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ORIGINAL PAPER

Identification of Cephalosporium stripe resistance quantitative trait loci in two recombinant inbred line populations of winter wheat

M. Dolores Vazquez · Robert Zemetra · C. James Peterson · Christopher C. Mundt

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Abstract

Key message **Identification of genome regions linked to Cephalosporium stripe resistance across two populations on chromosome 3BS, 4BS, 5AL, C5BL. Results were compared to a similar previous study.**

Abstract Cephalosporium stripe is a vascular wilt disease of winter wheat (*Triticum aestivum* L.) caused by the soil-borne fungus *Cephalosporium gramineum* Nisikado & Ikata. In the USA it is known to be a recurring disease when susceptible cultivars are grown in the wheat-growing region of Midwest and Pacific Northwest. There is no complete resistance in commercial wheat cultivars, although the use of moderately resistant cultivars reduces the disease severity and the amount of inoculum in subsequent seasons. The goal of this study was to detect and to compare chromosomal regions for resistance to Cephalosporium stripe in two winter wheat populations. Field inoculation was performed and Cephalosporium stripe severity was visually scored as percent of prematurely ripening heads (whiteheads) per plot. 'Tubbs'/'NSA-98-0995' and 'Einstein'/'Tubbs', each comprising a cross of a resistant and a susceptible cultivar, with population sizes of 271 and 259 $F_{(5:6)}$ recombinant inbred lines, respectively, were genotyped and phenotyped across four environments. In the quantitative trait loci (QTL) analysis, six and nine QTL were found, explaining in total, around 30 and 50 % of the phenotypic variation in 'Tubbs'/'NSA-98-0995' and 'Einstein'/'Tubbs', respectively. The QTL with the largest effect from both 'NSA-98-0995' and 'Einstein' was on chromosome 5AL.1 and linked to marker gwm291. Several QTL with smaller effects were identified in both populations on chromosomes 5AL, 6BS, and 3BS, along with other QTL identified in just one population. These results indicate that resistance to Cephalosporium stripe in both mapping populations was of a quantitative nature.

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M. D. Vazquez (✉) · C. C. Mundt
Department of Botany and Plant Pathology, Oregon State University, Corvallis, OR 97331, USA
e-mail: m.dolores.vazquez@oregonstate.edu

R. Zemetra
Department of Crop and Soil Science, Oregon State University, Corvallis, OR 97331, USA

C. J. Peterson
Limagrain Cereals Seeds, 3515 Richards Lake Road, Fort Collins, CO 80524, USA

Introduction

Cephalosporium stripe is a vascular wilt disease of winter wheat (*Triticum aestivum*) and other grasses that is caused by the soil-borne fungus *Cephalosporium gramineum* Nisikado & Ikata (Quincke et al. 2014). Characteristic symptoms include leaves with one-to-three broad, yellow-to-brown stripes that extend to the leaf sheaths and stems. Severe symptoms such as wilting, leaf necrosis, stunting, shorter culms, small heads, and prematurely ripening heads (whiteheads) are seen in the spring and summer after abundant colonization by *C. gramineum* (Johnston and Mathre 1972; Morton and Mathre 1980a; Morton et al. 1980; Mundt 2010). Under the appropriate environmental conditions, the disease can negatively impact yield with important economic losses to growers, largely due to reduced seed weight and seed number per head (Johnston and

Mathre 1972; Richardson and Rennie 1970). *C. gramineum* exists in at least four evolving populations (Baaj and Kondo 2011), with no evidence of substantial pathogenic variability (Cowger and Mundt 1998). The primary source of inoculum for Cephalosporium stripe is infected crop debris that remains after harvest, although seed may be an important source of inoculum in some situations (Lai and Bruehl 1967; Murray 2006). Cephalosporium stripe is favored by short crop rotations, early fall planting, presence of crop debris on the soil surface, cool and wet fall seasons, and root damage caused by soil freezing (Quincke et al. 2014).

Cephalosporium stripe was first reported in Japan in 1931 (Nisikado et al. 1934) and has been found in the United Kingdom (UK), Canada and other regions of Europe and East Asia (Richardson and Rennie 1970). In the USA, it was discovered during the mid-1950s (Bruehl 1956) and now it is known to be a recurring disease in the wheat-growing region of the Midwest and Pacific Northwest when susceptible cultivars are grown (Bockus and Sim 1982; Bockus et al. 1994; Morton and Mathre 1980a; Quincke et al. 2012). Cephalosporium stripe is an emerging problem in Scotland under conditions of short rotations, reduced tillage, and wet soils (Oxley 2009).

Cultural controls for Cephalosporium stripe include delayed planting, burning of crop residue, deep plowing, crop rotation, and the addition of lime to increase soil pH, but these practices all have significant economic and/or environmental impacts, and no chemicals are currently registered for control of the disease (Bockus and Claassen 1985; Martyniuk et al. 2006; Raymond and Bockus 1983). There is no complete resistance in commercial wheat cultivars, although the use of moderately resistant cultivars reduces disease severity in the current season and the amount of inoculum in subsequent seasons (Morton and Mathre 1980b; Mundt 2002; Shefelbine and Bockus 1989). In addition, it has been reported that progeny from crosses between the winter wheat relative *Thinopyrum ponticum* (*Agropyron elongatum*) and *Triticum aestivum* provide moderate-to-high resistance to Cephalosporium stripe (Cox et al. 2002; Mathre et al. 1985).

Field methods currently used to identify resistance to Cephalosporium stripe in breeding programs are time-consuming and space-limited. The identification of molecular markers associated with Cephalosporium stripe resistance would allow for genotypic selection of resistant genotypes. In a previous study, Quincke et al. (2011) performed a QTL analysis on a recombinant inbred line (RIL) population with two commonly grown Pacific Northwest USA winter wheat cultivars, Coda/Brundage (C/B) that varied for resistance to Cephalosporium stripe. They identified seven QTL for resistance, indicating that molecular markers may be useful for the identification of lines resistant to *C. gramineum*. The present study was undertaken to further explore the genetic resistance and identify QTL linked to Cephalosporium stripe resistance under artificially inoculated field conditions, using two mapping populations derived from crosses between European and Pacific Northwest USA winter wheat parents, and to determine if chromosomal regions associated with resistance are similar among populations.

Materials and methods

Mapping populations

Two populations of F_5-derived F_6 recombinant inbred lines (RILs) developed at Oregon State University were studied. The first population, consisting of 271 RILs, was derived from a cross between an awnless, hard red winter wheat experimental line 'NSA-98-0995' (Limagrain, UK), with a moderate-to-high level of resistance to Cephalosporium stripe, and the awned, soft white winter wheat cultivar 'Tubbs' (PI 651023), which is highly susceptible to Cephalosporium stripe. The second population, consisting of 259 RILs, was derived from a cross between the awnless, hard red winter wheat cultivar 'Einstein' (Limagrain, UK) with a high level of resistance to Cephalosporium stripe and the cultivar Tubbs. The initial crosses for both populations were done in 2003. The cultivar Einstein, bred by Nickerson Seeds and commercialized by Limagrain UK, is widely grown in Western Europe and has the pedigree (NHC 49/UK Yield Bulk) × (Haven/(Moulin/Galahad)) (Limagrain 2013). Tubbs is a cultivar released in 2000 that was widely grown in the Pacific Northwest until it became susceptible to stripe rust (*Puccinia striiformis* f. sp. *tritici*) and has the pedigree Madsen/Malcom (USDA-AMS 2013). NSA-98-0995 is an experimental line developed by Limagrain, UK, with no publically available pedigree.

Plant DNA extraction and genotyping

For both populations, DNA of parental and F_5-derived progeny was extracted from young leaves of greenhouse-grown plants using the DNeasy 96 Plant Kit (QIAGEN Science, Maryland, USA). DNA concentration was tested using NanoDrop ND-1000 UV–Vis Spectrophotometer (Thermo Fisher Scientific Inc. Wisconsin, USA.). A final volume of 15 ng/μl was sent to Triticarte Pty. Ltd Canberra, Australia, to be genotyped with DArT (Diversity Array Technology) markers (Akbari et al. 2006). Additional simple sequence repeat (SSR) markers were screened for polymorphism in the Tubbs/NSA-98-0995 (T/N) and the Einstein/Tubbs (E/T) populations in facilities at the USDA-ARS Wheat Genetics, Quality, Physiology and Disease Resistance Unit at Pullman, WA, USA, using approximately 50 ng

genomic DNA extracted from young leaves at Oregon State University.

Map construction and molecular analysis

For the populations used in this study, genotypic data were used to create the genetic linkage map with the software JoinMap v.4.0. (Van Ooijen 2006). Genetic distances were calculated using the Haldane function (Haldane 1919). For each linkage group, the best marker locus order was determined using the maximum likelihood in JoinMap v.4.0. The T/N map was constructed with 229 markers, 13 SSR and 216 DArT comprising 49 linkage groups, representing areas from all chromosomes of common wheat except 4D. The total genome length covered was 1,481 cM. The E/T map was constructed with 198 markers, 18 SSR and 180 DArT comprising 32 linkage groups, representing areas from all chromosomes of common wheat but 6D and 7D. For both populations, final linkage groups were assigned to each chromosome with data provided by Triticarte wheat map alignment (Triticarte 2013) and maps available on the database GrainGenes 2.0 (2013).

Field trials and phenotyping

The F_6-derived seed harvested from the greenhouse was used to establish plots in the field. For each population, the experimental design used was a randomized complete block with two replications. The parental cultivars, the RIL progeny, and two cultivar checks 'Stephens' (Kronstad et al. 1978) and 'Xerpha' (Jones et al. 2010) were included in the field trial. The cultivars Stephens and Xerpha were the high and low disease severity checks to Cephalosporium stripe, respectively. Experiments were conducted at the Columbia Basin Agricultural Research Center field station near Pendleton in 2010, 2011, and 2012 and in Moro, OR in 2010. Both locations are in semi-arid wheat producing areas of the Columbia Plateau with mean annual precipitation of 279 mm in Moro and 406 mm in Pendleton.

Plots consisted of two rows 2.5 m long that were later trimmed to 1.8 m long post-heading and prior to collecting phenotypic data. Fertilization and weed control were appropriate for commercial winter wheat production in eastern Oregon. Spring application of fungicide (Bumper® Makhteshim Agan Industries, Ltd. Israel) was applied to avoid eyespot and stripe rust while it does not affect *C. gramineum* establishment. For all locations, Cephalosporium stripe was established by artificial inoculation to ensure disease uniformity and high disease pressure. Before planting, oat kernels infested with *C. gramineum* (Mathre and Johnston 1975) were added to the seed envelopes in an amount equal to the volume of wheat seed. Planting dates were in early September to increase chances of high disease incidence. One reading was taken for each location in each year. Cephalosporium disease incidence was recorded every year during the last week of June on a plot basis by visual estimation of the percentage of tillers that were ripening prematurely (whiteheads) (Mathre and Johnston 1975; Quincke et al. 2012). The examination of lower stems and roots and observation of known check cultivars provided confidence that whiteheads were caused by *C. gramineum*. Tubbs and Stephens (the susceptible parent

Table 1 Mean disease severity values (% whiteheads on a plot basis) for the 272 recombinant inbred lines in the Tubbs/NSA-98-0995 (T/N) population, the parental lines, and two cultivar checks exposed to Cephalosporium stripe disease in four environments

T/N population	Cultivar checks		Parents		RILs population	
Environment	Xerpha	Stephens	NSA 98-0995	Tubbs	Mean	Range
Pendleton 2010	5.8	76.7	2.2	64.8	25.9	0–90
Moro 2010	1.5	49.0	0.6	41.9	14.6	0–80
Pendleton 2011	6.0	41.3	1.8	39.0	12.8	0–75
Pendleton 2012	2.8	33.5	3.6	38.8	15.3	0–80

Table 2 Mean disease severity values (% whiteheads on a plot basis) for the 259 recombinant inbred lines in the Einstein/Tubbs (E/T) population, the parental lines, and two cultivar checks exposed to Cephalosporium stripe disease in four environments

E/T population	Cultivar checks		Parents		RILs population	
Environment	Xerpha	Stephens	Einstein	Tubbs	Mean	Range
Pendleton 2010	9.4	79.1	24.7	69.9	24.8	0–95
Moro 2010	0.9	76.7	2.8	44.1	10.0	0–80
Pendleton 2011	7.8	34.2	2.2	35.7	5.7	0–50
Pendleton 2012	1.1	26.1	2.1	31.3	7.0	0–70

and susceptible check, respectively) showed above 40 % whiteheads, usually 2–3 weeks after heading. Developmental state of the lines ranged from early milk to early dough (Zadoks 50–60). Presence of awns, heading date, and height were recorded for possible association with Cephalosporium stripe resistance. Presence of awns and height were recorded in Pendleton 2010.

QTL and statistical analyses

The square-root transformation of whitehead percentage on a plot basis was used to calculate analysis of variance and heritability, with the transformation being used to better satisfy the assumptions of analysis of variance. The PROC GLM procedure in SAS version 9.1.3 (SAS Institute Inc. 2000) was used to calculate least squares means and to determine effects for RILs, environment, and RILs × Environment. The PROC MIXED procedure was used to calculate family heritability (h^2) on a plot basis as $h^2 = \sigma_g^2/\sigma_p^2 = \sigma_f^2/(\sigma_f^2 + \sigma_e^2/r)$, where the variance components are σ_f^2, genetic variance; σ_p^2, phenotypic variance; σ_f^2, family variance; σ_e^2, error variance; and r, number of replications (Holland et al. 2010). For all tests, a probability level of $P < 0.05$ was used.

The least squares means of the whitehead percentage on a plot basis as a measurement for disease incidence was used to perform QTL analyses. QTL analysis was performed using composite interval mapping (CIM) in WinQTL Cartographer v.2.5 (Wang et al. 2007). For both populations, the QTL analyses were done individually per location and with the arithmetic mean across environments to deduce balanced values for each RIL. Likelihood-odds (LOD) thresholds for declaring statistical significance were calculated by 1,000 permutations (Churchill and Doerge 1994). Window size was set at 5 cM for each dataset section using forward and backward stepwise regression. The additive effects (a) and phenotypic variance coefficients of determination (R^2) for each QTL were estimated by CIM for each individual environment and for the arithmetic mean across environments. Epistatic interactions analyses were performed with multiple interval mapping (MIM) in WinQTL Cartographer v.2.5 using the option "Scan through QTL mapping results file" and later refined using the option "Testing for existing QTLs" under the AIC-based selection criteria (Silva et al. 2012; Wang et al. 2007).

Results

Phenotypic values and statistical analysis

Significant disease pressure was obtained each year in each location for both populations. For the T/N population, disease severity values for the susceptible parent Tubbs ranged from 39 % whiteheads in Pendleton 2012 to 64.8 % in Pendleton 2010. Disease severity for the resistant parent NSA-98-0995 ranged from 0.6 % in Moro 2010 to 3.6 % in Pendleton 2012 (Table 1). For the E/T population, the disease severity values for the susceptible parent Tubbs ranged from 31.3 % whiteheads in Pendleton 2012 to 69.9 % in Pendleton 2010, while the disease severity scores for resistant parent Einstein ranged from 2.1 % in Pendleton 2012 to 24.7 % in Pendleton 2010 (Table 2). The resistant and

Table 3 Analyses of variance (Type III SS), coefficient of variation (CV), and heritability estimates (h^2) for disease severity in Tubbs/NSA-98-0995 (T/N) population (272 recombinant inbred lines) exposed to Cephalosporium stripe disease in four and combined environments

T/N population		Source of variation	
Environment		*df*	Mean square
Combined			
Rep (Env)		3	115.3**
RIL		271	18.4**
RIL × Env		808	1.1**
Error		1,047	0.8
CV (%)	34		
h^2 (SE)	0.8 (±0.017)		
Pendleton 2010			
Rep		1	91.7**
RIL		267	9.9**
Error		263	1.6
CV (%)	29		
h^2 (SE)	0.6 (±0.037)		
Moro 2010			
Rep		1	13.8**
RIL		270	6.5**
Error		245	2.1
CV (%)	45		
h^2 (SE)	0.5 (±0.047)		
Pendleton 2011			
Rep		1	5.1**
RIL		271	5.3**
Error		270	1.2
CV (%)	40		
h^2 (SE)	0.6 (±0.037)		
Pendleton 2012			
Rep		1	4.1*
RIL		271	4.2**
Error		265	1.3
CV (%)	32		
h^2 (SE)	0.6 (±0.039)		

* Significant at the 0.05 probability level

** Significant at the 0.01 probability level

Table 4 Analyses of variance (Type III SS), coefficient of variation (CV), and heritability estimates (h^2) in the E/T population (259 recombinant inbred lines) exposed to Cephalosporium stripe disease in four and combined environments

E/T population		Source of variation	
Environment		*df*	Mean square
Combined			
Rep (Env)		3	93.2**
RIL		258	8.6**
RIL × Env		773	1.1**
Error		1,003	0.6
CV (%)	42		
h^2 (SE)	0.3 (±0.025)		
Pendleton 2010			
Rep		1	12.6**
RIL		258	10.3**
Error		256	1.7
CV (%)	31		
h^2 (SE)	0.7 (±0.027)		
Moro 2010			
Rep		1	23.7**
RIL		258	5.6**
Error		258	1.3
CV (%)	45		
h^2 (SE)	0.6 (±0.041)		
Pendleton 2011			
Rep		1	10.5**
RIL		258	3.2**
Error		253	0.8
CV (%)	48		
h^2 (SE)	0.5 (±0.044)		
Pendleton 2012			
Rep		1	24.7**
RIL		257	3.1**
Error		232	1.2
CV (%)	51		
h^2 (SE)	0.3 (±0.057)		

* Significant at the 0.05 probability level

** Significant at the 0.01 probability level

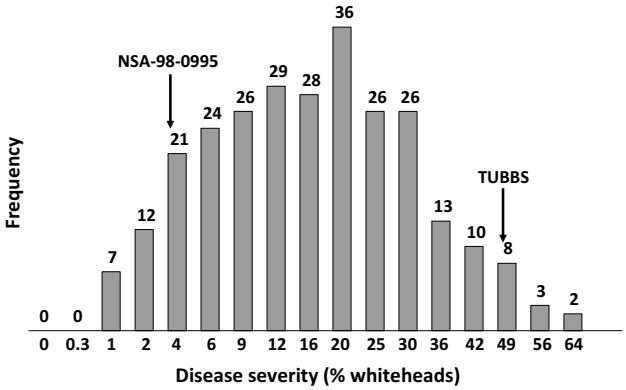


Fig. 1 Recombinant inbred lines histogram of the Tubbs/NSA-98-0995 (T/N) population, with the *arrows* indicating the arithmetic mean of the percentage whiteheads for the parents. *Numbers on top of the bars* are frequency for each bin

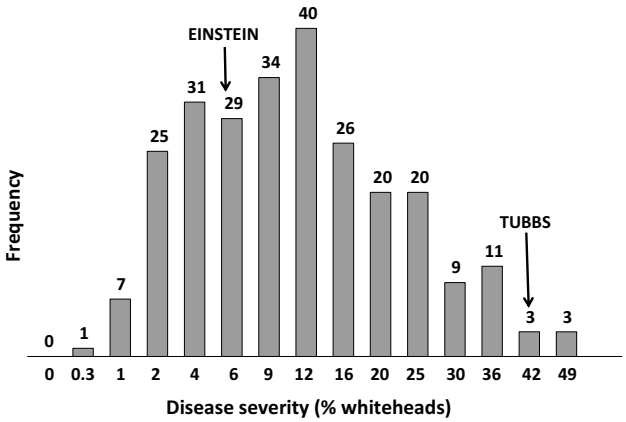


Fig. 2 Recombinant inbred lines histogram of the Einstein/Tubbs (E/T) population with the *arrows* indicating the arithmetic mean of the percentage whiteheads for the parents. *Numbers on top of the bars* are frequency for each bin

susceptible check cultivars Xerpha and Stephens were present in both populations and had disease severity scores ranging from 0.9 to 9.4 % and 26.1 to 79.1 %, respectively (Tables 1, 2).

Disease severity values in each environment for RIL in both populations suggest that the response is that of a quantitative trait. When data were square root transformed, disease severity responses were normally distributed in all environments. All statistical analyses were performed using the square root of whitehead percentage. *P* values in ANOVA test for both populations suggest line by environment interactions; significant differences among genotypes in each environment and that replication accounted for some of the variation. Heritabilities (h^2) were moderate-to-high depending on the environment (Tables 3, 4).

In the case of the E/T population, heritabilities ranged from 0.3 to 0.7; coefficients of variation (CVs) ranged from 31 to 51 % and when all data were combined, the heritability was 0.3. The low heritability in the E/T population (Table 4) is likely due to greater variation in disease severity among environments and missing values in the Pendleton 2012 experiment. For the T/N population, the values ranged from 0.5 to 0.6 per individual location and coefficients of variation (CVs) ranged from 29 to 45 %. When T/N data for all locations were combined, the heritability was 0.8. In the T/N population, there is little indication of

Table 5 Summary of the QTL detected in the Tubbs/NSA-98-0995 (T/N) population associated with disease response to Cephalosporium stripe, including closest linked markers, likelihood odds (*LOD*) scores, phenotypic coefficients (R^2), and estimated additive effects (a)

Environment	QTL	*QCsns.orz-5AL.1*	*QCsns.orz-6BS*	*QCstb.orz-3BS*	*QCsns.orz-C5BL*	*QCsns.orz-5AL.2*	*QCsns.orz-7BS*
	Closest marker	*gwm291*	*wPt-2726*	*wPt-9432*	*barc004*	*tPt-3642*	*wPt-0963*
Moro 2010	LOD	11.9	4.0	2.8	3.0	–	–
	R^2	18.0	5.5	3.6	4.0	–	–
	a	6.2	78.8	76.5	74.3	–	–
Pendleton 2010	LOD	6.8	7.2	–	–	3.2	–
	R^2	8.5	11.2	–	–	3.9	–
	a	6.0	6.8	–	–	4.1	–
Pendleton 2011	LOD	7.4	13.0	3.5	–	–	3.3
	R^2	9.7	19.6	4.1	–	–	4.2
	a	3.6	5.1	−2.3	–	–	2.3
Pendleton 2012	LOD	7.9	16.7	3.2	7.1	–	–
	R^2	10.0	21.7	3.3	7.7	–	–
	a	3.9	5.8	−2.3	3.5	–	–
Combined environment	LOD	13.6	13.1	4.7	5.3	–	–
	R^2	16.2	17.3	4.9	5.7	–	–
	a	5.1	5.2	−2.8	3.0	–	–

Negative additive effect values (a) indicate that the resistance allele is derived from parent 'Tubbs'

Positive additive effect values (a) indicate that the resistance allele is derived from parent 'NSA-98-0995'

Table 6 Summary of the QTL detected in the Einstein/Tubbs (E/T) population associated with disease response to Cephalosporium stripe, including closest linked markers, likelihood odds (LOD) scores, phenotypic coefficients (R^2), and estimated additive effects (a)

Environment	QTL	*QCsen. orz-5AL.1*	*QCsen. orz-4DS*	*QCsen. orz-5AL.2*	*QCstb. orz-2BL*	*QCsen. orz-4BS*	*QCsen. orz-6BS*	*QCstb. orz-1BL*	*QCstb. orz-2AS*	*QCstb. orz-3BS*
	Closest marker	*gwm291*	*wPt-0472*	*wPt-3563*	*wPt-9736*	*tPt-0602*	*cfd1*	*wPt-2315*	*cfd36*	*WPt-9432*
Moro 2010	LOD	5.3	–	–	3	–	–	–	–	–
	R^2	8.8	–	–	5.9	–	–	–	–	–
	a	3.4	–	–	−2.9	–	–	–	–	–
Pendleton 2010	LOD	4.6	–	5.9	–	6.6	–	3.9	–	–
	R^2	7.3	–	9.3	–	12	–	5.7	–	–
	a	5.6	–	6.2	–	7.3	–	−4.9	–	–
Pendleton 2011	LOD	4.2	8.2	–	–	–	6.2	–	–	3.4
	R^2	6.6	17	–	–	–	8.7	–	–	4.5
	a	1.7	2.8	–	–	–	2.4	–	–	−1.4
Pendleton 2012	LOD	4	4.3	–	–	–	–	–	3.9	–
	R^2	6.2	7.6	–	–	–	–	–	5.8	–
	a	2	2.1	–	–	–	–	–	−1.9	–
Combined environment	LOD	7.2	5	5.4	4.1	–	–	–	–	–
	R^2	11.6	7.9	8.6	6.2	–	–	–	–	–
	a	3.4	2.7	2.9	−2.6	–	–	–	–	–

Negative additive effect values (a) indicate that the resistance allele is derived from parent 'Tubbs'

Positive additive effect values (a) indicate that the resistance allele is derived from parent 'Einstein'

transgressive segregation, as severity ratings of RILs generally fell within parental values, but this was not the case for the E/T population, where disease severity values for 25 % of the population fell below the average value of the resistant parent Einstein, thus suggesting transgressive segregation (Figs. 1, 2).

Table 7 Summary of the epistatic interactions detected using multiple interval mapping (MIM) in the Tubbs/NSA-98-0995 (T × N) and Einstein/Tubbs (E/T) populations among identified QTLs, phenotypic variance by locations, and arithmetic means cross locations

Population	Location	MIM Phenotypic variance (*R*%)	Epistatic interaction	Markers interacting	Epistatic effect (%)
T/N	Moro 2010	50.0	5AL1 × 6BS	*gwm291*wPt-2726*	4.6
	Pendleton 2010	28.4	–	–	–
	Pendleton 2011	54.6	5AL1 × 6BS	*gwm291*wPt-2726*	5.7
	Pendleton 2012	43.0	–	–	–
	Mean across locations	50.0	5AL1 × 6BS	*gwm291*wPt-2726*	3.6
E/T	Moro 2010	16.3	–	–	–
	Pendleton 2010	31.9	–	–	–
	Pendleton 2011	48.8	5AL1 × 4DS	*gwm291*wPt-0472*	5.3
	Pendleton 2012	29.8	5AL1 × 4DS	*gwm291*wPt-0472*	4.3
	Mean across locations	30.7	–	–	–

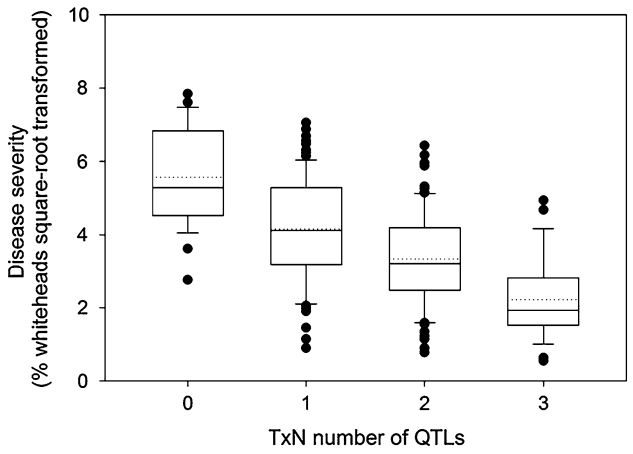


Fig. 3 Tubbs/NSA-98-0995 (T/N) population *boxplots* (medians are *thick lines*, means are *dotted lines*, quartiles are *boxes*, *whiskers* extend to the farthest points that are not outliers, and outliers are *black bullets*) for disease severity associated with the number of the three most frequently identified QTL

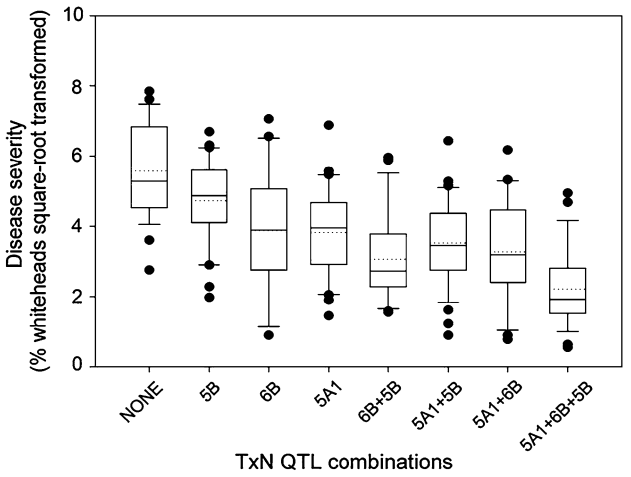


Fig. 4 Tubbs/NSA-98-0995 (T/N) population *boxplots* (quartiles are *boxes*, medians are *continuous lines*, means are *dotted lines*, *whiskers* extend to the farthest points that are not outliers, and outliers are *black dots*) of the three most frequently identified QTL (5BS, 6BS and 5AL.1) and specific QTL combinations among them

QTL analysis

T/N population

The six QTL contributing to disease resistance in the T/N population were identified in chromosomes 3BS, 5AL.1, 5AL.2, C5BL, 6BS, and 7BS (Table 5). The QTL *QCsns.orz-6BS* and *QCsns.orz-5AL.1* were identified in every environment and in the arithmetic mean across environments (Table 5). The QTL in the short chromosome of 6B linked to marker *wPt-2726*, showed the highest phenotypic variance response (5.5–21.7 %). The QTL *QCsns.orz-5AL.1* linked to the marker gwm291, explained a relative high percentage of the phenotypic variance response (8.5–18 %). The QTL identified in the short arm of chromosome 3B, *QCstb.orz-3BS* linked to the marker *wPt-9432*, was identified in three locations and the arithmetic mean across environments. The QTL located in the 5B centromeric-long arm region, *QCsns.orz-C5BL* was linked to marker barc004. It was identified in two environments and the combined mean analysis. The C5BL QTL is located in the translocation 5B:7B, which is known to be present in several west European wheat cultivars (Badaeva et al. 2007). Two QTL were identified in just one environment, QTL in 5AL, *QCsns.orz-5AL.2*, linked to marker tPt-3642 and in the short arm of 7B, *QCsns.orz-7BS,* linked to marker *wPt-0963*. The resistant parent NSA-98-0995 was the allele donor for five of the six identified QTL, while the susceptible parent Tubbs was the resistance allele donor for the QTL on chromosome 3BS. When accounting for the six QTL detected, the total phenotypic variance explained by location ranged from 23.6 to 42.7 %, while in the combined analysis was 44.1 % (Table 5). Epistatic interaction was detected between the QTL *QCsns.orz-5AL.1* and

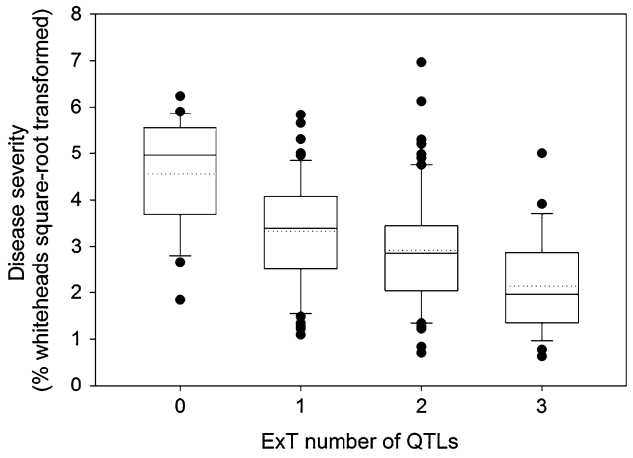


Fig. 5 Einstein/Tubbs (E/T) population *boxplots* (quartiles are *boxes*, medians are *continuous lines*, means are *dotted lines*, *whiskers* extend to the farthest points that are not outliers, and outliers are *black dots*) for disease severity associated with number of the three most frequently identified QTL (5AL.1, 4DS and 5AL.2)

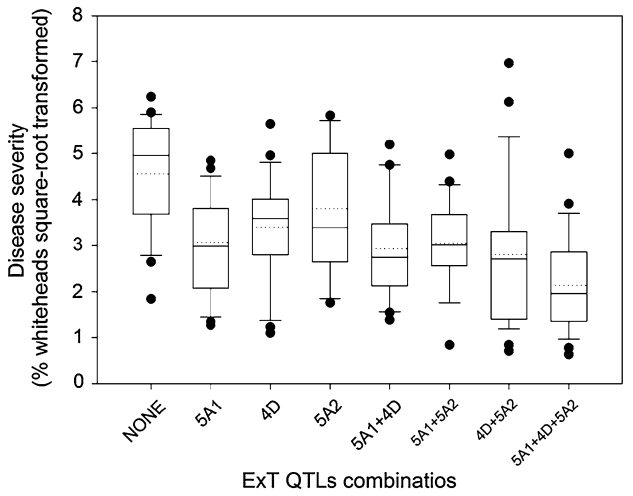


Fig. 6 Einstein/Tubbs (E/T) population *boxplots* (quartiles are the *boxes*, medians are the *continuous lines*, means are *dotted lines*, *whiskers* extend to the farthest points that are not outliers, and outliers are *black dots*) for disease severity associated with the three most frequently identified QTLs (5AL.1, 4DS and 5AL.2), and combinations among them

QCsns.orz-6BS in three locations with an effect ranging from 3.6 to 5.7 % (Table 7).

E/T population

Nine QTL contributing to disease resistance in the E/T population were identified in chromosomes 1BL, 2AS, 2BL, 3BS, 4BS, 4DS, 5AL.1, 5AL.2 and 6BS (Table 6). The QTL identified in one location are in chromosomes 1BL, 2AS, 3BS, with the susceptible parent Tubbs as the allele donor and each explaining a phenotypic variance around 5 %. Also identified in one location are QTL in chromosomes 4BS and 6BS, with the parent allele donor Einstein and explaining a phenotypic variance of 12.0 and 8.7 %, respectively. The first QTL in chromosome 5AL, *QCsen.orz-5AL.1* linked to marker gwm291, was identified in every environment and in the combined means analysis, explaining a phenotypic variance between 6.2 and 11.6 %, depending on the environment. The QTL in chromosome 4DS, *QCsen.orz-4DS,* linked to marker *wPt-0472* and with the resistance allele donor from Einstein, was identified in two environments and in the combined means analysis, explaining a phenotypic variance between 7.6 and 17.0 %, depending on the environment. The second QTL in 5AL, *QCsen.orz-5AL.2,* linked to marker *wPt-3563* and with the resistance allele derived from Einstein, was identified in one environment and in the combined means environment with a phenotypic variance response of around 9 %. For the QTL in chromosome long arm 2B, *QCstb.orz-2BL* linked to marker *wPt-9736*, the allele donor was Tubbs and was identified in one environment and in the combined means analysis, explaining a phenotypic variance of around 6 %. Total phenotypic variance explained by the identified QTL in the E/T population ranged between 14.7 and 36.8 %, depending of the environment (Table 6). Epistasis was detected in two locations between QTL *QCsen.orz-5AL.1* and *QCsen.orz-4DS*, with an effect of 4.3 and 5.3 % in each location, respectively; both alleles were from parent donor Einstein (Table 7).

Discussion

In the case of the T/N population, the three main QTL derived from the resistant parent NSA-98-0995 (6BS, C5BL, 5AL.1) accounted for around 30 % of the total phenotypic response and were found to be present in lines where disease severity was below 15 % whiteheads. The presence of these three QTL in combination reduced disease incidence substantially, but the presence of just one of these QTL showed little effectiveness in reducing disease (Figs. 3, 4). A similar result was found for the E/T population, where a combination of the three main QTL derived from the resistant parent Einstein (5AL.1, 4DS and 5AL.2) reduced disease severity substantially (Figs. 5, 6). Approximately 25 % of the E/T population showed transgressive segregation, which may be caused by recombination of additive alleles, epistatic effect between alleles or overdominance (Rieseberg et al. 1999).

In both populations, we detected epistatic interactions, with the interacting alleles derived from the resistant parents. The QTL 5AL.1 linked to marker gwm291 was

Table 8 Summary of QTL regions similarities among the Tubbs/NSA-98-0995 (T/N), Einstein/Tubbs (E/T) and Coda/Brundage (C/B) populations for Cephalosporium stripe

QTL	*QCs.orz-5AL.1*	*QCs.orz-5AL.2*	*QCs.orz-C5BL*	*QCs.orz-3BS*	*QCs.orz-4BS*
BIN[a]	5AL23-0.87-1.00	5AL12-0.35-0.57	C-5BL6-0.29	3BS1-0.33	4BS4-0.37
T/N	*NSA-09-0995 gwm291*	*NSA-09-0995 tPt-3642*[b]	*NSA-09-0995 barc004*	*Tubbs wPt-9432*	–
E/T	*Einstein gwm291*	*Einstein wPt-3563*	[c]*Tubbs barc004*	*Tubbs wPt-9432*	*Einstein tPt-0602*
C/B	*Brundage gwm291*	*Brundage wPt-3563*	*Coda gwm639*[b]	–	*Brundage wPt-3908*[b]

Coda/Brundage (C/B) (Quincke et al. 2011)

[a] Identification of bin using as reference (Marone et al. 2012)

[b] Markers localized in similar region by comparing their relative position from nearby markers using others populations' maps (Graingenes 2.0 and Cmap). For more details see Fig. 7

[c] Unpublished data

detected consistently across environments in both populations, making it a good candidate for use in marker-assisted selection. This region was identified previously (Quincke et al. 2011), located close and probably pseudo-linked to the B1 gene conditioning the awnless trait (Kato et al. 1998). In addition, several QTL for resistance to Cephalosporium stripe were found in common among the T/N population, E/T population, and the C/B population reported previously by Quincke et al. (2011) (Table 8). The models used in this study only accounted for additive effects and interaction among detected QTL. It would also be expected that epistatic interactions between non-detected QTL and other loci would have a role in the levels of disease resistance observed in this study. In both populations of the current study, there were QTL detected from the susceptible parent Tubbs that contributed to resistance in the recombinant inbred line populations. Although in both populations the combinations of three QTL from the resistant parent reduced disease incidence of Cephalosporium stripe substantially, QTL from the susceptible parent could play a role in the genetic background of the RILs so as to provide disease resistance, even when such resistance is not functional in the susceptible parent Tubbs. One potential explanation for this phenomenon is the presence of suppressor genes. Knott (2000), in a study suggested that wheat cultivar 'Medea' possessed suppressors for some of its genes for resistance; such genes for resistance were later expressed in progeny from a cross with another cultivar.

Interactions among QTL and background effects lead to questions regarding how to successfully validate QTLs in several wheat backgrounds and environments and how to estimate the best QTL combinations for their use in marker-assisted selection when pyramiding genes for resistance. Miedaner et al. (2006) highlighted the unexpected outcomes that can arise when combining QTLs for resistance in a different genetic background than their original source with Fusarium head blight (FHB). They introgressed two donor-QTL located in chromosomes 3B and 5A from 'CM82036', a non-adapted line, and one donor-QTL in chromosome 3A from 'Frontana', a Brazilian cultivar, into elite European spring wheat. Individual and combined QTL effects were estimated for FHB disease severity and Fusarium exoantigen content (DON). All three individual donor-QTL alleles significantly reduced DON. However, the donor-QTL allele 3A had no significant effect on FHB severity, either individually or in combination with other QTL. The highest effect was from the stacked donor-QTL alleles 3B and 5A for both traits.

The mechanisms of action of the QTL identified in this study are unknown. However, it has been suggested that host-selective toxins may be a mechanism of action of *C. gramineum* (Kobayashi and Ui 1979; Rahman et al. 2001). There is evidence that necrotrophic pathogens produce effectors (host-selective toxins) that interact with defense-associated proteins eliciting a resistance-like response that confers susceptibility. Defense-associated proteins, to which the host selective toxins may interact, belong to the class of resistance proteins used in the resistance mechanism that follow the gene-for-gene interaction system. Such resistance proteins consist of the N-terminal nucleotide-binding site (NBS) C-terminal leucine-rich repeat (LRR) proteins characterized as NB-LRR. Another class of defense-associated proteins is composed of serine/threonine protein kinase (S/TPK) domain (Faris et al. 2010; Lorang et al. 2007). Lorang et al. (2012) reported that necrotrophic pathogens that make use of host-selective toxins as determinants of pathogenicity may do so by interacting with the same genes that biotrophic pathogens use to induce resistance reactions (Dangl and Jones 2001; Wolpert et al. 2002). Lillemo et al. (2013) reported a QTL for the necrotrophic pathogen spot blotch (*Bipolaris sorokiniana*, telemorph *Cochliobolus sativus*) that co-locates to the locus *Lr34,* which provides resistance to leaf rust (*Puccinia triticina* Eriks). Joshi et al. (2004) reported the phenotypic marker leaf tip necrosis linked to at least three different loci of biotrophic disease resistance *L34/Yr18/Pm38*

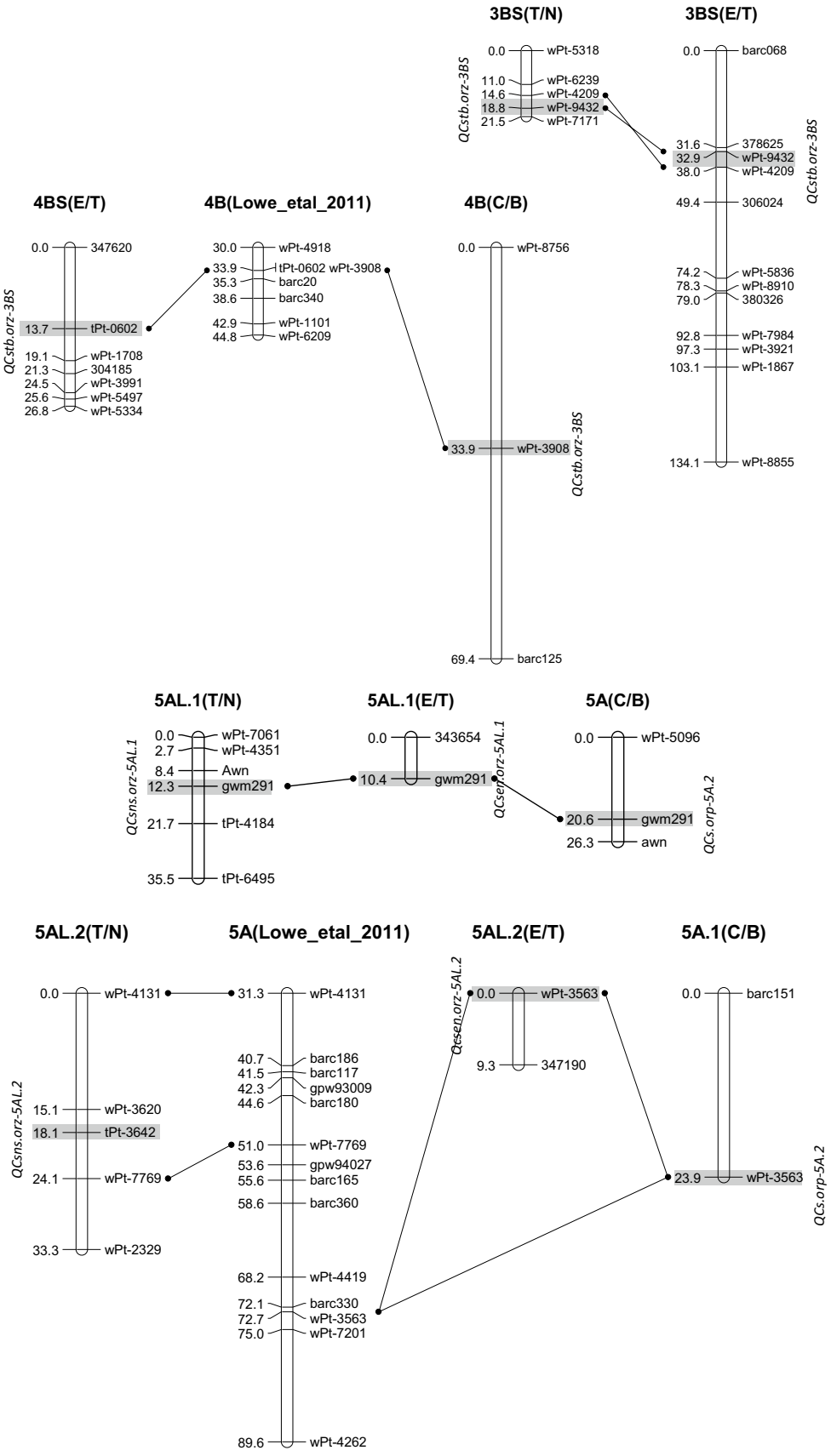
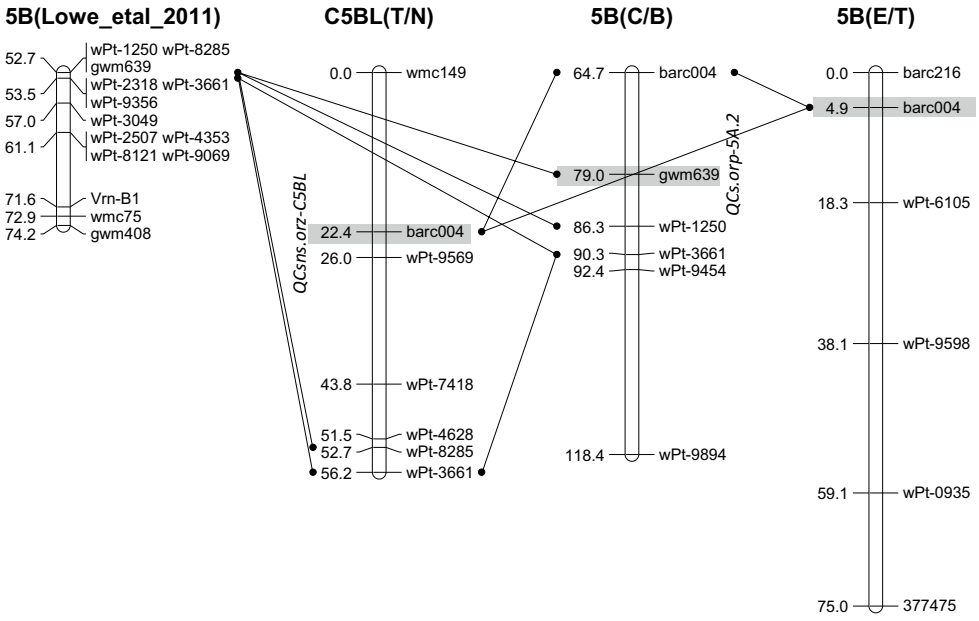


Fig. 7 Linkage maps of Tubbs/NSA-98-0995 (T/N), Einstein/Tubbs (E/T) and Coda/Brundage (C/B) where potential QTLs linked to Cephalosporium stripe resistance were found, indicated with a *colored bar* over the linked marker. Position similarity among populations are indicated with a *line*. In chromosomes 4BS, 5AL.2 and 5B, maps excepts are included (Lowe et al. 2011) for reference

Fig. 7 continued



is associated with moderate resistance to spot blotch. Adhikari et al. (2012) reported a QTL in 3BS linked to resistance to spot blotch in the same location where Poole et al. (2012) identified a QTL linked to resistance to Fusarium crown rot (*Fusarium pseudograminearum*) and where Chen et al. (2013) identified a QTL for resistance to sharp eyespot (*Rhizoctonia cerealis*). This is the same region where the QTL in 3BS from E/T and T/N (derived from Tubbs) was detected in this study, but it was not a QTL with strong effect and was detected in only one location for both populations. In addition, Lowe et al. (2011) evaluated a cross between UC1110, an adapted California spring wheat, and PI610750, a synthetic derivative from CIMMYT's Wide Cross Program, for its response to current California races of stripe rust (*Puccinia striiformis* f. sp. *tritici*). They reported a QTL in chromosome 5AL that is located in the same region as 5AL.1 for Cephalosporium stripe found in E/T, T/N populations and in C/B (Quincke et al. 2011). In addition, there is one QTL identified in 5AL from Cappelle Desprez that confers resistance to eyespot-strawbreaker foot rot (*O. yallundae* and *O. acuformis*) that is in a similar region of 5AL.2 that was identified in the E/T, T/N and C/B (Quincke et al. 2011) studies for Cephalosporium stripe. Some of these regions have also been related to disease resistance to other diseases, such as FHB (*Fusarium gramineum*) and Septoria tritici blotch (*Zymoseptoria tritici*), suggesting that either these regions are hot spots for multiple specific genes or for general genes that give resistance to multiple pathogens (Bovill et al. 2006; Buerstmayr et al. 2009; Cuthbert et al. 2007; Liu et al. 2013; Miedaner et al. 2012; Muhovski et al. 2012; Risser et al. 2011).

Results of this study are part of the first step to develop genotypic markers for their use in breeding for resistance to Cephalosporium stripe. In addition, potential QTLs for resistance to Cephalosporium stripe identified in this study (Fig. 7) have been identified in another study (Quincke et al. 2011), indicating those may be useful QTL for breeding for Cephalosporium stripe resistance across an array of breeding backgrounds. The discovery of epistatic interactions among the QTL for resistance provides an explanation for the variability in disease resistance response when combining QTL among different combinations. Further work is needed to improve the molecular markers identified in this study by saturating the chromosomes regions of interest to identify markers tightly linked to the Cephalosporium stripe resistance QTL.

Author contribution statement Conceived and designed the experiments: MDV CJP CCM. Performed the experiments: MDV CCM. Analyzed the data: MDV. Contributed reagents/materials/analysis tools: CJP RZ CCM. Wrote the paper: MDV RZ CCM.

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Conflict of interest The authors declare that they have no conflict of interests.

Ethical standards The experiments comply with the current US laws.

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