Identification and Fine Mapping of *qSCR4.01*, a Novel Major QTL for Resistance to *Puccinia polysora* in Maize

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Abstract

Southern corn rust (SCR) is a prevalent foliar disease that can lead to severe yield losses in maize. Growing SCR-resistant varieties is the most effective way to control the disease. To identify major quantitative trait loci (QTLs) for SCR resistance, a recombinant inbred line population derived from a cross between CIMBL83 (resistant) and Lx9801 (susceptible) was analyzed. The resistance to SCR had high heritability within the population, and a major QTL on chromosome 4 (*qSCR4.01*), which can explain 48 to 65% of the total phenotypic variation, was

consistently detected across multiple environments. Using a progeny-based fine-mapping strategy, we delimited *qSCR4.01* to an interval of ~770 kb. In contrast to other major QTLs for SCR resistance previously reported on the short arm of chromosome 10, *qSCR4.01* is a novel QTL and, therefore, a desirable source of SCR resistance in maize breeding programs.

Keywords: maize, southern corn rust, novel QTL, fine mapping

Southern corn rust (SCR), caused by the fungus *Puccinia polysora* Underw., is a destructive foliar disease in maize that has been frequently reported in tropical and subtropical areas (Raid et al. 1988). As the results of climate change on a global level, SCR has gradually spread to temperate regions in recent years (Ramirez-Cabral et al. 2017). During infection, reddish-orange pustules appear on the upper surface of leaves and become progressively worse as plants mature. In severe cases, pustules can be found on the husk, stalk, and sheath, which may lead to the death of infected tissues and prematurely desiccated plants (Rodriguez-Ardon et al. 1980). Significant yield losses due to SCR outbreaks have been reported worldwide during the last few decades (Agarwal et al. 2001; Liu et al. 2003; Raid et al. 1988; Rodriguez-Ardon et al. 1980). Disease management strategies aimed at reducing fungal inoculum, such as crop rotation and fungicides, have been applied to manage SCR; however, the most effective means of control is the breeding and planting of resistant hybrids.

Both quantitative and race-specific resistance to SCR have been identified, and multiple quantitative trait loci (QTLs)/genes conferring SCR resistance have been reported. The first of these was *Rpp9*, a dominant gene located on the short arm of chromosome 10 that confers resistance to *P. polysora* race PP.9 (Ullstrup 1965). In the same region of chromosome 10, several closely linked SCR QTLs/genes have been reported from a diverse sets of maize germplasm, including qualitative resistance genes (*RppQ*, *RppD*, *RppP25*, and *RppS*) from Chinese germplasm (Wu et al. 2015; Zhang et al.

2010; Zhao et al. 2013; Zhou et al. 2007) and major QTLs from U.S. germplasm (Holland et al. 1998; Jines et al. 2007). In addition to the major QTLs/genes on chromosome 10S, QTLs with small effects or partial resistance to *P. polysora* have been reported on all maize chromosomes except chromosome 7 (Deng et al. 2019; Souza Camacho et al. 2019; Wanlayaporn et al. 2013; Zhou et al. 2018).

The deployment of major genes for SCR resistance has long been adopted in maize breeding programs. The best known of these is *Rpp9*, which was identified from the South African line PI186208 (Ullstrup 1965). *Rpp9* was used successfully in the northern United States to provide effective resistance against the prevalent race of *P. polysora*, PP.9, for more than 20 years (Brewbaker et al. 2011); however, *Rpp9* has been overcome by a new, virulent race of the pathogen (Dolezal et al. 2009). Another widely used *R* gene is *RppQ* from Qi319, which has been introduced into many hybrids in Chinese breeding programs since the 1990s. The resistance effect of Qi319, however, has gradually decreased in southern China as a result of the emergence of new pathogen races (Wang et al. 2019).

Due to the rapid evolution of the SCR pathogen, *P. polysora*, the continuous discovery of new resistance genes is required to protect maize and minimize yield losses. In this regard, we report a major QTL, *qSCR4.01*, conferring resistance to *P. polysora* in CIMBL83, a tropical inbred line from the International Maize and Wheat Improvement Center (CIMMYT). Located on chromosome 4, this is a novel QTL and, therefore, a desirable source of SCR resistance for maize breeding programs.

Materials and Methods

Plant materials. A mapping population of 117 F_{5:6} recombinant inbred lines (RILs) was developed by single-seed descent from the BC₁ population, (CIMBL83 × Lx9801) × Lx9801. The parental line CIMBL83, tropical germplasm introduced from CIMMYT, is completely resistant to *P. polysora*, whereas the susceptible line, Lx9801, derived from the cross 502 × H21, is an elite inbred line that has been used widely in maize breeding programs in China (Sun et al. 2014).

Field design and disease phenotyping. The RILs were planted at the Changge station in Henan Province in China during 2016 and 2017 and at the Ledong station in Hainan Province during 2017. All of the RILs were arranged in a randomized incomplete block design with two replications in the field.

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Natural infection has been confirmed as an effective method to initiate the occurrence of SCR in fields (Jines et al. 2007; Zhao et al. 2013). At the late milk stage (30 to 35 days after pollination), the plants were evaluated for SCR severity using a 1 to 9 rating scale (Deng et al. 2019), where a score of 1 indicated a highly resistant plant free of pustules and a score of 9 indicated a highly susceptible plant with leaves completely covered with rust pustules. The scores for each plot represented mean values for individuals within the plot and were used for subsequent analysis.

Data analysis. Standard analysis of variance (ANOVA) was performed using the Proc GLM procedure of SAS (SAS Institute, Cary, NC) to determine variation of SCR scoring among the RILs. The components of variance were estimated using a complete random effects model (Knapp et al. 1985). The broad-sense heritability (h^2) was calculated as follows: $h^2 = \sigma_g^2 / (\sigma_g^2 + \sigma_{ge}^2/n + \sigma_e^2/nr)$, where σ_g^2 , σ_{ge}^2 , and σ_e^2 were estimates of genotypic, genotype $\times$ environment interaction, and error variances, respectively, and n and r were the number of environments and replication per environment.

Linkage map construction. The parental lines and RILs were genotyped using the MaizeSNP9.4K BeadChip, which including 9,433 single nucleotide polymorphism (SNP) markers developed from the B73 reference sequence (<http://www.cgmb.com.cn/index.php/Home/Index>). Monomorphic SNPs were removed, and polymorphic SNPs were selected to construct the genetic map. Heterozygous SNPs were treated as missing data, and SNPs for each line with >20% missing data were removed. Markers significantly distorted from the expected segregation ratio ($P < 0.01$) were also excluded from the analysis. Linkage analysis was performed using the map function of the IciMapping software (Meng et al. 2015). Linkage was inferred using a logarithm of odds (LOD) threshold of 3.0, and repetitive use of the “rippling” command identified the best order of markers within the linkage group. Recombination fractions were converted into map distances in centimorgans using the Kosambi mapping function (Kosambi 1944).

QTL analysis. The composite interval mapping (Zeng 1994) method was undertaken for QTL mapping in each environment using the software windows QTL Cartographer 2.5. Model 6 of the Zmapqtl module of QTL Cartographer was used to identify QTLs associated with resistance to SCR with a scanning window size of 10 cM. Forward and backward stepwise regression was performed with 10 controlling markers for background control. The QTL cutoff was set as a LOD > 3.5 for an experiment-wise type I error rate ($\alpha = 0.05$) by 1,000 random permutations. For QTLs with additive effects, positive and negative signs of the estimates indicated that resistance effects were contributed by CIMBL83 or Lx9801, respectively. The proportion of the phenotypic variation explained by a single QTL was determined by the square of the partial correlation coefficient (R^2).

Development of near-isogenic lines (NILs) for QTL validation. Residual heterozygosity occurs widely in RILs. To develop NILs for fine mapping of *qSCR4.01*, NILs derived from a RIL (LMP2567) heterozygous for *qSCR4.01* were developed, and a small NIL F₂ population was planted at Ledong station in 2017. Three types of genotypes in the NIL F₂ population—namely, homozygous resistance allele (homozygote CIMBL83/CIMBL83), homozygous susceptible allele (homozygote Lx9801/Lx9801), and heterozygous allele (heterozygote CIMBL83/Lx9801)—across the target region were identified with markers. Least significant difference (LSD) was used to test

for significant differences of SCR resistance between NILs with *qSCR4.01* and those without *qSCR4.01*.

Fine mapping of *qSCR4.01*. Fine mapping of *qSCR4.01* was carried out by using BC₂F₁ segregating populations that were derived from BC₁F₁ plants harboring the *qSCR4.01* region. The identified recombinants from the BC₂F₁ population were backcrossed to the susceptible parental line, Lx9801, and their progenies were used for phenotypic analysis. The progenies derived from a given recombinant were divided into two categories based on their genotypes at the *qSCR4.01* region: heterozygous CIMBL83/Lx9801 and homozygous Lx9801/Lx9801. One-way ANOVA was used to determine the significant differences of SCR resistance between the two genotypes.

Results

Disease severity evaluations. The average performance and the descriptive statistics for SCR in the two parents and 117 RILs are given in Table 1. The parental line CIMBL83 was completely resistant to *P. polysora* across the three environments, whereas Lx9801 was highly susceptible to *P. polysora* with mean SCR score 7.4 across environments (Supplementary Fig. S1). Significant differences in SCR resistance among RILs were also observed. Broad sense heritability was 0.94 across the three environments, and the variance was mainly derived from differences among RILs, indicating that much of the phenotypic variance in the RIL population is genetically controlled.

Genetic linkage mapping and QTL mapping. Of the 9,433 SNPs used to genotype the RIL population, 3,786 SNPs showing polymorphism between CIMBL83 and Lx9801 were selected to construct the genetic map. The SNPs were distributed along all 10 chromosomes with no obvious gaps. In linkage analysis, some markers were located within the same block with the recombination frequency estimated as 0. In such cases, one marker in each block was randomly selected as the bin marker. This resulted in a genetic map with 963 bin markers spanning a total length of 1,414.7 cM, with an average distance of 1.48 cM between adjacent markers.

After the construction of the genetic map, SCR phenotypic data from three separate environments were used for QTL analysis (Table 2). Only one QTL, named *qSCR4.01* (located on chromosome 4, bin 4.01), was identified after 1,000 permutation tests in Changge in 2016 and 2017, which can explain 62 and 65% of the phenotypic variation, respectively. In Ledong in 2017, *qSCR4.01* was repeatedly detected with a LOD score of 21.3 and can account for 48% of the phenotypic variation. A second QTL, located on chromosome 9 (*qSCR9.03* in bin 9.03), was only detected in Ledong in 2017; however, this minor QTL with a LOD score of 3.6 can only explain 3% of the phenotypic variation. Both *qSCR4.01* and *qSCR9.03* were derived from the resistant parent, CIMBL83; however, only *qSCR4.01* is a major QTL identified in multiple locations over multiple years. Based on previously reported data from the literature, no other SCR resistance QTLs have been mapped to the same location as *qSCR4.01*, indicating it is likely to be a novel source of resistance.

Developing NILs to validate *qSCR4.01*. To develop NILs for validating *qSCR4.01*, we screened for heterogeneous inbred family from 117 BC₁RIL lines. We found one family, LMP2567, showing segregation for SCR resistance in 2017 in Changge (Fig. 1A, Supplementary Fig. S2). Of the six plants in the plot, two showed heterozygosity in the *qSCR4.01* region. These plants were self-crossed to

Table 1. Mean and range of southern corn rust (SCR) scores for parental lines (CIMBL83 and Lx9801) and the recombinant inbred line (RIL) population, together with variance components and heritability estimates for SCR responses across multiple environments^a

Environments	Lx9801	CIMBL83	RILs					H^2
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	Range	σ_g^2	σ_{ge}^2	σ_e^2	
Changge (2016)	7.1 $\pm$ 0.02	1.0 $\pm$ 0.00	4.83 $\pm$ 0.15	1.0–7.0				
Changge (2017)	7.5 $\pm$ 0.01	1.0 $\pm$ 0.00	4.95 $\pm$ 0.14	1.0–7.0				
Ledong (2017)	7.7 $\pm$ 0.02	1.0 $\pm$ 0.00	5.84 $\pm$ 0.20	1.0–8.5				
Combined	7.4 $\pm$ 0.02	1.0 $\pm$ 0.00	5.21 $\pm$ 0.16	1.0–7.17	2.76***	0.26	0.48	0.94

^a Three asterisks (***) indicate significance at $P = 0.001$.

produce a NIL F₂ population of 63 plants, which were genotyped by markers within the QTL region (Supplementary Table S1, Fig. 1B) and evaluated for SCR resistance at Ledong station in the winter of 2017. As shown in Figure 1B, the mean disease scores for plants homozygous for CIMBL83, heterozygous, and homozygous for Lx9801 within the QTL region were 1.25, 1.30, and 6.05, respectively. LSD tests showed that the difference in the SCR scores between NILs with and without *qSCR4.01* was significant, whereas the difference between plants that were homozygous for CIMBL83 and those that were heterozygous within the QTL region was not

significant. Taken together, *qSCR4.01* is a full dominant QTL and has a significant resistance effect against SCR in both NIL and RIL populations.

Fine mapping of *qSCR4.01*. Based on our preliminary results, *qSCR4.01* was mapped to an interval of ~2.54 Mb with the flanking markers AX86245743 and AX86301276 (Table 2). In the initial QTL region, two markers, PM01-000058W and PM01-00002MH, which are polymorphic between the parental lines, were used to screen the BC₂F₁ segregating population consisting of 456 plants. A total of six recombinants were identified. These recombinants were

Table 2. Quantitative trait loci (QTLs) for resistance to southern corn rust determined by composite interval mapping using the phenotypic data in three environments

QTL ^a	Flanking markers ^b	Marker interval ^c (Mb)	Change 2016			Change 2017			Ledong 2017		
			LOD	R ^{2d}	Add ^e	LOD	R ^{2d}	Add ^e	LOD	R ^{2d}	Add ^e
<i>qSCR4.01</i>	AX86245743–AX86301276	0.71–3.25	27.5	0.62	1.81	23.3	0.65	1.86	21.3	0.48	1.94
<i>qSCR9.03</i>	AX86320797–AX91792221	27.0–78.0							3.6	0.03	0.46

^a The QTL was named by *qSCR* plus the chromosome bin where it is located.

^b Markers in a QTL region that flank the peak of the logarithm of odds (LOD) scan.

^c Interval between the two markers on the B73 genome RefGen_v4 sequence.

^d Percentage of phenotypic variation explained by the QTL.

^e Positive value indicates the allele from parent CIMBL83 is to increase resistance.

Fig. 1. Validation of *qSCR4.01* using a near-isogenic line (NIL) population. **A**, *qSCR4.01* was detected on chromosome 4 in the CIMBL83/Lx9801 recombinant inbred line population. A heterogeneous inbred family line heterozygous in the quantitative trait locus region was chosen to self for developing NIL population. **B**, *qSCR4.01* was validated through the progeny test. For each type, the chromosomal composition at *qSCR4.01* is depicted as the solid bars with different colors/shades, corresponding to homozygote CIMBL83/CIMBL83, homozygote Lx9801/Lx9801, and heterozygote CIMBL83/Lx9801. *n* indicates the total number of progeny of a given type. LSD was used to test for the significant differences of disease score among genotypes, and superscripted letters show the difference ($P < 0.01$). Significant differences of SCR resistance were detected between lines with *qSCR4.01* (homozygote CIMBL83/CIMBL83 and heterozygote CIMBL83/Lx9801) and lines without *qSCR4.01* (homozygote Lx9801/Lx9801).

backcrossed to Lx9801 to generate six corresponding BC₃F₁ families. The sizes of the donor regions were estimated by genotyping with five markers, including three newly developed markers, PZE-104005694, SOURST-83_2035716, and SOURST-83_2465654 (Fig. 2). In the winter of 2018, a total of 115 BC₃F₁ individuals were planted in Ledong, Hainan. All BC₃F₁ plants were evaluated for resistance to SCR and genotyped in the donor region.

For a given BC₂F₁-derived BC₃F₁ family, there are two possible genotypes in the QTL region, CIMBL83/Lx9801 and Lx9801/Lx9801. Based on this, the six BC₃F₁ recombinants were classified into four types: types I, II, III, and IV. Type I carried the donor segments upstream of the marker, SOURST-83_2035716, whereas type II carried the donor segment downstream of PZE-104005694. A significant correlation between genotype and SCR resistance was observed in the BC₃F₁ progeny for each of these two types, indicating the presence of *qSCR4.01*. In contrast, a significant correlation between genotype and a susceptible SCR score was detected in the BC₃F₁ progeny for both types III and IV, indicating the absence of *qSCR4.01* (Fig. 2). Based on this analysis, the *qSCR4.01* region was delimited to an interval between the markers SOURST-83_2035716 and PZE-104005694 with a physical distance of ~770 kb based on the publicly available B73 physical map (AGPv4) (Fig. 2).

Discussion

Precise phenotypic evaluation is critical for QTL mapping. For the evaluation of resistance to SCR, both artificial inoculation and natural infection have been reported in the literature (Holland et al. 1998; Zhang et al. 2010). In the present study, natural infection was used to evaluate the SCR severity. Plants were grown in Ledong in Hainan Province and Change in Henan Province, two areas where SCR is prevalent. A high heritability of *qSCR4.01* was observed in RILs across multiple years and environments. Based on this, we conclude that natural infection is a reliable and economical method for SCR evaluation.

It is widely believed that flowering time (FT) may impact disease resistance, because late-flowering lines generally appear to be more resistant to disease (Wisser et al. 2006). In the present study, the RIL population was derived from the resistant line CIMBL83 and susceptible line Lx9801, which represent tropical and temperate

germplasm, respectively. To determine if *qSCR4.01* is associated with FT, the days from sowing to anthesis were noted while surveying SCR resistance in RILs during three field trials in 2016 and 2017. We found the correlation coefficient (*r*) was not significant between the phenotype of SCR severity and FT (Supplementary Table S3). We also analyzed the FT QTL in each of the three environments and found that there was no FT QTL colocalizing with *qSCR4.01* (data not shown). Therefore, it is unlikely that the *qSCR4.01* from CIMBL83 is a false positive caused by late maturity.

Many QTLs or genes against *P. polysora* have been identified in diverse maize germplasm (reviewed by Brewbaker et al. [2011] and Wanlayaporn et al. [2013]). We compared the QTLs in the present study (*qSCR4.01* and *qSCR9.03*) with those published in the literature based on bin position in the maize genome. We found that *qSCR9.03* may colocalize with a QTL identified from an F₂ population derived from a cross between Z-95 and Z-93 (Brunelli et al. 2002). On chromosome 4, QTLs for resistance to *P. polysora* were detected in bin 4.02 (Jiang et al. 1999), bin 4.03 (Holland et al. 1998), and bin 4.08 (Deng et al. 2019), and also one QTL in bin 4.05 (Lopez-Zuniga et al. 2019; Martins et al. 2019) conferring multiple disease resistance in the maize genome. However, *qSCR4.01* located in bin 4.01 did not overlap with any of these QTLs, indicating that *qSCR4.01* may be a novel source of resistance to SCR. This not only has important implications for maize breeding programs but also opens the door for further research into the genetic mechanism of SCR resistance. Interestingly, a QTL for resistance to Stewart's wilt has been mapped to the same general region as *qSCR4.01*, implying that chromosome 4, bin 4.01 may be important for multiple diseases (Brown et al. 2001).

To look for candidate genes in the *qSCR4.01* region, we first analyzed the ~770 kb interval in the B73 genome (AGPv4) and found a poor assembly quality for about 500 kb of Ns (i.e., the specific sequences have not been determined). According to the analysis of the remaining B73 sequence between the flanking markers, 11 genes are predicted in the *qSCR4.01* region, of which five genes encode putative NBS-LRR proteins and one gene is annotated as wall-associated receptor kinase (Supplementary Table S2). Because the genome size varies between tropical and temperate germplasm in maize (Tenaillon et al. 2016), we further analyzed the *qSCR4.01*

Fig. 2. *qSCR4.01* was fine mapped to a ~770-kb interval. The recombinants were classified into four types (I to IV). For each type, the chromosomal composition at *qSCR4.01* is depicted as the solid bars with gray (dark gray in print) or yellow (light gray in print), corresponding to homozygote Lx9801/Lx9801 or heterozygote CIMBL83/Lx9801, respectively. The total number of plants refers to all progeny of a given type. The disease score was calculated for each genotype of the Lx9801/Lx9801 and CIMBL83/Lx9801, respectively. ANOVA was used to test for the significance of disease score between genotypes. A significant difference ($P < 0.01$) between homozygous and heterozygous genotypes indicated the presence of *qSCR4.01* in the donor region, and the corresponding phenotype was deduced to be resistant (R). The lack of a significant difference ($P > 0.01$) between the two genotypes suggested the absence of *qSCR4.01* in the donor region, and the corresponding phenotype was deduced to be susceptible (S). Based on analysis of the deduced phenotype and the donor region for all recombinants, the map of *qSCR4.01* was refined to a ~770 kb region with flanking markers PZE-104005694 and SOURST-83_2035716.

region (~200 kb) in a tropical landrace (i.e., SK) genome, which was assembled more recently (Yang et al. 2019). The 200-kb region of the SK genome harbors 10 genes, in which two genes encode putative NBS-LRR proteins. However, we evaluated B73 and SK for their resistance to SCR in Ledong and Changde and found both of them were highly susceptible, which meant the *R* genes identified from the B73 and SK genome might not be the candidate genes for *qSCR4.01*. Considering the overall complexity of the maize genome, de novo assembly of the CIMBL83 genome is still needed in order to identify the resistance gene underlying *qSCR4.01*.

The short arm of chromosome 10 (bin 10.00/01) is a region rich in disease resistance genes against SCR in maize. Although multiple major QTLs/genes have been identified in this region (Holland et al. 1998; Jines et al. 2007; Wu et al. 2015; Zhang et al. 2010; Zhao et al. 2013; Zhou et al. 2007), the effects of these resistance genes have been gradually lessened or lost over time due to wide deployment in maize breeding programs. This is exemplified by *Rpp9*, which provided effective resistance to SCR in the northern United States for over 20 years, has been overcome by a new race of *P. polysora* (Brewbaker et al. 2011; Dolezal et al. 2009). Another example is *RppQ* from Qi319, widely used in Chinese maize breeding programs, which has recently become ineffective (Wang et al. 2019). Because of this, the continuous discovery of new genes is required to protect maize from SCR and minimize yield losses. In the present study, we identify a major QTL on chromosome 4, *qSCR4.01*, which is stable across multiple environments. To our knowledge, this is the first major SCR resistance QTL mapped to chromosome 4 and, therefore, is an important source for the future of SCR resistance breeding programs in maize.

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