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ORIGINAL PAPER

QTL for spot blotch resistance in bread wheat line Saar co-locate to the biotrophic disease resistance loci *Lr34* and *Lr46*

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Abstract Spot blotch caused by *Bipolaris sorokiniana* is a major disease of wheat in warm and humid wheat growing regions of the world including south Asian countries such as India, Nepal and Bangladesh. The CIMMYT bread wheat line Saar which carries the leaf tip necrosis (LTN)-associated rust resistance genes *Lr34* and *Lr46* has exhibited a low level of spot blotch disease in field trials conducted in Asia and South America. One hundred and fourteen recombinant inbred lines (RILs) of Avocet (Susceptible) × Saar, were evaluated along with parents in two dates of sowing in India for 3 years (2007–2008 to 2009–2010) to identify quantitative trait loci (QTL) associated with spot blotch resistance, and to determine the potential association of *Lr34* and *Lr46* with resistance to this disease. *Lr34* was found to constitute the main locus for spot blotch resistance, and explained as much as 55 % of the phenotypic variation in the mean disease data across the six environments. Based on the large effect, the spot blotch resistance at this locus has been given the gene designation *Sb1*. Two further, minor QTL were detected in the sub-population of RILs not containing *Lr34*. The first of these was located about 40 cM distal to *Lr34* on 7DS, and the other corresponded to *Lr46* on 1BL. A major implication for wheat breeding is that *Lr34* and *Lr46*, which are widely used in wheat breeding to improve resistance to rust diseases and powdery mildew, also have a beneficial effect on spot blotch.

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Introduction

The importance of bread wheat *(Triticum aestivum* L.) as a staple food of South Asia is well recognized. Its enhanced productivity in the post Green Revolution period played a key role in ensuring food security in this thickly populated part of the world, which is mainly composed of India, Pakistan, Nepal and Bangladesh (Evenson et al. 1999; Joshi et al. 2007a). Spot blotch, which is caused by the hemi-biotrophic pathogen *Bipolaris sorokiniana* (Sacc.) Shoem syn. *Drechslera sorokiniana* (Sacc.) Subrm and Jain (syn. *Helminthosporium sativum*, teleomorph *Cochliobolus sativus*) (Kumar et al. 2002), has emerged as a major production constraint in the eastern part of South Asia's intensive cropping system (Chatrath et al. 2007; Joshi et al. 2007a). Saari (1998) reported that the average yield losses due to leaf blight in the Indian subcontinent were as much as 17.5 %. The disease becomes severe during the grain filling stage and causes significant yield loss and grain

quality deterioration in susceptible varieties (Saari 1998). *B. sorokiniana* usually induces symptoms on leaf, sheath and stem (Chand et al. 2003). However, under severe conditions it also infects spikelets resulting in shrivelled grains (Kiesling 1985) with black point at the embryo end of kernels (Kumar et al. 2002).

It is generally believed that the level of resistance in high-yielding wheat genotypes is still unsatisfactory and needs to be improved significantly in warmer humid regions of South Asia (Joshi et al. 2007b; Sharma and Duveiller 2006). Consequently, an integrated approach, with host resistance as a major component, is generally considered best for controlling the disease (Hetzler et al. 1991; Mehta et al. 1992). Resistance to spot blotch in wheat behaves like a quantitative trait (Joshi et al. 2004b), but until recently little has been known about its genetics. Quantitative trait loci (QTL) for resistance have recently been mapped in the resistance sources 'Yangmai 6' (Kumar et al. 2009), 'Ning 8201' and 'Chirya 3' (Kumar et al. 2010). Joshi et al. (2004a) observed that wheat genotypes expressing leaf tip necrosis (LTN) in general showed less spot blotch symptoms, and demonstrated a genetic association between this phenotypic marker and spot blotch resistance in a segregating population. LTN is known as a phenotypic marker for at least three different loci of biotrophic disease resistance: *Lr34/Yr18/Pm38* on 7DS (Singh 1992a), *Lr46/Yr29/Pm39* on 1BL (Rosewarne et al. 2006) and *Lr67/Yr46/Pm46* on 4DL (Herrera-Foessel et al. 2011).

The objectives of the present study were to map the main genetic factors for spot blotch resistance in a segregating population derived from the elite spring wheat line 'Saar' that is known to carry both *Lr34* and *Lr46*, and to study the potential effects of these two LTN-associated loci on spot blotch resistance under field conditions.

Materials and methods

Plant materials

A total of 114 single seed descent (SSD) derived recombinant inbred lines (RILs) and parents of the cross 'Avocet-S' × 'Saar' were obtained from one of the co-authors (Ravi P. Singh, CIMMYT, Mexico). These RILs which have earlier been used for mapping genes for resistance to powdery mildew, leaf rust and stripe rust (Lillemo et al. 2008) were evaluated in field trials for resistance to spot blotch. Avocet-S (later referred to as Avocet) is susceptible to spot blotch while Saar (Sonoita F81/Trap#1//Baviacora M92) shows a moderate level of resistance and carries the two LTN-associated disease resistance genes *Lr34/Yr18/Pm38* and *Lr46/Yr29/Pm39* (Lillemo et al. 2008).

Field evaluation for disease severity

The 114 RILs were evaluated along with the two parents in the field in two replications during the crop seasons 2007–2008, 2008–2009 and 2009–2010 at the Agricultural Research Farm of Banaras Hindu University, Varanasi, India (North-Eastern Plains Zone, 25.2°N and 83.0°E). The trials were laid out following a randomized complete block design. Each line was sown in single rows of 3 m under irrigated conditions. Row-to-row and plant-to-plant distance was 25 and 5 cm, respectively. To promote disease build up and spread, one row of the susceptible parent was planted after every 20th row and in alleys along the plots. As a safeguard to achieve good results, two nurseries were planted each year at different dates (first and last week of December) to allow the post-anthesis stage to coincide with warm temperature conducive to the disease that occurs in March (Chaurasia et al. 2000).

Creation of artificial epiphytotic conditions in the field

Artificial epiphytotic conditions were created as described by Kumar et al. (2009). The most aggressive isolate of *B. sorokiniana* (isolate no. ICMP 13584, Auckland, New Zealand) identified at Banaras Hindu University, Varanasi, India (Chaurasia et al. 2000) was multiplied on wheat grains, and a spore suspension adjusted to approximately 10^4 spores/ml of water was uniformly sprayed at three different growth states (GS), viz., tillering (GS20), flag leaf emergence (GS37) and anthesis (GS65) during evening hours. The field was irrigated immediately after inoculation and a total of six irrigations were given in the entire crop period to provide a favourable environment for the development of spot blotch disease. Furrow irrigation was used with intervals of approximately 15–20 days between two irrigations. The first irrigation was given 21 days after sowing.

Disease assessment

Disease severity (%) displayed by all the leaves of each row was recorded at three different growth stages (GS), viz., GS 63 (beginning of anthesis to half complete), GS 69 (anthesis complete) and GS 77 (late milking). The area under disease progress curve (AUDPC) based on disease severity (GS63, GS69 and GS77) over time was estimated using the following formula (Roelfs et al. 1992):

$$\text{AUDPC} = \sum_{i=1}^{n} \left[\{ (Y_i + Y_{(i+1)})/2 \} \times (t_{(i+1)} - t_i) \right]$$

where Y_i is disease level at time t_i, $t_{(i+1)} - t_i$ = time (days) between two disease scores, and n is the number of dates on which spot blotch was recorded. For proper

comparison, AUDPC values were standardized by maturity recorded for each genotype at each location to make it AUDPC percent days (Reynolds and Neher 1997).

Statistical analysis

Analysis of variance was performed with the PROC GLM procedure in SAS (SAS Institute Inc., v. 9.1.) by treating genotypes, years, sowing dates and replications as random factors. Heritability was estimated from the ANOVA information using the formula $h^2 = \sigma^2_G/(\sigma^2_G + \sigma^2_e/r)$ for single environments and the formula $h^2 = \sigma^2_G/(\sigma^2_G + \sigma^2_{GY}/y + \sigma^2_{GD}/d + \sigma^2_{GYD}/yd + \sigma^2_e/ryd)$ across sites and years, where σ^2_G = genetic variance, σ^2_{GY} = genotype-by-year interaction, σ^2_{GD} = genotype-by-date interaction, σ^2_{GYD} = genotype-by-year-by-date interaction, σ^2_e = error variance, y = number of years, d = number of sowing dates, and r = number of replicates. Pearson correlation coefficients among traits were calculated by the PROC CORR procedure in SAS.

QTL mapping

Molecular marker genotyping and linkage map construction for the Avocet × Saar population is previously described (Lillemo et al. 2008). For the present study, a few more markers were added to the *Lr46* region on chromosome 1BL. These were the CAPs marker csLV46G22 and the PCR marker CSHM46 with unpublished primer sequences kindly provided by Evans Lagudah (CSIRO Plant Industry, Canberra, Australia) and the unpublished PCR markers ncw1-V and CJ958400 kindly contributed by Gina Brown-Guedira (USDA-ARS, Raleigh, NC, USA). QTL mapping was performed with the software package QTL IciMapping v 3.1 (http://www.isbreeding.net) using the inclusive interval mapping algorithm for additive gene effects (Li et al. 2007). Linkage maps and LOD curves were drawn using MapChart, v. 2.1 (Voorrips 2002). Boxplots for visualization of QTL effects were created in Minitab (Minitab Inc., v. 16).

Results

Phenotypic evaluation

The spot blotch epidemics developed well in all six field trials, but with higher disease severities in 2007 and 2009 compared to 2008. Histograms of the mean AUDPC values of the 114 RILs revealed continuous distributions in all environments (Fig. 1). Two peaks are clearly visible for most of the environments, indicating that spot blotch resistance in the Avocet × Saar population might be under the control of one large effect gene. Correlations among the spot blotch data from field trials in 2007 and 2009 were high, while the 2008 data showed weaker, but still highly significant correlations with the other environments (Table 1). The analysis of variance showed a significant effect of genotypes across environments (Table 2). The data quality was good, with broad sense heritabilities in the range of 0.95–0.97 for single environments (Table 1) and 0.84 across environments (Table 2). Based on this, we concluded that the data were suitable for QTL mapping.

QTL mapping

One major QTL was detected with both simple interval mapping (SIM) and inclusive composite interval mapping (ICIM). This was located at the *Lr34* locus on the short arm of chromosome 7D (Fig. 2), and explained from 17 to 49 % of the phenotypic variation in AUDPC for spot blotch severity in single environments and as much as 55 % of the variation using the mean data (Table 3). The *Lr34* locus had a major effect in all environments (Fig. 3), and was the only QTL detected above the permutation-based LOD threshold of 3.1, which is equivalent to a Type I error rate of 0.05. Putative QTL with LOD scores above 2.5 were detected on chromosomes 5B and 7A in just one single environment each, but not for the mean data and these were not considered further. Inspection of the LOD curves indicated a minor effect of the *Lr46* locus on chromosome 1BL (maximum LOD score of 1.4 in 2007 date 1), but far below the significance threshold.

To further elucidate the effect of the *Lr34* locus we split the population into two based on the closely linked markers *gwm1220* and *swm10*. The major effect of the *Lr34* locus is clearly demonstrated by the histograms of the two subpopulations, which also show a much broader variation in spot blotch severity among the RILs with the susceptibility allele of *Lr34* compared to the resistant group (Fig. 4). Since the major effect of *Lr34* could have the potential to mask other minor QTL, we performed separate QTL mapping in the two sub-populations. No further QTL were detected in the sub-population of 55 RILs with *Lr34*, while two QTL were detected in the sub-population of 52 RILs with the susceptibility allele of *Lr34* (Table 4; Fig. 5). The most frequently detected QTL was located on 7DS, about 40 cM distal to *Lr34* and flanked by markers *wPt-7654* and *gdm88*. This was detected in four environments (both sowing dates in 2007 and 2009) and for the mean data. The other QTL was located at the *Lr46* locus on 1BL and only detected in one environment (2007 date 1). The resistance allele at both loci was contributed by Saar. The effects of these minor

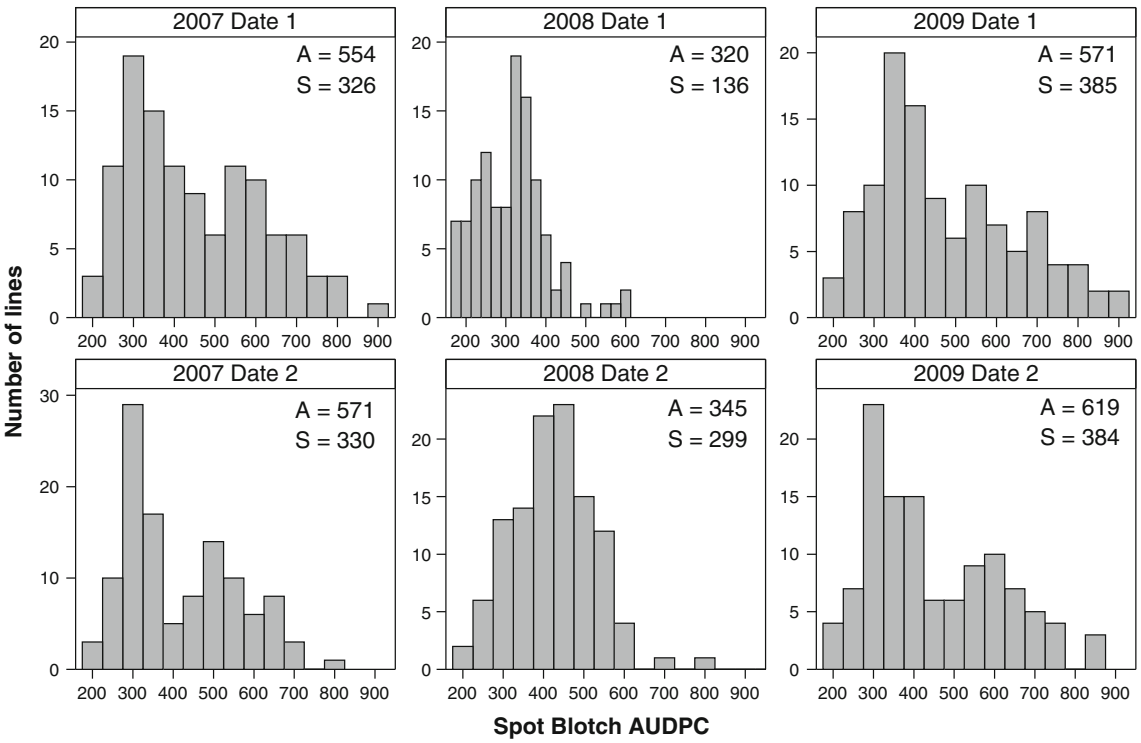

Fig. 1 Histograms of mean AUDPC for spot blotch severity in the six field trials of the Avocet × Saar RIL population. The mean values for the parents Avocet and Saar are indicated by the letters *A* and *S*, respectively

Table 1 Pearson correlation coefficients among AUDPC values for spot blotch severity in single environments and broad sense heritability estimates in the Avocet × Saar RIL population

All correlations were highly significant ($P < 0.01$)

	Correlation coefficients					Heritability h^2
	2007 date 2	2008 date 1	2008 date 2	2009 date 1	2009 date 2	
2007 date 1	0.917	0.504	0.326	0.920	0.963	0.97
2007 date 2		0.535	0.390	0.892	0.940	0.96
2008 date 1			0.677	0.429	0.511	0.95
2008 date 2				0.293	0.338	0.96
2009 date 1					0.880	0.97
2009 date 2						0.96

Table 2 Analysis of variance for AUDPC of spot blotch severity and overall heritability estimate across environments in the Avocet × Saar RIL population

Source	DF	Mean square	*F* value	*P*	Heritability h^2
Genotype	113	171815	6.17	<0.0001	0.84
Year	2	972267	1.17	0.45	
Date	1	105000	0.13	0.75	
Genotype*year	226	26966	4.64	<0.0001	
Genotype*date	113	6683	1.15	0.19	
Year*date	2	808079	1.53	0.29	
Genotype*year*date	226	5814	4.03	<0.0001	
Rep (year*date)	6	525209	364.35	<0.0001	
Error	678	1441			

QTL in combinations with *Lr34* are visualized for the mean AUDPC data in Fig. 6. It shows that these QTL were only detectable in the absence of the resistance allele at the *Lr34* locus, and that their effects were rather modest compared to the major effect of *Lr34* in this population.

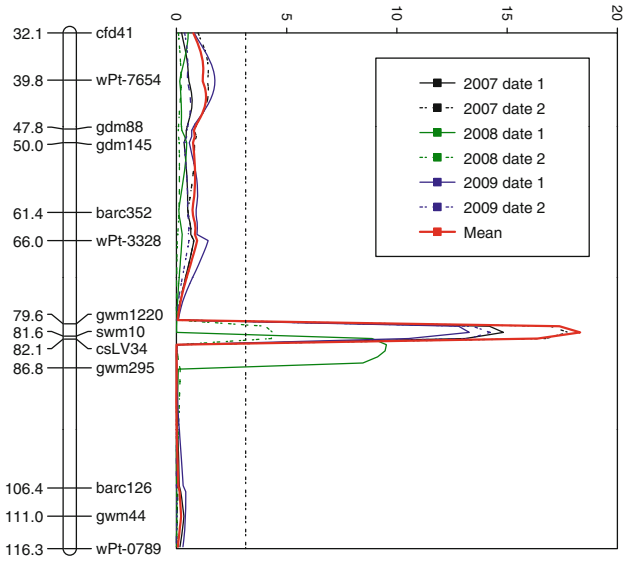

Fig. 2 Linkage map of the short arm of chromosome 7D with LOD curves obtained from inclusive composite interval mapping (ICIM) of AUDPC for spot blotch severity in single environments and the overall mean across all six environments. The LOD threshold of 3.1 determined by permutations is indicated by the *vertical dashed line*

Discussion

The bimodal distributions of AUDPC values of spot blotch severity (Fig. 1) indicated that most of the resistance in the Avocet × Saar RIL population was controlled by one gene. This was indeed confirmed in the subsequent QTL analysis with the detection of the *Lr34* locus as a major determinant of spot blotch resistance in this mapping population accounting for 55 % of the phenotypic variation in the mean data (Table 3). Although no further QTL could be detected above the significance threshold, the heritability estimate across environments of 0.84 (Table 2) indicated that a substantial part of the variation could not be explained by the *Lr34* locus alone. The histograms of the two subpopulations with and without *Lr34* (Fig. 4) indeed suggested that the major effect of *Lr34* could have masked the effects of potential minor effect QTL. This was confirmed by the detection of two minor QTL in the subpopulation of RILs with the susceptibility allele of *Lr34* (Table 4; Fig. 5). Since the power of QTL detection is low in such small subpopulations it is possible that several small effect QTL went undetected. Incomplete linkage maps for some of the chromosomes could also have limited the number of QTL detected. This does, however, not affect the main conclusion from this study: that the *Lr34* locus was a major determinant of spot blotch resistance in our mapping population.

Lr34, which has recently been cloned and found to encode an ATP-binding cassette (ABC) transporter of the ABC transporter subfamily G (ABCG), formerly known as pleiotropic drug resistance (PDR) subfamily (Krattinger et al. 2009), is known to confer broad-spectrum resistance to at least four biotrophic diseases: leaf rust (Dyck et al. 1966), stripe rust (Singh 1992b), stem rust (Dyck 1987) and powdery mildew (Lillemo et al. 2008; Spielmeyer et al. 2005). *Lr34* is also associated with a premature senescence of the leaf tips, commonly referred to as leaf tip necrosis (LTN) (Singh 1992a).

Our results are in good agreement with a previous study that found a statistical association of LTN with spot blotch severity based on field data from a collection of 1407 wheat lines (Joshi et al. 2004a). The same study also confirmed the effect of LTN in *Ltn*+ and *Ltn*− reselections of the wheat cultivar HUW234, which was heterogeneous for *Lr34*. Based on the large and consistent effect of the spot blotch resistance at this locus, it has been given the gene designation *Sb1* (R. McIntosh, pers. comm.)

It is interesting to note that one of the two minor QTL that were detected in the present study, *Lr46* is also associated with LTN. This might point to a physiological relationship of the LTN phenotype with resistance to spot blotch, which has also been suggested for resistance to rust diseases and powdery mildew (Krattinger et al. 2009). *Lr46* had a rather modest effect compared to *Lr34*, which is also a common phenomenon for its resistance to rust diseases

Table 3 List of detected QTL with inclusive composite interval mapping (ICIM) based on AUDPC for spot blotch severity in the Avocet × Saar RIL population

Environment	Chromosome	Position (cM)	Marker interval	LOD	R^2	Source of resistance
2007 date 1	7DS	81	*gwm1220-swm10*	14.8	47.5	Saar
2007 date 2	7DS	81	*gwm1220-swm10*	17.8	49.1	Saar
2008 date 1	7DS	83	*csLV34-gwm295*	9.5	35.5	Saar
2008 date 2	7DS	81	*gwm1220-swm10*	4.4	17.1	Saar
2009 date 1	7DS	81	*gwm1220-swm10*	13.3	38.2	Saar
2009 date 2	7DS	81	*gwm1220-swm10*	14.2	45.6	Saar
Mean	7DS	81	*gwm1220-swm10*	18.3	55.2	Saar

The LOD threshold of 3.1 was determined by permutations

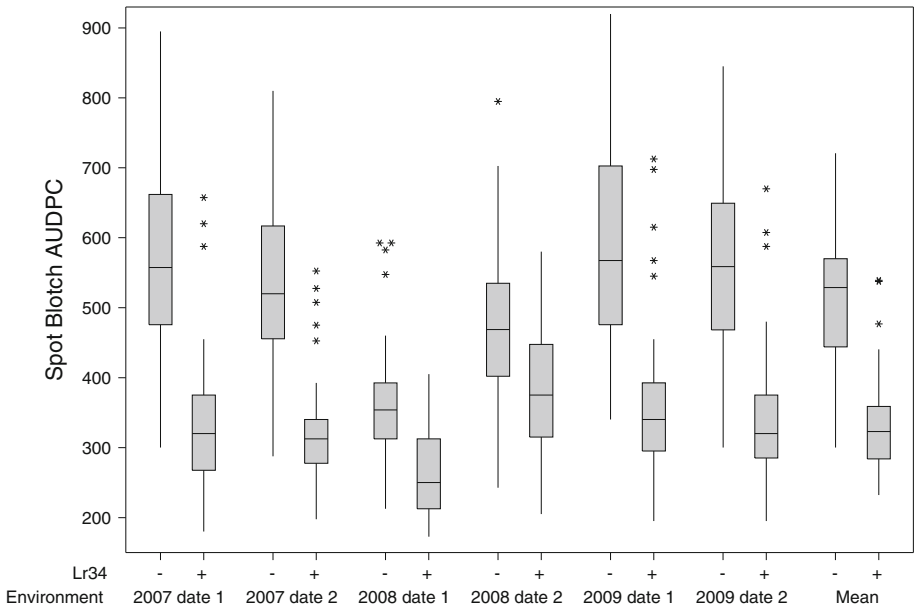

Fig. 3 Boxplot showing the effects of *Lr34* on AUDPC for spot blotch severity in six individual environments and for the overall mean of the Avocet × Saar RIL population. The allelic state of *Lr34* was determined by the flanking markers *gwm1220* and *swm10*

and powdery mildew (Lillemo et al. 2008; Martinez et al. 2001).

Although the molecular functions of *Lr34* and *Lr46* are yet to be unravelled, these general disease resistance genes are likely interfering with the basal disease resistance against biotrophic diseases due to their quantitative and pathogen non-specific resistance to multiple pathogens. Their involvement in resistance to spot blotch is not unreasonable, considering the hemibiotrophic life style of the causal pathogen *Bipolaris sorokiniana*. The infection process starts with the formation of appressoria-like structures on the leaf surface and subsequent penetration of the cell wall to form a network of infection hyphae within epidermal host cells (Kumar et al. 2002). This biotrophic phase is confined to a single epidermal cell. Then, the pathogen enters the necrotrophic phase by invading the mesophyll layer, which is accompanied by host cell death (Kumar et al. 2002). The cell wall penetration is similar to the infection process of biotrophic pathogens, and the pre-infectional defence reactions to *B. sorokiniana* have been shown to involve both papilla formation and hypersensitive cell death (Schäfer et al. 2004), which is another commonality with biotrophic disease resistance. Although more direct evidence remains to be produced, these observations point to the possibility that the spot blotch resistance associated with *Lr34* and *Lr46* could be mediated by these genes themselves rather than caused by linkage.

In comparison with the two previously published QTL studies on spot blotch resistance in wheat (Kumar et al. 2009, 2010), none of the three QTL detected in the present study have been reported before. Kumar et al. (2010) detected a major QTL on chromosome 7DS in 'Ning 8201' and 'Chirya 3', but based on the published

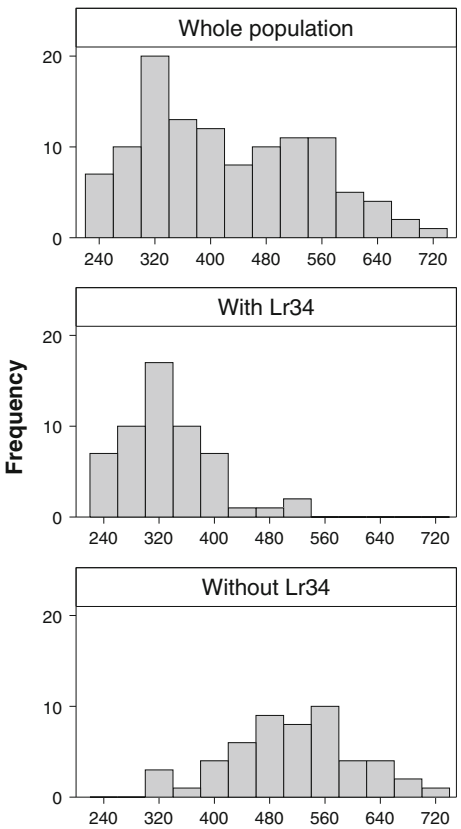

Fig. 4 Histograms of mean AUDPC for spot blotch severity across all six environments for the whole population of 114 RILs and the two *Lr34* sub-populations as determined by the flanking markers *gwm1220* and *swm10*

linkage maps this QTL was located close to the centromere and at genetic distances of about 35–70 cM from the position of *Lr34*. These QTL are therefore clearly

Table 4 List of minor QTL detected with inclusive composite interval mapping (ICIM) based on AUDPC for spot blotch severity in the subpopulation of 52 RILs with the susceptibility allele of *Lr34*

Environment	Chromosome	Position (cM)	Marker interval	LOD	R^2	Source of resistance
2007 date 1	1BL	120	*hbe248-ncw1-V*	3.78	15.1	Saar
	7DS	59	*gdm145-barc352*	4.59	21.9	Saar
2007 date 2	7DS	45	*wPt-7654-gdm88*	4.57	40.4	Saar
2008 date 1	No QTL detected					
2008 date 2	No QTL detected					
2009 date 1	7DS	42	*wPt-7654-gdm88*	5.18	29.0	Saar
2009 date 2	7DS	44	*wPt-7654-gdm88*	3.12	22.6	Saar
Mean	7DS	42	*wPt-7654-gdm88*	5.81	37.1	Saar

The LOD threshold of 3.45 was determined by permutations

QTL with LOD scores above 2.0 are listed if they reached the LOD threshold of 3.45 in at least one environment

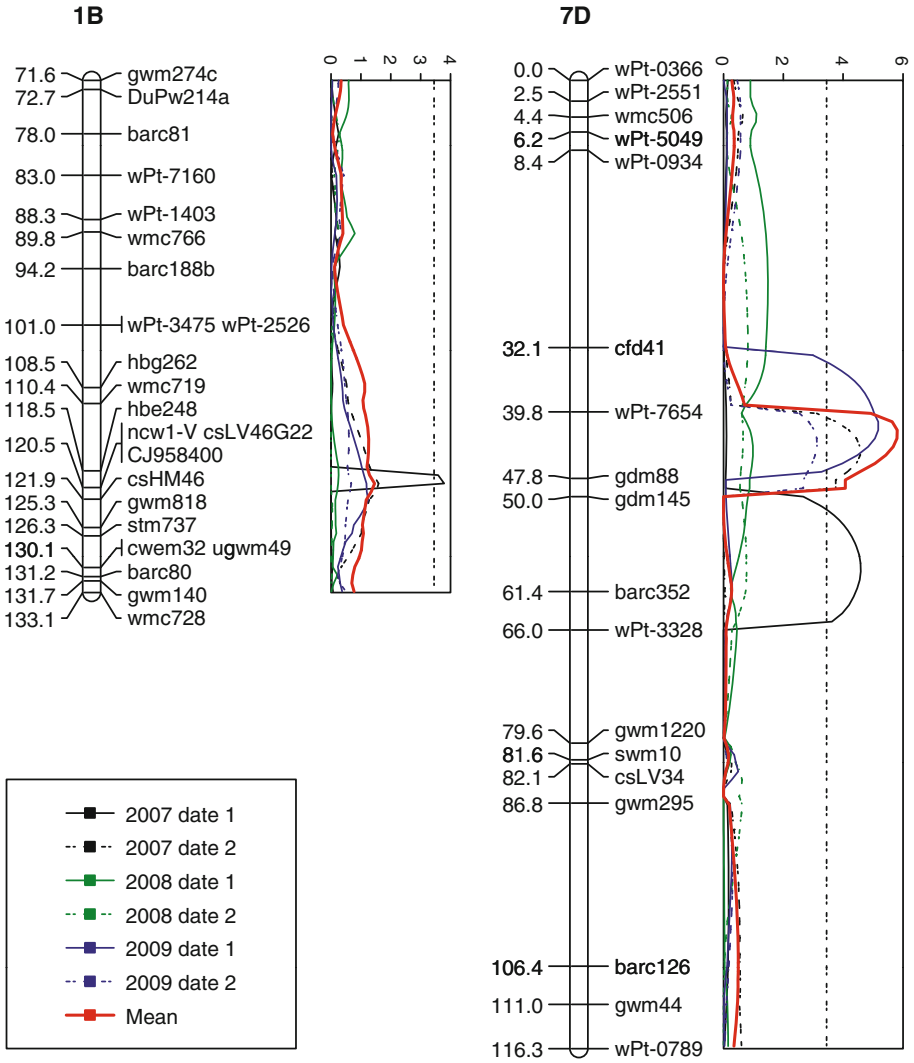

Fig. 5 Linkage maps of chromosomes 1B and 7D showing the LOD curves of minor QTL detected in the subpopulation of RILs with the susceptibility allele of *Lr34*. The LOD threshold of 3.45 determined by permutations is indicated by the *vertical dashed line*

different, but inspection of the LOD curves in the above-mentioned study does, however, reveal two minor peaks that based on consensus map information (Somers et al. 2004) could correspond to the locations of *Lr34* and the minor QTL on 7DS flanked by *wPt-7654* and *gdm88* in the present study. To our best knowledge, no QTL for spot blotch resistance has previously been detected at the *Lr46* locus on 1BL.

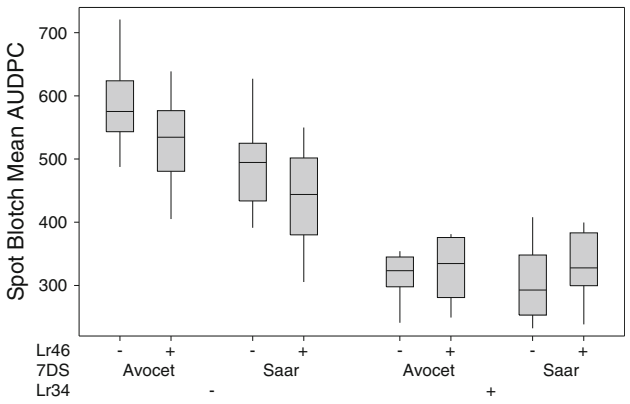

Fig. 6 *Boxplot* showing the effects of allelic combinations of *Lr34*, *Lr46* and the minor QTL on 7DS on the overall mean of AUDPC for spot blotch severity in the Avocet × Saar RIL population. The RILs were grouped according to marker alleles at the flanking markers *gwm1220* and *swm10* for *Lr34*, *hbe248* and *ncw1-V* for *Lr46* and *wPt-7654* and *gdm88* for the minor QTL on 7DS

A major implication for resistance breeding is the positive association of both *Lr34* and *Lr46* with resistance to spot blotch. These genes are widely used in international wheat breeding to improve partial and potentially durable resistance to rust diseases and powdery mildew. Since leaf rust is a major disease problem in most of the epidemic area of spot blotch in south Asia, it is beneficial that these genes in addition to improving leaf rust resistance also contribute some resistance to spot blotch. However, it should be noted that although *Lr34* appeared as a major genetic factor in the present population, the effect is quantitative and the gene needs to be combined with several other resistance genes in order to achieve adequate levels of resistance in the field.

In conclusion, we have shown that the general disease resistance gene *Lr34* is the major determinant of spot blotch resistance in wheat line Saar, and that the other LTN-associated resistance gene in this line, *Lr46* was associated with a minor disease reduction in the absence of *Lr34*. A third, minor disease resistance locus was detected on the short arm of chromosome 7D, about 40 cM distal to the location of *Lr34*. The resistance at all three loci was contributed by Saar. A major implication from this study is that the use of *Lr34* and *Lr46* in breeding for partial and potentially durable resistance to rust diseases and powdery mildew has the additional benefit of improving resistance to spot blotch.

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