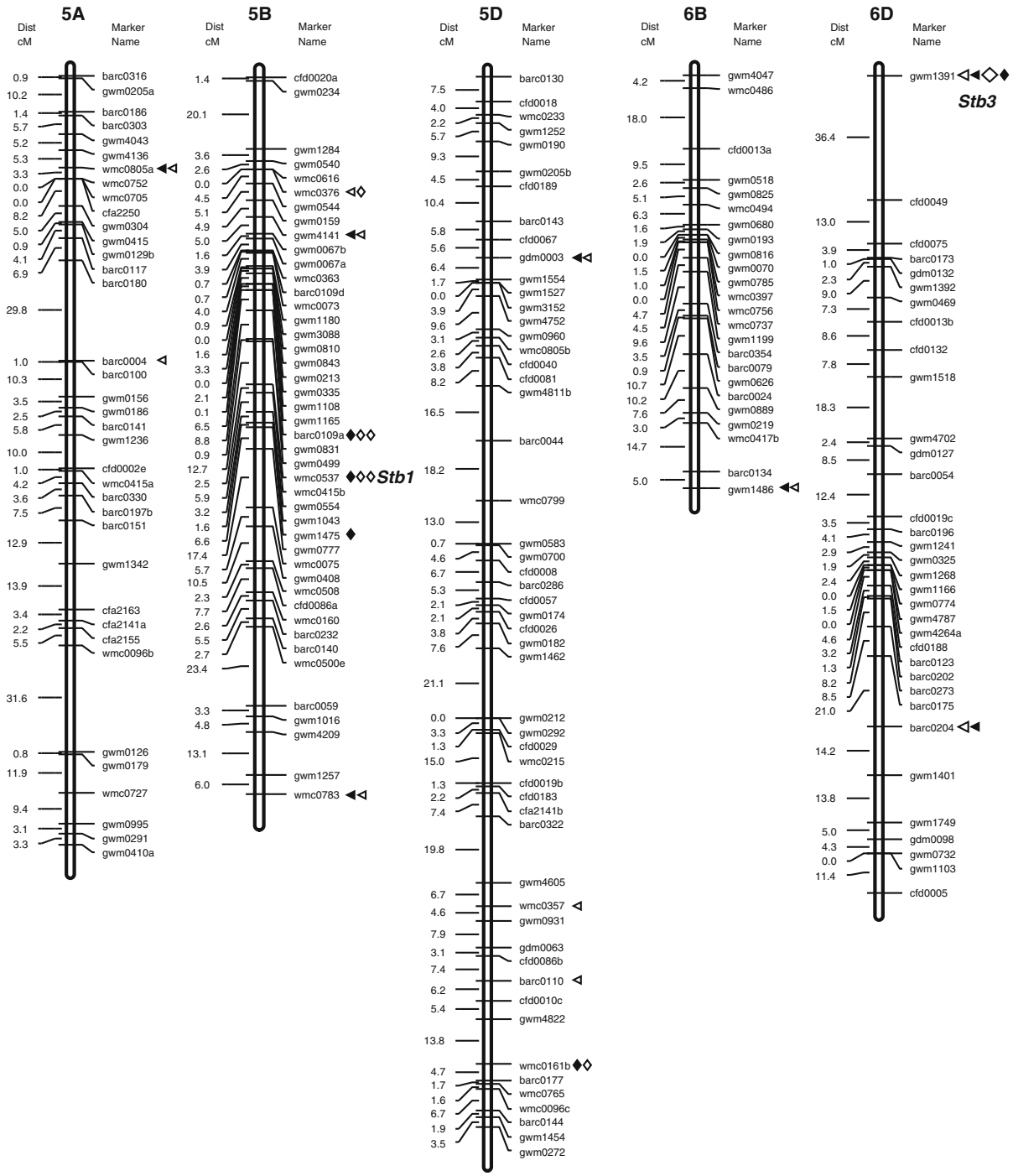

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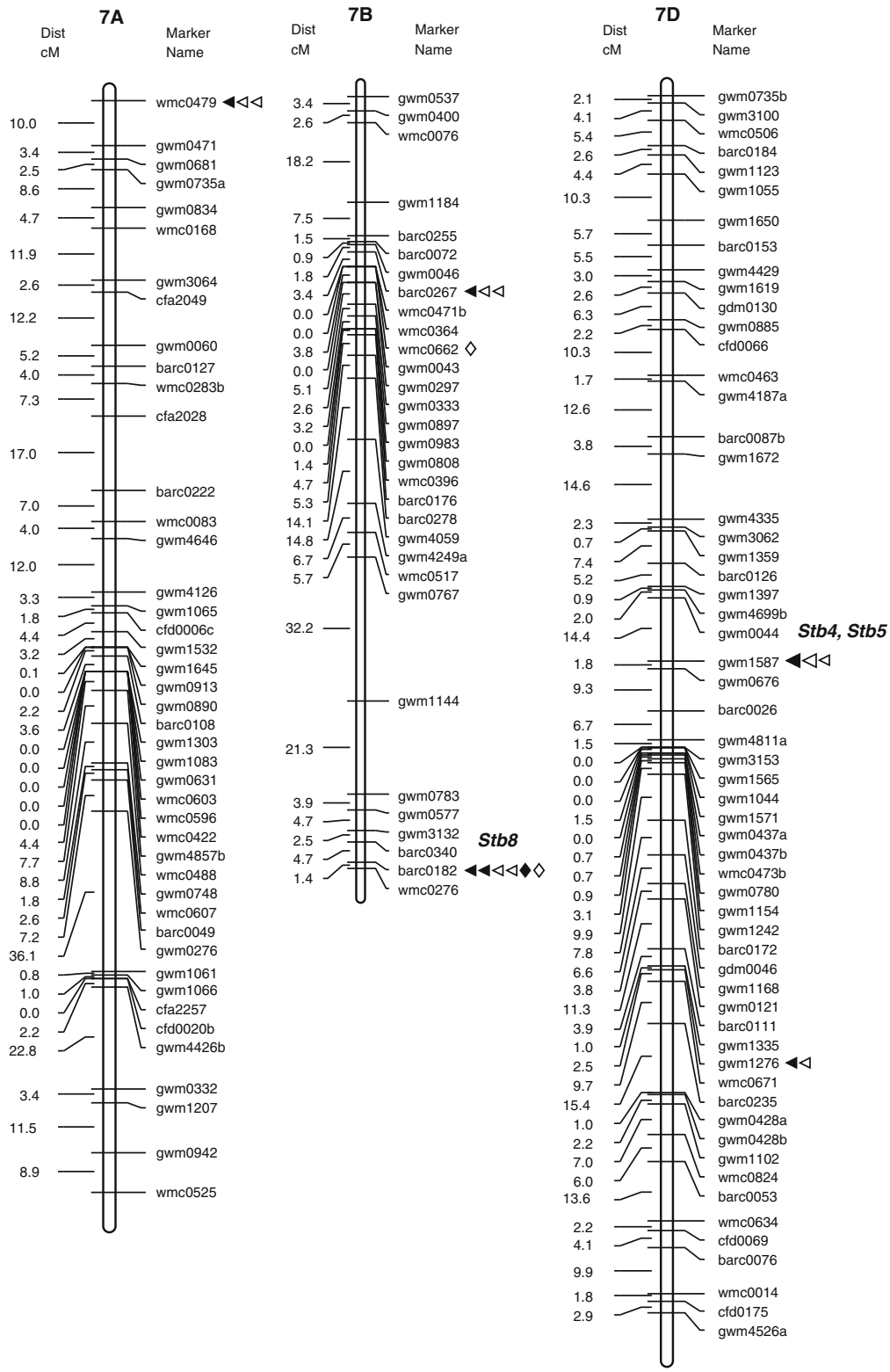

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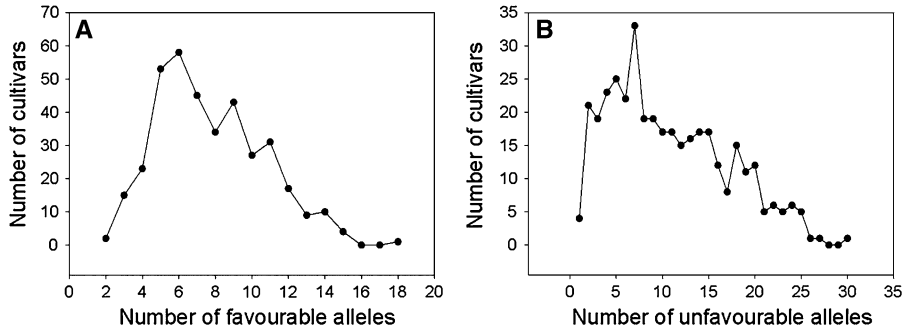

Fig. 3 Frequency of **a** favourable or **b** unfavourable alleles for *Septoria tritici* blotch resistance in individual cultivars

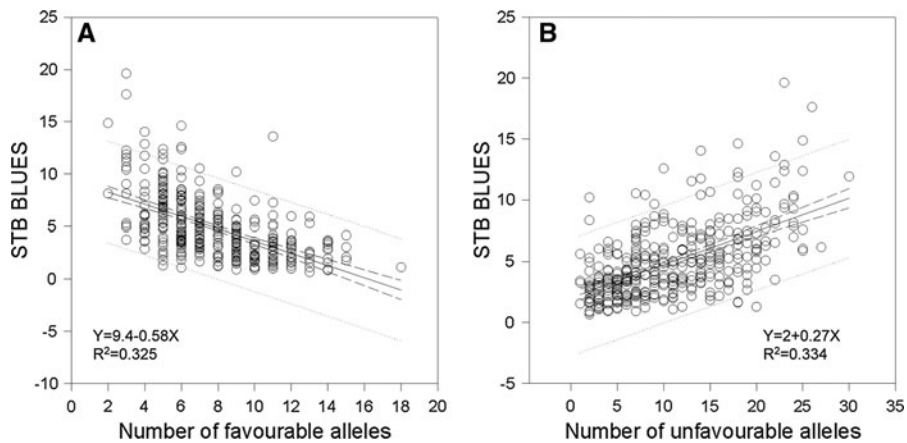

Fig. 4 Linear regression of best linear unbiased estimations (BLUEs) for *Septoria tritici* blotch (*STB*) resistance to **a** number of favourable and **b** number of unfavourable alleles per cultivar

major resistance genes have been described in the literature.

The *Stb6* gene which originated from the cultivars Flame and Hereward was reported to be closely linked to marker GWM369 (Brading et al. 2002) and it was shown to be present in many cultivars and landraces worldwide (Chartrain et al. 2005b). We observed one MTA for marker WMC532 which neighbours GWM369. In addition, in the bi-parental mapping studies, Kelm et al. (2012) and Eriksen et al. (2003) reported a major QTL on chromosome arm 3AS where GWM369 is located.

Marker GWM1391 detected four MTA in our study. This marker is located on chromosome arm 6DS at a distance of 2.5 cM from GDM132 (Pestsova et al. 2000; Ganal and Röder 2007), which is reported to be linked with *Stb3* (Adhikari et al. 2003). While the location of *Stb3* on chromosome 6DS was questioned by Goodwin (2007), our data provide good evidence for the presence of a resistance factor to *Septoria tritici* on chromosome 6DS.

On chromosome 7D, three MTA were detected near locus GWM1587. Two resistance genes were reported in this chromosomal region: *Stb5* originating from synthetic wheat (Arraiano et al. 2001; Simón et al. 2007) and *Stb4* originating from cultivar Tadinia (Adhikari et al. 2004a). While *Stb5* was mapped distal to marker GWM44 (Arraiano et al. 2001; Simón et al. 2007), *Stb4* was located 0.7 cM distal to GWM111 (Adhikari et al. 2004a). In Ganal and Röder (2007), GWM1587 was located in a similar region distal to GWM111, but proximal to GWM44, and therefore the detected QTL may represent *Stb4*.

For the distal end of chromosome 7BL, marker BARC182 detected six MTA. In this chromosomal region *Stb8* was mapped between markers GWM577 and GWM146 (Adhikari et al. 2003). In our map GWM577 is located ca. 12 cM proximal to BARC182; however, *Stb8* could still be considered as a candidate gene for the observed MTA, since the distal chromosomal regions are usually rich in recombination events.

On chromosome 1B, marker WMC626 detected two MTA for *Septoria* resistance. Resistance gene *Stb11* was linked to marker BARC008 on this chromosome (Chartrain et al. 2005c). BARC008 was located on the short arm of chromosome 1B, while WMC626 was mapped to the long arm of 1B though close to the centromere (Somers et al. 2004). The detected MTA is therefore most likely not identical to *Stb11*. Recently, resistance gene *Stb2* was re-located on chromosome 1B; it mapped on the short arm in a similar region to *Stb11* (Liu et al. 2013). On chromosome 1B, a QTL linked to WMC419 was also reported for the mapping population Florett × Biscay (Risser et al. 2011). WMC419 was located in the centromeric region of chromosome 1B in the interval between BARC008 and WMC626, and therefore the indicated QTL region may be identical to the MTA detected by WMC626.

On chromosome arm 5BL, three MTA were found for marker WMC537. In this region resistance gene *Stb1* was mapped ca. 7.4 cM distal to markers GWM213 and GWM335 (Adhikari et al. 2004c). In our map, WMC537 was located at a distance of ca. 28 cM distal to GWM213 and GWM335, thus it can neither be excluded nor confirmed whether the MTA detected by WMC537 is based on *Stb1*. In this genomic region, Miedaner et al. (2012) also reported a meta-QTL for resistance to STB in the interval GWM371 to GWM274. Marker GWM371 maps close to GWM831 in the map of Ganal and Röder (2007), while GWM831 is 9.7 cM proximal to WMC537; it could therefore be the same QTL.

On chromosome arm 1AS, Kelm et al. (2012) reported a QTL for necrotic leaf area in the interval GWM1223 to GWM1097, which covers the genomic region of WMC336. Risser et al. (2011) also reported a QTL linked to WMC0024 for the population Tuareg × Biscay. WMC0024 is the next marker to GWM3094, which detected a MTA in our map.

Three linked markers on chromosome 3B, BARC164, GWM802 and GWM3144, detected MTA. This region coincides with a QTL linked to GWM131b in the Arina × Forno population (Miedaner et al. 2012). Another QTL in this population reported for the interval GWM274 to GWM371 on chromosome 5BL (Miedaner et al. 2012) coincides with three MTA discovered for marker BARC109. GWM 371 and BARC109 are both closely linked to GWM831 (Ganal and Röder 2007). The QTL of the History × Rubens population (Miedaner et al. 2012), flanked by markers GWM263 and GWM400 on chromosome 7B, appears to be located more distal than the MTA discovered by marker BARC267 on the same chromosome in our study. The MTA discovered for marker GWM1369 on chromosome 1B may coincide with the QTL linked to GWM752 in the Solitär × Mazurka population (Kelm et al. 2012). Both markers are located proximal to GWM11.

In several cases single markers detected MTA in at least three environments or BLUEs, such as WMC522 and GWM1115 on chromosome 2AS, GWM1419 on chromosome 2D, GWM391 on chromosome 3AL, BARC109 on chromosome 5B, WMC479 on chromosome 7AS and BARC267 on chromosome 7B. Major *Stb* genes have not yet been reported for any of these regions. The current results may therefore indicate the presence of further as-yet-undescribed *Stb* genes in the germplasm. Our results also provide additional information about novel markers linked to known *Stb* genes, such as GWM1391 for *Stb3,* GWM1587 for *Stb4*, and possibly BARC182 for *Stb8* and WMC537 for *Stb1*.

A negative correlation of STB resistance with plant height indicated that taller cultivars tended to be more resistant. These results are in accordance with the observed MTA for the dwarfing gene *Rht1-D1*, with the wild-type allele increasing the resistance. Plant height was described, along with other traits concerning plant architecture such as leaf spacing and leaf prostrateness, as disease-escape mechanisms for STB (Arraiano et al. 2009), but also for other fungal diseases, such as *Fusarium* head blight (Miedaner and Voss 2008; Srinivasachary et al. 2008). Simón et al. (2004a) also reported a negative correlation between plant height and necrosis percentage for STB.

As well as plant height, a significant correlation was detected for heading date, with later flowering cultivars being more resistant, which was in accordance with the observations of Arraiano et al. (2009). A strong MTA was observed with the photoperiodism gene *Ppd-D1* on chromosome 2DS, with the *Ppd*-sensitive allele increasing resistance. A significant QTL for resistance to STB as well as heading date in the respective region was described for the Balance × Apache population (Ghaffary et al. 2011), though the authors did not consider the possible influence of *Ppd-D1* in their population. Simón et al. (2004a) reported a positive correlation between heading date and observed necrosis values, which is in

contradiction to our results; however, the authors point out the strong influence of climatic conditions on the disease severity.

Our results provide an overview of the resistance spectrum for *Septoria tritici* blotch present in European winter wheat cultivars. Besides the known genes and QTL, several significant marker loci indicated the presence of as-yet-undetected resistance genes and QTL. Furthermore, the association mapping approach resulted in the identification of new markers for known as well as unknown *Stb* genes for marker-assisted selection during the breeding process. Additive effects of the numbers of favourable or unfavourable alleles per cultivar indicated that a breeding strategy of pyramiding favourable alleles and avoiding unfavourable alleles may increase field resistance to *Septoria tritici* blotch in a cultivar.

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