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ORIGINAL PAPER

Quantitative trait loci analysis for resistance to Cephalosporium stripe, a vascular wilt disease of wheat

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Abstract Cephalosporium stripe, caused by *Cephalosporium gramineum*, can cause severe loss of wheat (*Triticum aestivum* L.) yield and grain quality and can be an important factor limiting adoption of conservation tillage practices. Selecting for resistance to Cephalosporium stripe is problematic; however, as optimum conditions for disease do not occur annually under natural conditions, inoculum levels can be spatially heterogeneous, and little is known about the inheritance of resistance. A population of 268 recombinant inbred lines (RILs) derived from a cross between two wheat cultivars was characterized using field screening and molecular markers to investigate the inheritance of resistance to Cephalosporium stripe. Whiteheads (sterile heads caused by pathogen infection) were measured on each RIL in three field environments under artificially inoculated conditions. A linkage map for this population was created based on 204 SSR and DArT markers. A total of 36 linkage groups were resolved, representing portions of all chromosomes except for chromosome 1D, which lacked a sufficient number of polymorphic markers. Quantitative trait locus (QTL) analysis identified seven regions associated with resistance to Cephalosporium stripe, with approximately equal additive effects. Four QTL derived from the more susceptible parent (Brundage) and three came from the more resistant parent (Coda), but the cumulative, additive effect of QTL from Coda was greater than that of Brundage. Additivity of QTL effects was confirmed through regression analysis and demonstrates the advantage of accumulating multiple QTL alleles to achieve high levels of resistance.

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Introduction

Molecular markers can be used to increase the efficiency with which qualitative and quantitative disease resistance genes are manipulated in breeding programs (Horvath et al. 1995). Marker-assisted selection offers the potential to assemble target traits in the same genotype more precisely, with less loss of favorable traits, and fewer selection cycles than with conventional breeding (Edwards and McCouch 2007; Gupta et al. 1999; Xu and Crouch 2008). Gene discovery through QTL analysis is a basic step preceding the implementation of molecular marker-assisted selection.

Cephalosporium stripe of wheat is caused by the soil-borne fungal pathogen *Cephalosporium gramineum* Nisikado and Ikata (syn. *Hymenula cerealis* Ellis & Everh.) (Bruehl 1956; Ellis and Everhart 1894; Nisikado et al. 1934). In North America, it is widespread throughout the Pacific Northwest, where it is a chronic yield-reducing disease, and in western Provinces of Canada (Bruehl 1957; Mundt 2002; Murray 2006; Wiese 1987). The disease is economically important only in winter wheat production areas. Wheat growers in erosion-prone areas are particularly affected when early plantings and reduced or no tillage are practiced. Infested crop residue is the primary source of inoculum, but low rates of seed transmission have also been reported (Murray 2006). Entry of the pathogen into wheat roots is facilitated through wounds caused by freeze injury, root feeding insects (wireworm and nematodes), or other mechanical injury (Bailey et al. 1982; Douhan and Murray 2001; Slope and Bardner 1965). Active penetration of host tissue has been reported as well (Douhan and Murray 2001). Once inside the roots, the fungus has the potential to colonize the entire plant. Successful establishment of *C. gramineum* inside the host is enhanced by the production of toxic metabolites that block the vascular system, thus preventing normal movement of water and nutrients (Bruehl 1957; Spalding et al. 1961; Wiese 1987).

In areas conducive to Cephalosporium stripe, up to 80% yield reduction from a generalized infection on a susceptible cultivar can occur. Loss in grain yield is the result of a reduction in the number of fertile florets per spike and smaller seed size (Bockus et al. 1994; Johnston and Mathre 1972; Mathre et al. 1977; Morton and Mathre 1980b; Richardson and Rennie 1970). Disease levels are highly dependent on environmental conditions (Specht and Murray 1990). Temperature, moisture, soil pH, and root wounding can have great impact on the severity of Cephalosporium stripe (Bruehl and Lai 1968; Martin et al. 1989; Pool and Sharp 1969). The disease is most severe in cool, wet soils with low pH (Blank and Murray 1998).

Management of Cephalosporium stripe has relied on reducing inoculum in the soil via cultural controls such as crop rotation, management of crop residues, altering soil pH with lime applications, and fertilizer management (Bockus et al. 1983; Latin et al. 1982; Martin et al. 1989; Mathre and Johnston 1975b; Murray et al. 1992; Pool and Sharp 1969; Raymond and Bockus 1984). However, these practices are only partially effective in reducing the incidence and severity of disease (Li et al. 2008) and often are practically or economically infeasible (Murray et al. 1992; Raymond and Bockus 1984). Additionally, Cephalosporium stripe cannot be controlled with fungicides.

Host resistance currently offers the best approach for control of Cephalosporium stripe. Two types of resistance have been observed: exclusion of the pathogen, expressed as a reduction in the percentage of diseased plants; and restriction of spread of the pathogen after successful colonization of the host, expressed as a reduction in the percentage of diseased tillers per infected plant and also as a reduced rate and severity of symptom development. Morton and Mathre (1980a) found that these two types of resistance were expressed independently, leading them to conclude that maximum resistance would be attained if both types of resistance were incorporated into a single genotype. Although variation in the degree of resistance among cultivars has been confirmed, complete resistance to *C. gramineum* has not been found in the common wheat gene pool (Bruehl et al. 1986; Martin et al. 1983; Mathre et al. 1977; Morton and Mathre 1980a). Severe levels of Cephalosporium stripe do not occur annually under natural conditions (Martin et al. 1986) and resistance is inherited quantitatively (Martin et al. 1983; Mathre et al. 1985; Morton and Mathre 1980a). Therefore, incorporating genetic resistance into new wheat cultivars through conventional breeding remains difficult and challenging.

At present, no molecular breeding approaches are available to facilitate selection for resistance to Cephalosporium stripe. Therefore, the objectives of this study were to characterize the inheritance of resistance to Cephalosporium stripe and to explore the application of molecular markers to detect and locate major genomic regions responsible for resistance to the disease that can be targeted for selection of Cephalosporium stripe resistance in wheat.

Materials and methods

Plant material

A population of 268 F_6-derived recombinant inbred lines (RILs) was developed at the University of Idaho from a cross between the soft white winter club wheat cultivar ‘Coda’ (PI 594372) (Allan et al. 2000) and the soft white winter common wheat cultivar ‘Brundage’ (PI 599193) (Zemetra et al. 1998). The pedigree for Coda is ‘Tres’//

'Madsen'/'Tres' and the pedigree for Brundage is 'Stephens'/'Geneva'. The initial cross was done in 1999 and the resulting F_1 seed was grown in the greenhouse and allowed to self. Single-seed descent was then used to arrive at the $F_{6:7}$ generation. This population was previously used to develop an improved molecular marker for the *Pch1* gene for resistance to eyespot (incited by *Oculimacula yallundae* and *O. acuformis*) (Leonard et al. 2008) and to map the *compactum* locus for club head type in wheat (Johnson et al. 2008). The parents of the population were included in each trial, together with six check varieties (Stephens, Madsen, Tubbs, Rossini, OR9800924 and WA7437) of known response to Cephalosporium stripe.

Phenotyping

Field experiments were conducted at the Columbia Basin Agricultural Research Center field stations near Pendleton, OR in 2006–2007 (2007) and 2007–2008 (2008) and in Moro, OR, in 2006–2007 (2007). 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. Field evaluations for Cephalosporium stripe were carried out using a randomized complete block design. Availability of seed limited the experiment to two replications at each site in 2007, but there were three replications in 2008. Both parents and the checks were replicated four times in each block. To ensure high and uniform disease pressure, pathogen inoculum was added to the seed envelopes before planting. This was done using autoclaved oat kernels that were infested with *C. gramineum* and then dried, as described by Mathre and Johnston (1975a). Infested oat kernels were added at a dose equal in volume to the wheat seed.

Plots were seeded into stubble mulch on 12 September 2006 in Moro and Pendleton, and on 11 September 2007 in Pendleton; sowing dates in early September greatly increase severity of Cephalosporium stripe at these sites. Each plot was two rows $\times$2.5 m long. A Hege 500 series plot drill (H&N Manufacturing, Colwich, KS, USA) with deep furrow openers was used to reach soil moisture. Fertilization and weed control practices were appropriate to commercial winter wheat production at the two sites. A spring application of fungicide (Bumper 41.8EC, propiconazole) was applied to avoid eyespot, which can mask symptoms of Cephalosporium stripe. Plots were mowed to 1.8 m in length post-heading and prior to collecting phenotypic data.

Cephalosporium stripe incidence was recorded on a plot basis by visually estimating the percentage of tillers that were ripening prematurely, and which usually expressed complete or partial reduction of grain-fill (whiteheads) (Mathre and Johnston 1975a; Morton and Mathre 1980a). Evaluation of known check cultivars and random examination of lower stems and roots provided confidence that whiteheads were caused predominately by Cephalosporium stripe. Disease notes were taken at each location approximately 2–3 week after heading, and over 2 consecutive days (one block per day) owing to the large number of entries. Developmental stage of the entries ranged from early milk to early dough at this time. Additional agronomic traits were also recorded to study possible association with Cephalosporium stripe resistance (club and common head type and presence of awns in all environments; physiological maturity and plant height at maturity for Pendleton 2007 only).

Statistical analyses

Whitehead percentages were square root-transformed to meet the assumptions of normality and homogeneity of variance. Tests of significance of RIL, replication, environment, and RIL $\times$ environment effects were performed with Type III F statistics estimated by PROC GLM of the Statistical Analysis System (SAS) (SAS v9.1, SAS Institute Inc., Cary, NC, USA). For analyses over environments, replications were considered nested within environment. The presence of significant transgressive segregants was verified in SAS by least squares means comparison tests adjusted by the Dunnett–Hsu method to control the error rate for multiple comparisons. The parents of the RIL population were used as the controls for this test.

Narrow-sense heritability (h^2) was calculated on a RIL mean basis for single environments and across environments, using an approach similar to that of Fernandez et al. (2008) and Tang et al. (2006). The REML method of SAS PROC VARCOMP was used to estimate variance components. For single-year data, $h^2 = \sigma^2_G/\left(\sigma^2_G + \sigma^2_E/r\right)$, where r is the number of repetitions, σ^2_G is the genotypic variance, and σ^2_E is the residual variance. For combined environments, $h^2 = \sigma^2_G/\left(\sigma^2_G + \sigma^2_{GL}/1 + \sigma^2_E/lr\right)$, where l is number of locations, r is the number of repetitions per location, σ^2_G is the genotypic variance, σ^2_{GL} is the variance due to genotype $\times$ location interaction, and σ^2_E is the residual variance. Exact 90% confidence intervals were calculated for single-environment h^2 estimates following the method described by Knapp et al. (1985).

Genotyping

'Coda' and 'Brundage' were evaluated at the Western Regional Genotyping Center (Pullman, Washington), Oregon State University, and the University of Idaho, using PCR to identify polymorphic markers. The RILs were scored with 158 polymorphic SSR markers. In addition, the Coda $\times$ Brundage population was sent for DArT analysis (Diversity Array

Technologies, Triticarte Pty. Ltd. Canberra, Australia). DNA extraction was done from fresh young leaf tissue using the DNeasy 96 Plant DNA extraction Kit (QIAGEN) following their protocol. While 233 DArT markers were identified, at first only 180 were used to develop an initial linkage map. The remaining DArT markers, of lower quality, were progressively incorporated into the map.

Linkage map construction was performed using JoinMap 4.0 (Van Ooijen and Kyazma 2006). Markers with high segregation distortion or with more than 50% missing values were excluded from the map. Exclusion of other markers from the map was based on their goodness-of-fit Chi-square contribution. Map distances are given in centiMorgan (Kosambi function). The linkage map used for QTL analysis was adjusted to obtain evenly spaced markers throughout the genome and clusters of markers were reduced to only one marker per locus.

QTL analysis

QTL analysis was performed using the composite interval mapping (CIM) procedure (Zeng 1994) implemented in WinQTL Cartographer v.2.5 (Wang et al. 2007). CIM analyses were performed on least square means for RILs estimated by SAS PROC GLM for each environment independently and for the combined data across locations. Model 6 of Win QTL Cartographer was used and up to ten cofactors for CIM were chosen using a stepwise forward–backward regression method, with a significance threshold of 0.05. Walk speed was set to 2 cM and the scan window to 10 cM beyond the markers flanking the interval tested. Significance likelihood ratio test (LR) thresholds for QTL identification were established by 1,000 permutations at $\alpha = 0.05$. The additive effects and coefficients of determination for individual QTL were estimated by CIM. Results from CIM with the combined data were used to estimate possible epistatic effects between significant QTL. The multiple interval mapping (MIM) procedure (Kao et al. 1999) implemented in Win QTL Cartographer was used for this purpose. In order to verify the additivity of QTL effects, RILs were classified according to the number of resistance alleles present at each of the QTL detected with combined data, and regression analysis (PROC GLM) was used to estimate the change in disease incidence associated with an increasing number of resistance alleles.

Results

Phenotypic evaluation and statistical analysis

Cephalosporium stripe pressure was moderately severe in all field trials, with the percentage of whiteheads on the susceptible check Stephens averaging 34, 58, and 31% in Moro 2007, Pendleton 2007, and Pendleton 2008, respectively. Brundage, the susceptible parent of the RIL population, possesses some resistance to the disease, as its mean whitehead percentage (Fig. 1) was always less than that of Stephens. A wide range in disease ratings for the RILs was observed and transformed disease values appeared to be normally distributed in all three environments (Fig. 1).

Combined analysis of variance of disease response showed significant influence of environments, RILs, and RIL by environment interaction ($P < 0.01$) (Table 1). Mean squares for the interaction terms were relatively small (29%) relative to the main effect of RIL; thus, combined analyses over environments were considered to be informative. The parents differed for mean whiteheads, and the RIL population mean was intermediate between the two parents. Averaged over environments, the mean percent whiteheads (square-root transformed) was 4.39 (SE = 0.15) for Brundage, 2.73 (SE = 0.15) for Coda, and 3.23 (SE = 0.020) for the RILs. The mean whiteheads of the individual RILs averaged over environments ranged from 0.788 to 5.96.

Significant differences among RILs were observed for whiteheads in each environment ($P < 0.01$) (Table 1). Disease responses of the parents were consistent between years at Pendleton. Coda had significantly lower disease

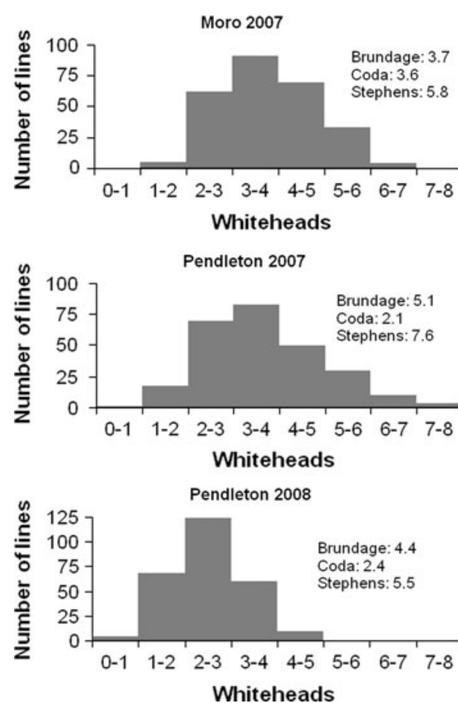

Fig. 1 Frequency distributions of Cephalosporium stripe response (square root of percent whiteheads) for 268 recombinant inbred lines of wheat derived from a cross between the cultivars Coda and Brundage, in three field environments; the cultivar Stephens was a susceptible check

Table 1 Analyses of variance, coefficients of variation (CV), and heritability estimates (h^2) for percentage whiteheads for a population of 268 recombinant inbred wheat lines exposed to Cephalosporium stripe disease in three field environments (Moro 2007, Pendleton 2007, and Pendleton 2008)

Environment		
Source of variation	DF	Mean square[a]
Combined		
Rep(Env)	4	3.25**
Environment	2	337.16**
RIL	267	5.06**
RIL × Env	534	1.48**
Error	1,066[b]	0.72
CV (%)		26.17
h^2		0.71
Moro 2007		
Rep	1	0.43
RIL	267	2.26**
Error	267	0.92
CV (%)		25.14
h^2 (90% CI)		0.59 (0.50–0.67)
Pendleton 2007		
Rep	1	1.75
RIL	267	3.62**
Error	266	0.77
CV (%)		23.85
h^2 (90% CI)		0.79 (0.74–0.83)
Pendleton 2008		
Rep	2	5.41**
RIL	267	2.00**
Error	532	0.58
CV (%)		30.09
h^2 (90% CI)		0.71 (0.66–0.76)

* Significant at the 0.05 probability level

** Significant at the 0.01 probability level

[a] Whitehead percentages were square root-transformed prior to analysis

[b] Due to 1 missing value, whiteheads had only 1,065 degrees of freedom

scores than Brundage. At Moro, disease ratings of Coda and Brundage were not significantly different ($P = 0.695$). Mean and ranges of whiteheads were similar in each environment (Fig. 1 and Supplementary Table 1).

One-tailed Dunnett tests for RILs compared with Coda and Brundage confirmed the presence of transgressive segregants. Averaged across environments, two lines had significantly lower average whiteheads than Coda ($P < 0.01$), while two lines had significantly higher mean whiteheads than Brundage ($P < 0.05$). These findings suggest that the more susceptible parent may carry genes that contribute to resistance.

Cephalosporium stripe response in Pendleton 2007 was weakly correlated with plant height ($r = 0.15$, $P < 0.001$) and maturity ($r = -0.17$, $P < 0.001$).

Heritability and 90% exact confidence intervals were calculated for each location and over locations (Table 1). Whitehead heritability estimates were moderately high, with a maximum of 0.79 at Pendleton 2007 and a minimum of 0.59 at Moro 2007.

Construction of linkage map

A total of 220 SSR markers were found to be polymorphic between Coda and Brundage. Of these, 158 were successfully used to genotype and differentiate individual RILs. A total of 233 polymorphic markers were identified using DArT marker technology. An initial genetic map was constructed based on 313 total markers. A total of 36 linkage groups were created, representing areas from all chromosomes except for chromosome 1D, which lacked a sufficient number of polymorphic markers. The total map length was 1879.3 cM, with an average interval between markers of 6.0 cM. Significant segregation distortion was observed for 18 markers, all in favor of Coda alleles and all mapped to the 1B chromosome.

For QTL analysis, the number of markers in the linkage map was reduced by eliminating co-segregating markers, leaving only one per cluster. Markers that showed high Chi-square contributions were also discarded from the map, leaving 204 well-distributed markers, with a total coverage of 1,910.2 cM and a mean distance of 9.4 cM between markers.

QTL identification

Composite interval mapping from the combined experiments identified QTL in seven chromosomal regions that were significantly associated with resistance to Cephalosporium stripe. LOD scores at the peak for these QTL ranged from 3.5 to 10.9, and all had similar additive effects on disease response. Three QTL were contributed by Coda, while four were contributed by Brundage, the more susceptible parent. The total amount of phenotypic variation explained jointly by all QTL for whitehead expression averaged 50.4%, but varied with the background markers used as cofactors (Table 2).

Four QTL for whiteheads were detected on chromosomes 2B, 2D (two QTL), and 4B accounting for 8.1, 10.7, 3.9, and 5.4% of the phenotypic variance, respectively (Table 2, Fig. 2). The first QTL on 2D was consistently mapped to the *Compactum* (*C*) locus, which controls spike compactness. The second QTL on 2D was located about 70 cM downstream of the first (Fig. 2). The first resistance allele was contributed by Coda and the second by

Table 2 Results of composite interval mapping (CIM) of Cephalosporium stripe response (square root of percentage whiteheads) over three environments

Trait	QTL name	Linkage group[a]	QTL peak position (cM)	1.0-LOD support interval (cM)	Closest marker	LOD	Additive effect[b]	R^{2c}
WH_SQRT	*QCs.orp-2B*	2B.1	8.0	1.5–16.0	wmc453-2B	4.5	−0.26	8.1
LOD[d] 3.02	*QCs.orp-2D.1*	2D	23.7	22.9–25.8	Compactum	10.9	−0.29	10.7
Total R^{2e}: 50.4	*QCs.orp-2D.2*	2D	94.0	90.4–96.3	barc206	3.5	0.18	3.9
SD: 2.8	*QCs.orp-4B*	4B.1	33.9	22.6–43.5	wpt-3908	5.3	0.21	5.4
	QCs.orp-5A.1	5A.2	22.0	15.5–23.9	wpt-3563-5A	6.3	0.25	7.7
	QCs.orp-5A.2	5A.3	24.6	22.5–26.3	Awns	4.5	0.20	4.9
	QCs.orp-5B	5B	100.4	90.7–107.4	gwm639-5B	4.7	−0.31	12.4

[a] Linkage groups correspond to chromosomes and Arabic numbers denote a specific linkage group within a chromosome

[b] Additive effects indicate an additive main effect of the parent contributing the higher value allele: positive values indicate that higher value alleles are from Coda (the more resistant parent) and negative values indicate that higher value alleles are from Brundage (the less resistant parent)

[c] Proportion of the phenotypic variance explained by the QTL after accounting for co-factors

[d] Threshold level to declare a significant QTL, based on 1,000 permutations

[e] Total phenotypic variance explained by all QTL after accounting for co-factors, and standard deviation

Brundage. The QTL on chromosome 4B, contributed by Brundage, also was consistent across environments. The estimated position of the QTL on 2B varied among environments and the combined analysis, although the resistance could be attributed to Coda.

Three additional QTL for whiteheads were located on two different linkage groups on 5A and one linkage group on 5B (Table 2, Fig. 2). The two QTL for resistance on 5A, both derived from Brundage, jointly accounted for 12.6% of the phenotypic variation. One of these 5A QTL co-located with the phenotypic marker for awns, which is regulated by the *B1* gene. The QTL for disease response mapped to chromosome 5B was revealed by the combined analysis only. Although LOD scans of this chromosome for Moro and Pendleton 2007 suggested a peak, it was below the threshold for statistical significance. *QCs.orp-5B* had a LOD score at the peak of 4.7 and accounted for the highest proportion of the phenotypic variance for whiteheads (12.4%). The allele conferring resistance at this QTL, derived from Coda, contributed the highest additive effect (−0.31).

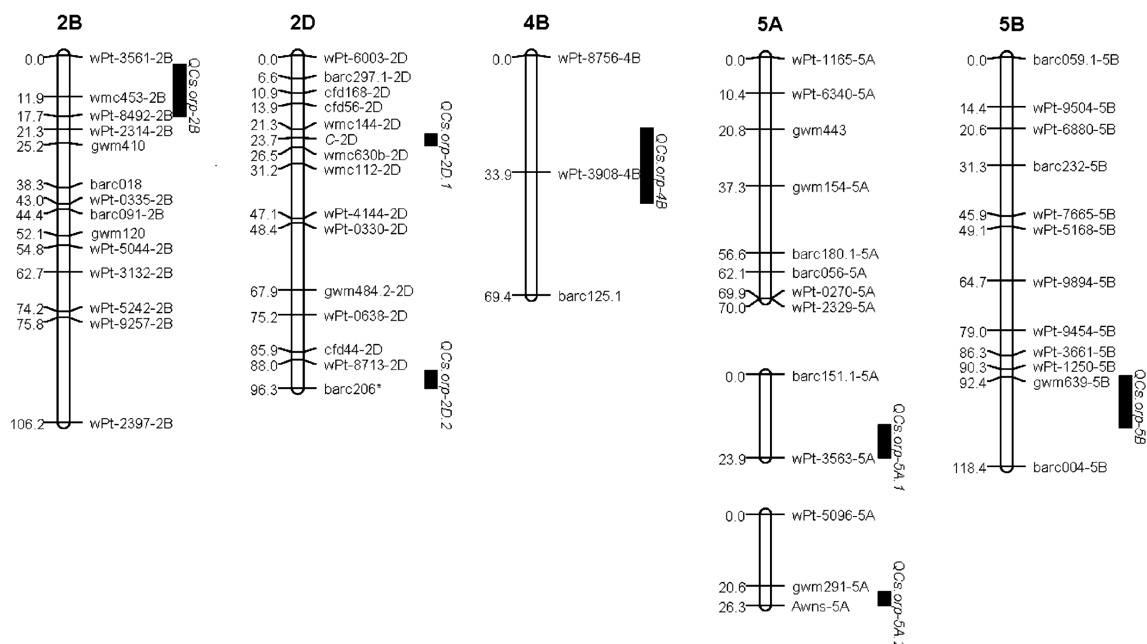

Fig. 2 Partial linkage map of the Coda x Brundage recombinant inbred line population ($n = 268$), with graphical presentation of significant QTL revealed by CIM on disease response (square root of percent whiteheads) over three combined field environments

Results of the multiple interval mapping analysis to account for possible interactions among identified QTL across environments revealed only non-significant QTL × QTL interactions (data not shown); therefore, these were not considered.

Effect of number of resistance alleles

Combinations of QTL for Cephalosporium stripe resistance could be associated with disease response in the RIL population. The regression of disease response on number of resistance alleles at significant QTL showed a significant ($P < 0.01$) slope of -0.41 and a coefficient of determination (R^2) of 0.38 (Fig. 3). The effect of pyramiding resistance QTL was not heavily affected by head morphology (presence/absence of compact heads and awns), since similar trends were observed for the four head type classes (Fig. 3). In addition, RILs within each level of number of resistance alleles showed higher levels of variability than can be explained by head morphology traits alone.

Discussion

The Coda × Brundage RIL population was initially developed for mapping genetic loci for resistance to *Puccinia striiformis* (stripe rust) and *O. yallundae* and *O. acuformis* (eyespot) (Leonard et al. 2008), as well as various agronomic traits. Both parents possess some resistance, but have different genetic backgrounds. Field experiments showed a wide range of variability for Cephalosporium stripe response in the Coda × Brundage RIL population. Despite the high number of RILs observed with mean trait values numerically higher or lower than Coda or Brundage,

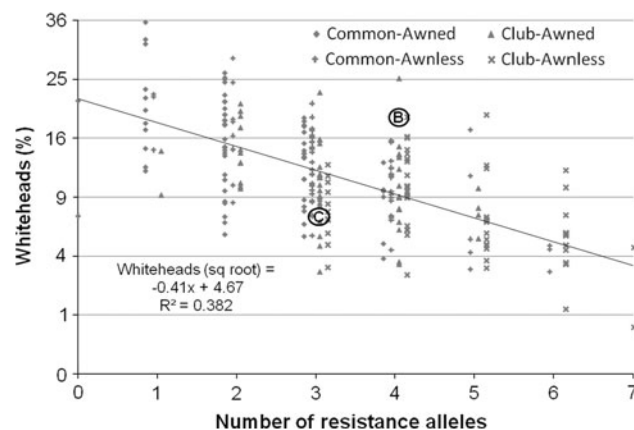

Fig. 3 Regression of least squares means of percent whiteheads caused by Cephalosporium stripe on number of resistance alleles present at 7 QTL for a population of 268 recombinant inbred lines derived from a cross between the wheat cultivars Coda (C) and Brundage (B)

few transgressive segregants were statistically confirmed. Nevertheless, it appears that both parents carry genes for resistance against Cephalosporium stripe. Disease response was found to be associated statistically with plant height and maturity in the Pendleton 2007 trial, but correlations were very weak. In addition, graphical examination of the data showed high levels of variation for disease response at all maturity levels and plant heights (data not shown).

Quantitatively expressed disease resistance is often considered to be inherited polygenically. Reviews of published studies, however, suggest a median QTL number of only three (Young 1996) or four (Kover and Caicedo 2001), often with one or two QTL accounting for a large proportion of the total variance in disease response. These results are consistent with previous studies using traditional quantitative genetic approaches (Geiger and Heun 1989). However, limitations in population size or phenotyping (Vales et al. 2005) may bias estimates of QTL number downwards (Kover and Caicedo 2001; Young 1996). Our study fits more with the exceptions, as we identified seven putative QTL associated with resistance to Cephalosporium stripe and no single QTL accounted for a large proportion of the total variation. Some researchers had hypothesized that resistance to soil-borne pathogens would be genetically similar to that of specialized, foliar pathogens (e.g., Ellingboe 1983), while others suggested that it may be more complex (e.g., Bruehl 1983). Several recent QTL studies of resistance to soil-borne pathogenic fungi have identified only 1–4 QTL for resistance (Hernandez-Delgado et al. 2009; Kim et al. 2008; Lein et al. 2008; Li et al. 2009; Rygulla et al. 2008; Taguchi et al. 2009), while two others identified a larger number of QTL (Bovill et al. 2010; Wang et al. 2008). The moderately high heritability estimates obtained in this study were similar to previous estimates from segregating breeding populations (unpublished). Such levels of heritability for resistance are common for other soil-borne pathogens (Li et al. 2009; Schneider et al. 2001) as well and reinforce the likelihood of success in breeding for resistance.

Of the seven QTL for Cephalosporium stripe identified, two were associated with head morphology traits. The first region on chromosome 2D (*QCs.orp-2D.1*) was centered on the *Compactum* gene (*C* locus), which defines ear compaction, among other spike morphology traits (Gul and Allan 1972; Zwer et al. 1995). This locus was mapped by Johnson et al. (2008) to the centromeric region of chromosome 2D and, thus, could be controlled by closely linked resistance gene(s), rather than due to pleiotropic effects of the *Compactum* gene. Bruehl (1983) proposed that non-specific resistance to soil-borne pathogens frequently involved changes in fundamental physiological processes within the host. This is in contrast to highly specialized pathogens where, in most cases, the resistance

genes have no known role in the basic physiology of the plant.

Influences of the *Compactum* gene at various developmental stages were determined with co-isogenic lines by Tsunewaki and Koba (1979). This gene was associated with increased node diameter, number of spikelets per ear, and spike density, but decreased lengths of ear rachis and grains, and decreased grain index (grain length/thickness). Mathre and Johnston (1990), suggested that the superior level of resistance to *C. gramineum* found in related wheat species (*Thinopyron intermedium* and *Elytrigia elongata*) was due to restricted movement of the pathogen through the roots, and particularly through crown tissues. The transition zone from the roots to culm tissue was suggested as the morphological structure presenting a potential barrier to the pathogen's movement. An indirect effect of the *Compactum* gene, as suggested by Tsunewaki and Koba (1979), could be an increased complexity of the root-crown transition zone that decreases the pathogen's ability to enter the aboveground portion of the plant and limit its access to the vascular system for movement and colonization. If this is the case, then club wheat germplasm would have an inherent resistance and relative advantage over common winter wheat. However, the compactum gene alone is not sufficient to confer adequate levels of resistance in absence of other QTL.

The QTL for resistance identified on chromosome 5A was associated with the region that determines presence or absence of awns. In fact, the closest locus to *QCs.orp-5A.2* was the phenotypic marker for presence of awns. This QTL explained 5% of the phenotypic variance for whiteheads, which is similar to the proportion explained by the other QTL. Resistance was associated with the awnless trait from Brundage. This trait is regulated by the *B1* gene, located on the distal part of the long arm of chromosome 5A (Kato et al. 1998). No evidence is available to determine if *B1* has a pleiotropic effect on Cephalosporium stripe resistance, or if the observed effect is due to strong linkage between the morphological trait and a resistance factor. *B1* is an awn development inhibitor, but it also affects grain index and kernel thickness (Tsunewaki and Koba 1979). A variety of resistance genes and QTL have been mapped to chromosome 5A (stripe rust, Fusarium head blight, powdery mildew, leaf and glume blotch, Hessian fly, and cereal cyst nematode), as well as heading date and lodging resistance (Gupta et al. 2008; Lillemo et al. 2008).

CIM analysis with the whitehead trait combined over locations identified a particularly interesting QTL on chromosome 5B, attributed to Coda. *QCs.orp-5B* had the highest additive effect (0.31) and explained the highest proportion of the phenotypic variability for disease response (12.4%). Numerous mapping studies have placed disease resistance genes on chromosome 5B, e.g., *Lr52* for leaf rust and *Pm30* for powdery mildew, as well as resistance QTL for stripe rust and Fusarium head blight (Gupta et al. 2008; Lillemo et al. 2008). For this study, the most relevant trait found to be controlled by chromosome 5B is tolerance to a specific host-selective toxin (HST) produced by *Pyrenophora tritici-repentis* (tan spot) and *Stagonospora nodorum* (Stagonospora nodorum blotch) known as ToxA and SnToxA, respectively (Faris et al. 1996; Friesen et al. 2006; Tomas et al. 1990). Evidence suggests that *P. tritici-repentis* acquired the ability to produce the toxin through a recent horizontal gene transfer event from *S. nodorum* (Friesen et al. 2006). *Tsn1* has been postulated to govern sensitivity to both toxins, and therefore serves as a major determinant for susceptibility to both Stagonospora nodorum blotch and tan spot (Liu et al. 2006). However, Gonzalez-Hernandez et al. (2009) reported a QTL proximal to *Tsn1* in durum wheat and indicated that resistance to each disease was controlled by different but linked locus. Graminin A is a toxic compound produced by *C. gramineum* that has been implicated in the proliferation and development of the fungus in wheat (Creatura et al. 1981; Kobayashi and Ui 1979; Rahman et al. 2001; Van Wert et al. 1984). This relationship opens the possibility that the region on 5B might have evolved some kind of specialization to resist the infection of fungal pathogens that rely on toxins or toxin-like compounds to enhance pathogenesis.

In addition to the two QTL associated with head morphology markers, four other QTL for Cephalosporium stripe resistance were mapped to chromosomes 2B, 2D, 4B, and 5A. Those on 2D and 5A were unlinked to the other resistance QTL on the same chromosomes. These explained between 3.9 and 8.1% of the variance for whiteheads. Resistance genes and QTL for many wheat diseases and pests have been mapped to these chromosomes, including leaf rust, stripe rust, Fusarium head blight, powdery mildew, Septoria tritici blotch, wheat streak mosaic virus, Hessian fly, and cereal cyst nematode (Gupta et al. 2008; Lillemo et al. 2008; Marshall et al. 2001). Agronomic characters such as heading date (*Ppd-B1*, *Eps*), plant height, spike length and compactness, grain weight, and coleoptile growth were reported to be controlled, at least in part, by factors located on 2B (Gupta et al. 2008; Kumar et al. 2006; Marshall et al. 2001; Marza et al. 2006; Rebetzke et al. 2001; Sourdille et al. 2000). Resistance to lodging and stem strength QTL have been located on chromosome 2D (Gupta et al. 2008). Different authors have associated chromosome 4B with plant height (*Rht-B1*), coleoptile, internode, and peduncle length, and grain size (Gupta et al. 2008; Marshall et al. 2001; Rebetzke et al. 2001). It is currently unclear if any of these agronomic traits are directly associated with resistance to Cephalosporium stripe.

Of the seven resistance QTL identified by CIM, three were assigned to Coda and four to Brundage. Yet the cumulative effect of the QTL from Coda, based on additive effects, is higher than the cumulative effect of the QTL from Brundage. Regression analysis suggests that superior levels of resistance can be attained through accumulation of resistance QTL (Fig. 3). The relative importance of individual QTL was estimated by obtaining additive effects through CIM. These differences were not considered in the regression analysis. Class mean comparisons (Fisher's Protected least significant difference, $P < 0.05$) suggest that there are three distinct levels of resistance. Susceptible types possess 0, 1 or 2 resistant alleles, with class means approximately 4.0 or higher, and are not statistically different from each other. The intermediate types, with mean whitehead scores in the range of 3.0–3.5, possess 3 or 4 resistance alleles with class means that are not significantly different from each other. The resistant types, with 5, 6 or 7 resistance alleles, showed the lowest mean level of disease. There was no difference in disease response among classes with 5, 6, or 7 alleles. The two transgressive segregants with whitehead scores significantly lower than Coda had either 6 or 7 resistance alleles. Head type morphology did not have a substantial influence on the observed regression between QTL number and disease response, however, and there was substantial variability among RILs within each head morphology class (Fig. 3).

This study indicates that an acceptable level of resistance to Cephalosporium stripe requires combining at least three to four resistance QTL, followed by evaluation in infested fields to confirm performance, as it was shown that high levels of variability are present within each class of number of accumulated resistance alleles. Validation of QTL identified in this study is essential (Collard et al. 2005; Melchinger et al. 2004) prior to implementing a marker-assisted selection program, however, since these results are based on one population only. For example, Bovill et al. (2010) recently found that only a minority of QTL identified for resistance to Fusarium crown rot of wheat were effective in other genetic backgrounds. It thus will be important to confirm that QTL and genomic regions identified for Cephalosporium stripe resistance perform similarly in other genetic backgrounds to assure broad applicability over a diverse range of germplasm. New mapping populations are being phenotyped in similar field experiments and extensive genotyping is in progress to address these issues.

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References

Allan RE, Morris CF, Line RF, Anderson JA, Walker-Simmons MK, Donaldson E (2000) Registration of 'Coda' club wheat. Crop Sci 40:578–579

Bailey JE, Lockwood JL, Wiese MV (1982) Infection of wheat by *Cephalosporium gramineum* as influenced by freezing of roots. Phytopathology 72:1324–1328

Blank CA, Murray TD (1998) Influence of pH and matric potential on germination of *Cephalosporium gramineum* conidia. Plant Dis 82:975–978

Bockus WW, O'Connor JP, Raymond PJ (1983) Effect of residue management method on incidence of Cephalosporium stripe under continuous winter wheat production. Plant Dis 67:1323–1324

Bockus WW, Davis MA, Todd TC (1994) Grain-yield responses of winter wheat coinoculated with *Cephalosporium gramineum* and *Gaeumannomyces graminis* var *tritici*. Plant Dis 78:11–14

Bovill WD, Horne M, Herde D, Davis M, Wildermuth GB, Sutherland MW (2010) Pyramiding QTL increases seedling resistance to crown rot (*Fusarium pseudograminearum*) of wheat (*Triticum aestivum*). Theor Appl Genet 121:127–136

Bruehl GW (1956) Cephalosporium stripe disease of wheat in Washington. Phytopathology 46:178–180

Bruehl GW (1957) Cephalosporium stripe disease of wheat. Phytopathology 47:641–649

Bruehl GW (1983) Nonspecific genetic-resistance to soilborne fungi. Phytopathology 73:948–951

Bruehl GW, Lai P (1968) Influence of soil pH and humidity on survival of *Cephalosporium gramineum* in infested wheat straw. Can J Plant Sci 48:245–252

Bruehl GW, Murray TD, Allan RE (1986) Resistance of winter wheats to Cephalosporium stripe in the field. Plant Dis 70:314–316

Collard BCY, Jahufer MZZ, Brouwer JB, Pang ECK (2005) An introduction to markers, quantitative trait loci (QTL) mapping and marker-assisted selection for crop improvement: the basic concepts. Euphytica 142:169–196

Creatura PJ, Safir GR, Scheffer RP, Sharkey TD (1981) Effects of *Cephalosporium gramineum* and a toxic metabolite on stomatal conductance of wheat. Physiol Plant Pathol 19:313–323

Douhan GW, Murray TD (2001) Infection of winter wheat by a beta-glucuronidase-transformed isolate of *Cephalosporium gramineum*. Phytopathology 91:232–239

Edwards JD, McCouch SR (2007) Molecular markers for use in plant molecular breeding and germplasm evaluation. In: Guimaraes E, Ruane J, Scherf B, Sonnino A, Dargie J (eds) Guimarês. Marker-assisted selection: current status and future perspectives in crops, livestock, forestry and fish. FAO, Rome, pp 29–49

Ellingboe AH (1983) Genetic aspects of interaction between plant hosts and their soilborne pathogens. Phytopathology 73:941–944

Ellis JB, Everhart BM (1894) New species of fungi from various localities. Proc Acad Nat Sci Phila 46:322–386

Faris JD, Anderson JA, Francl LJ, Jordahl JG (1996) Chromosomal location of a gene conditioning insensitivity in wheat to a necrosis-inducing culture filtrate from *Pyrenophora tritici-repentis*. Phytopathology 86:459–463

Fernandez MGS, Hamblin MT, Li L, Rooney WL, Tuinstra MP, Kresovich S (2008) Quantitative trait loci analysis of endosperm color and carotenoid content in sorghum grain. Crop Sci 48:1732–1743

Friesen TL, Stukenbrock EH, Liu ZH, Meinhardt S, Ling H, Faris JD, Rasmussen JB, Solomon PS, McDonald BA, Oliver RP (2006)

Emergence of a new disease as a result of interspecific virulence gene transfer. Nat Genet 38:953–956

Geiger HH, Heun M (1989) Genetics of quantitative resistance to fungal diseases. Annu Rev Phytopathol 27:317–341

Gonzalez-Hernandez JL, Singh PK, Mergoum M, Adhikari TB, Kianian SF, Simsek S, Elias EM (2009) A quantitative trait locus on chromosome 5B controls resistance of *Triticum turgidum* (L.) var. *diccocoides* to *Stagonospora nodorum* blotch. Euphytica 166:199–206

Gul A, Allan RE (1972) Relation of club gene with yield and yield components of near-isogenic wheat lines. Crop Sci 12:297–301

Gupta PK, Varshney RK, Sharma PC, Ramesh B (1999) Molecular markers and their applications in wheat breeding. Plant Breed 118:369–390

Gupta PK, Mir RR, Mohan A, Kumar J (2008) Wheat genomics: present status and future prospects. Int J Plant Genomics 2008:1–36

Hernandez-Delgado S, Reyes-Valdes MH, Rosa R, Mayek-Perez N (2009) Molecular markers associated with resistance to *Macrophomina phaseolina* (Tassi) Goid. in common bean. J Plant Pathol 91:163–170

Horvath DP, Dahleen LS, Stebbing JA, Penner G (1995) A codominant PCR-based marker for assisted selection of durable stem rust resistance in barley. Crop Sci 35:1445–1450

Johnson EB, Nalam VJ, Zemetra RS, Riera-Lizarazu O (2008) Mapping the compactum locus in wheat (*Triticum aestivum* L.) and its relationship to other spike morphology genes of the Triticeae. Euphytica 163:193–201

Johnston RH, Mathre DE (1972) Effect of infection by *Cephalosporium gramineum* on winter wheat. Crop Sci 12:817–819

Kao CH, Zeng ZB, Teasdale RD (1999) Multiple interval mapping for quantitative trait loci. Genetics 152:1203–1216

Kato K, Miura H, Akiyama M, Kuroshima M, Sawada S (1998) RFLP mapping of the three major genes, Vrn1, Q and B1, on the long arm of chromosome 5A of wheat. Euphytica 101:91–95

Kim HJ, Nahm SH, Lee HR, Yoon GB, Kim KT, Kang BC, Choi D, Kweon O, Cho MC, Kwon JK, Han JH, Kim JH, Park M, Ahn J, Choi S, Her N, Sung JH, Kim BD (2008) BAC-derived markers converted from RFLP linked to *Phytophthora capsici* resistance in pepper (*Capsicum annuum* L.). Theor Appl Genet 118:15–27

Knapp SJ, Stroup WW, Ross WM (1985) Exact confidence intervals for heritability on a progeny mean basis. Crop Sci 25:192–194

Kobayashi K, Ui T (1979) Phytotoxicity and anti-microbial activity of graminin A, produced by *Cephalosporium gramineum*, the causal agent of Cephalosporium stripe disease of wheat. Physiol Plant Pathol 14:129–133

Kover PX, Caicedo AL (2001) The genetic architecture of disease resistance in plants and the maintenance of recombination by parasites. Mol Ecol 10:1–16

Kumar N, Kulwal PL, Gaur A, Tyagi AK, Khurana JP, Khurana P, Balyan HS, Gupta PK (2006) QTL analysis for grain weight in common wheat. Euphytica 151:135–144

Lai P, Bruehl GW (1966) Survival of *Cephalosporium gramineum* in naturally infested wheat straws in soil in the field and in the laboratory. Phytopathology 56:213–218

Latin RX, Harder RW, Wiese MV (1982) Incidence of Cephalosporium stripe as influenced by winter wheat management practices. Plant Dis 66:229–230

Lein JC, Sagstetter CM, Schulte D, Thurau T, Varrelmann M, Saal B, Koch G, Borchardt DC, Jung C (2008) Mapping of Rhizoctonia root rot resistance genes in sugar beet using pathogen response-related sequences as molecular markers. Plant Breed 127:602–611

Leonard JM, Watson CJW, Carter AH, Hansen JL, Zemetra RS, Santra DK, Campbell KG, Riera-Lizarazu O (2008) Identification of a candidate gene for the wheat endopeptidase Ep-D1 locus and two other STS markers linked to the eyespot resistance gene Pch1. Theor Appl Genet 116:261–270

Li HJ, Conner RL, Murray TD (2008) Resistance to soil-borne diseases of wheat: contributions from the wheatgrasses *Thinopyrum intermedium* and *Th. ponticum*. Can J Plant Sci 88:195–205

Li HB, Zhou MX, Liu CJ (2009) A major QTL conferring crown rot resistance in barley and its association with plant height. Theor Appl Genet 118:903–910

Lillemo M, Asalf B, Singh RP, Huerta-Espino J, Chen XM, He ZH, Bjornstad A (2008) The adult plant rust resistance loci Lr34/Yr18 and Lr46/Yr29 are important determinants of partial resistance to powdery mildew in bread wheat line Saar. Theor Appl Genet 116:1155–1166

Liu ZH, Friesen TL, Ling H, Meinhardt SW, Oliver RP, Rasmussen JB, Faris JD (2006) The Tsn1-ToxA interaction in the wheat-*Stagonospora nodorum* pathosystem parallels that of the wheat-tan spot system. Genome 49:1265–1273

Marshall DR, Langridge P, Appels R (2001) Wheat breeding in the new century—preface. Aust J Agric Res 52:1–4

Martin JM, Mathre DE, Johnston RH (1983) Genetic variation for reaction to *Cephalosporium gramineum* in four crosses of winter wheat. Can J Plant Sci 63:623–630

Martin JM, Mathre DE, Johnston RH (1986) Winter wheat genotype responses to *Cephalosporium gramineum* inoculum levels. Plant Dis 70:421–423

Martin JM, Johnston RH, Mathre DE (1989) Factors affecting the severity of Cephalosporium stripe of winter wheat. Can J Plant Pathol 11:361–367

Marza F, Bai GH, Carver BF, Zhou WC (2006) Quantitative trait loci for yield and related traits in the wheat population Ning7840 × Clark. Theor Appl Genet 112:688–698

Mathre DE, Johnston RH (1975a) Cephalosporium stripe of winter wheat—procedures for determining host response. Crop Sci 15:591–594

Mathre DE, Johnston RH (1975b) Cephalosporium stripe of winter wheat: infection processes and host response. Phytopathology 65:1244–1249

Mathre DE, Johnston RH (1990) A crown barrier related to Cephalosporium stripe resistance in wheat relatives. Can J Bot 68:1511–1514

Mathre DE, Johnston RH, McGuire CF (1977) Cephalosporium stripe of winter wheat—pathogen virulence, sources of resistance, and effect on grain quality. Phytopathology 67:1142–1148

Mathre DE, Johnston RH, Martin JM (1985) Sources of resistance to *Cephalosporium gramineum* in *Triticum* and *Agropyron* species. Euphytica 34:419–424

Melchinger AE, Utz HF, Schon GC (2004) QTL analyses of complex traits with cross validation, bootstrapping and other biometric methods. Euphytica 137:1–11

Morton JB, Mathre DE (1980a) Identification of resistance to Cephalosporium stripe in winter wheat. Phytopathology 70:812–817

Morton JB, Mathre DE (1980b) Physiological effects of *Cephalosporium gramineum* on growth and yield of winter wheat cultivars. Phytopathology 70:807–811

Mundt CC (2002) Performance of wheat cultivars and cultivar mixtures in the presence of Cephalosporium stripe. Crop Prot 21:93–99

Murray T (2006) Seed transmission of *Cephalosporium gramineum* in winter wheat. Plant Dis 90:803–806

Murray TD, Walter CC, Anderegg JC (1992) Control of Cephalosporium stripe of winter wheat by liming. Plant Dis 76:282–286

Nisikado Y, Matsumoto H, Yamuti K (1934) Studies on a new *Cephalosporium*, which causes the stripe disease of wheat.

Bericht des Ohara Instituts fur Landwirtschaftliche Forschungen 6:275–306

Pool RAF, Sharp EL (1969) Some environmental and cultural factors affecting Cephalosporium stripe of winter wheat. Plant Dis Reptr 53:898–902

Rahman M, Mundt CC, Wolpert TJ, Riera-Lizarazu O (2001) Sensitivity of wheat genotypes to a toxic fraction produced by *Cephalosporium gramineum* and correlation with disease susceptibility. Phytopathology 91:702–707

Raymond PJ, Bockus WW (1984) Effect of seeding date of winter wheat on incidence, severity, and yield loss caused by Cephalosporium stripe in Kansas. Plant Dis 68:665–667

Rebetzke GJ, Appels R, Morrison AD, Richards RA, McDonald G, Ellis MH, Spielmeyer W, Bonnett DG (2001) Quantitative trait loci on chromosome 4B for coleoptile length and early vigour in wheat (*Triticum aestivum* L.). Aust J Agric Res 52:1221–1234

Richardson MJ, Rennie WJ (1970) An estimate of the loss of yield caused by *Cephalosporium gramineum* in wheat. Plant Pathol 19:138–140

Rygulla W, Snowdon RJ, Friedt W, Happstadius I, Cheung WY, Chen D (2008) Identification of quantitative trait loci for resistance against *Verticillium longisporum* in oilseed rape (*Brassica napus*). Phytopathology 98:215–221

Schneider KA, Grafton KF, Kelly JD (2001) QTL analysis of resistance to Fusarium root rot in bean. Crop Sci 41:535–542

Slope DB, Bardner R (1965) Cephalosporium stripe of wheat and root damage by insects. Plant Pathol 14:184–187

Sourdille P, Tixier MH, Charmet G, Gay G, Cadalen T, Bernard S, Bernard M (2000) Location of genes involved in ear compactness in wheat (*Triticum aestivum*) by means of molecular markers. Mol Breed 6:247–255

Spalding DH, Bruehl GW, Foster RJ (1961) Possible role of pectinolytic enzymes and polysaccharide in pathogenesis by *Cephalosporium gramineum* in wheat. Phytopathology 51:227–235

Specht LP, Murray TD (1990) Effects of root-wounding and inoculum density on Cephalosporium stripe in winter wheat. Phytopathology 80:1108–1114

Taguchi K, Ogata N, Kubo T, Kawasaki S, Mikami T (2009) Quantitative trait locus responsible for resistance to Aphanomyces root rot (black root) caused by *Aphanomyces cochlioides* Drechs. in sugar beet. Theor Appl Genet 118:227–234

Tang SX, Leon A, Bridges WC, Knapp SJ (2006) Quantitative trait loci for correlated seed traits are tightly linked to branching and pericarp pigment loci in sunflower. Crop Sci 46:721–734

Tomas A, Feng GH, Reeck GR, Bockus WW, Leach JE (1990) Purification of a cultivar-specific toxin from *Pyrenophora tritici-repentis*, causal agent of tan spot of wheat. Mol Plant Microbe Interact 3:221–224

Tsunewaki K, Koba T (1979) Production and genetic-characterization of the co-isogenic lines of a common wheat *Triticum aestivum* CV. S-615 for ten major genes. Euphytica 28:579–592

Vales MI, Schon CC, Capettini F, Chen XM, Corey AE, Mather DE, Mundt CC, Richardson KL, Sandoval-Islas JS, Utz HF, Hayes PM (2005) Effect of population size on the estimation of QTL: a test using resistance to barley stripe rust. Theor Appl Genet 111:1260–1270

Van Ooijen JW, Kyazma BV (2006) JoinMap 4, Software for the calculation of genetic linkage maps in experimental populations. Wageningen, Netherlands

Van Wert SL, Ravenscroft AV, Fulbright DW (1984) Screening wheat lines as seedlings for resistance to *Cephalosporium gramineum*. Plant Dis 68:1036–1038

Wang S, Basten CJ, Zeng Z-B (2007) Windows QTL Cartographer 2.5. Department of Statistics, North Carolina State University, Raleigh

Wang HM, Lin ZX, Zhang XL, Chen W, Guo XP, Nie YC, Li YH (2008) Mapping and quantitative trait loci analysis of Verticillium wilt resistance genes in cotton. J Integr Plant Biol 50:174–182

Wiese MV (1987) Compendium of wheat diseases, 2nd edn. APS Press, St. Paul

Xu YB, Crouch JH (2008) Marker-assisted selection in plant breeding: from publications to practice. Crop Sci 48:391–407

Young ND (1996) QTL mapping and quantitative disease resistance in plants. Annu Rev Phytopathol 34:479–501

Zemetra RS, Souza EJ, Lauver M, Windes J, Guy SO, Brown B, Robertson L, Kruk M (1998) Registration of 'Brundage' wheat. Crop Sci 38:1404

Zeng ZB (1994) Precision mapping of quantitative trait loci. Genetics 136:1457–1468

Zwer PK, Sombrero A, Rickman RW, Klepper B (1995) Club and common wheat yield component and spike development in the Pacific Northwest. Crop Sci 35:1590–1597