QTL mapping for resistance to southern leaf blight in sweet corn

Liu Pengfei¹,², Jiang Feng², Zhang Jinfeng², Zhang Zili², Wang Hanning¹ and Wang Xiaoming¹,²*

¹College of Agronomy, Gansu Agricultural University, Lanzhou 730070, China.
²College of Agronomy, Zhongkai University of Agriculture and Engineering, Guangzhou 510225, China.

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To identify quantitative trait loci (QTL) associated with southern leaf blight (SLB) in sweet corn, a population consisting of 330 F²3 families was developed from a T14 (resistant) × T4 (susceptible) cross. The genetic linkage map was constructed based on the F² population, with 192 SSR markers covering a total map distance of 1260 cm, and an average interval length of 6.56 cm. These 330 F²3 families were evaluated in 2009 and 2010 for SLB resistance following inoculation at the seedling stage and milk stage. Using composite interval mapping (CIM) in QTL Cartographer v.2.5, 7 QTL out of 18 total QTL were identified at the seedling stage on chromosomes 3, 4, 6 and 9, and the phenotypic variance explained by each QTL ranged from 4.02 to 35.4%. The 11 other QTL were identified at the milk stage on chromosomes 3, 4, 6, 8, 9 and 10 and accounted for 3.50 to 13.65% of the phenotypic variance. Two QTL were detected in both years; these were located on chromosome 3 and 4 in the marker intervals umc2275 to umc2008 and umc2287 to bnlg2162, respectively. One QTL, located on chromosome 3 between umc2275 and umc2008, was detected at both developmental stages. The findings in this study may provide useful information for marker-assisted selection (MAS) and with further genetic and breeding studies for maize SLB.

Key words: Sweet corn, southern leaf blight (SLB), composite interval mapping (CIM), quantitative trait loci (QTL).

INTRODUCTION

Southern leaf blight (SLB) is a worldwide maize (Zea mays L.) fungal disease caused by the imperfect fungi Bipolaris maydis (anamorph) (Wang et al., 2001). Three physiological races O, T, and C were found to be harmful in maize (Hooker et al., 1970, 1972). SLB occurs throughout the growing period, and infection peaks after tasseling. Susceptible varieties generally show a 10% yield reduction when infected, and yield losses of up to 30% are seen in years with greater infection rates (Wang et al., 2001). In 1970, yield losses caused by SLB in the United States cost about 10 billion dollars and attracted the attention of maize breeders to this disease (Hooker, 1972). Developing SLB tolerant varieties with a high yield potential is the most economic, effective and safe means of limiting SLB infection.

Smith et al. (1973) identified a race O-specific recessive resistance gene (rhm) in South African maize and transferred rhm to a set of maize inbred lines. The rhm gene was located on the short arm of maize chromosome 6 near the region with the rgl1 gene and the AFLP marker umc85 (Zaitlin et al., 1993). Cai et al. (2003) detected a co-dominant AFLP marker (p7m36) 1.0cM away from the rhm gene in near-isogenic lines by the bulk segregant analysis method. The genetic distance from rhm to the RFLP marker agrp144 was 0.5 cm (Holley et al., 1989).

Quantitative trait locus (QTL) mapping can provide useful information for breeding programs, since it allows the estimation of genomic locations and genetic effects of chromosomal regions related to the expression of quantitative traits. To date, studies have been published on mapping quantitative trait loci for field resistance to...
SLB in maize from populations derived from different crosses (Balint-Kurti et al., 2006, 2008; Bubeck et al., 1991; Carson et al., 2004; Jiang et al., 1999), but different parental but different parental lines, segregation population or genetic map can bring about different results in QTL number, position or effect, and thus, it is necessary and significant that different parents be selected to detect the QTL for maize SLB. Moreover, previous studies were focused on one of the developmental stages. Further more, ecological conditions can affect gene expression, and same gene will probably present expression variation under different environments and different time.

In this study, we constructed a population from a T14 (resistant) × T4 (susceptible) cross and graded disease severity of inoculated individuals in F2:3 families. A genetic linkage map based on the F2 population was constructed with SSR markers, and quantitative trait loci (QTL) associated with SLB was identified by composite interval mapping. The objectives were to (1) identify and evaluate the QTL for SLB resistance from sweet corn, and (2) screen steady QTL in different developmental stages and environments. QTL found in this study may provide useful information for marker-assisted selection (MAS) and may further genetic and breeding studies on maize SLB.

MATERIALS AND METHODS

Material and assay

The resistant line T14 and the susceptible line T4 are sweet corn inbred lines that were developed by the maize research group at the College of Agronomy, Zhongkai University of Agriculture and Engineering. The difference in resistance to SLB between these two lines is significant and stable. In the spring of 2008, an F2 mapping population was developed from the cross between T14 (female) and T4 (male) at the Zhongcun teaching farm at the Zhongkai University of Agriculture and Engineering. The resultant 330 F2 individuals were selfed in the spring of 2009. Ten individuals from each family for all 330 F2:3 families were planted in the field and in the greenhouse and were used to evaluate resistance at the seedling stage and in mature plants in the autumn of 2009 and the spring of 2010.

Inoculation and evaluation of SLB resistance

In this experiment, artificial inoculations were used to evaluate SLB resistance in maize. The Bipolaris maydis (Nisikado and Miyake) Shoemaker were grown on potato dextrose agar for 7 to 10 days at 25°C then added the hyphae suspension to flasks containing sorghum grain culture. (Sorghum grain culture was prepared as follows: sorghum grains were soaked overnight, boiled for 30 to 40 min, put into flasks, sterilized at 121°C for 1 h, then allowed to cool). After dark culture for 5 to 7 days at 25°C, the hyphae-covered sorghum grains were plated onto clean plates while maintaining a constant humidity level. After significant sporulation, a spore suspension with a concentration between 1.0 × 108 and 1.0 × 109 spores/ml was prepared and sprayed on corn leaves by a knapsack sprayer. Three hundred and thirty F2:3 families were planted in the fall of 2009 and inoculated with the O race at the 6 to 7 leaf seedling stage and at the adult stage prior to tasseling (Chen et al., 2004; Wang et al., 2001). Disease severity was measured 7 d after inoculation, and disease rating were used to represent the level of SLB resistance. Disease severity was determined according to the national standard. Disease severity were rated on a 0 to 5 scale, with 0 being symptomless plant and 5 being a completely dead and each increasing number representing an 25% increase in disease severity (Wang et al., 2001). The disease rating of each individual in each F2:3 families were measured, and the average within each F2:3 families were calculated to represent the resistance of the corresponding F2.

DNA extraction and SSR analysis

Genomic DNA was isolated from fresh leaf tissue of the parental lines and the F2 individuals following the CTAB method used by Paterson (1993). The F2 individuals were genotyped using 192 SSR markers. All SSR marker sequences were obtained from the maize GDB database (http://www.maizegdb.org/ssr.php) (Wang et al., 2007; Yu et al., 2007; Liu et al., 2008; Li et al., 2009; Zhao et al., 2006). The PCR, gel electrophoresis and silver stain procedures used in this study followed the referenced methods (Wei, 2004; Guo et al., 2007).

Linkage map construction and QTL mapping

Linkage maps were constructed using JoinMap 3.0 (Van et al., 2001). QTL were identified by composite interval mapping using Windows QTL Cartographer 2.5 (Zeng, 1994; Wang et al., 2007). A stringent LOD threshold, calculated by a permutation test (1,000 permutations), was used to determine QTL significance in this study. The genetic effect and contribution rate for each QTL was estimated. The ANOVA of the phenotypic data were calculated using SAS V8.02 (SAS Institute Inc. Cary, North Carolina, 1991 to 2001). QTL nomenclature followed the method of McCouch et al. (1997) and corresponds to “QTL” + “traits” + “chromosome” + “number of QTL”. QTL were designated with a lower case “q”. Traits were represented by English abbreviations, and the QTL level of the resistance to SLB was represented as “DR”. When several QTL were present on the same chromosome, they were numbered in sequential order following the chromosome number.

RESULTS

Phenotypic variation

The disease rating distribution across the F2:3 families are described in Table 1. Based on the skewness and kurtosis, the distribution of disease ratings fit a normal distribution with large variance, demonstrating the characteristics of a quantitative trait. The disease rating trait can be used for QTL mapping.

Construction of linkage map

A linkage map was constructed from 192 SSR markers showing co-dominant segregation (Figure 1). The linkage map has a total length of 1260 cm with an average between marker intervals of 6.5 cm.
Table 1. Normal distribution test of DR in \( F_{2:3} \) population at two stages and for two years.

<table>
<thead>
<tr>
<th>Date</th>
<th>Mean</th>
<th>Range</th>
<th>SD</th>
<th>CV (%)</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>DR at seedling stage in 2009</td>
<td>2.487</td>
<td>0.930-4.183</td>
<td>0.902</td>
<td>36.264</td>
<td>-0.921</td>
<td>0.0236</td>
</tr>
<tr>
<td>DR at milk stage in 2009</td>
<td>2.620</td>
<td>0.994-4.292</td>
<td>0.936</td>
<td>35.749</td>
<td>-0.944</td>
<td>-0.0499</td>
</tr>
<tr>
<td>DR at seedling stage in 2010</td>
<td>2.713</td>
<td>1.036-4.424</td>
<td>0.969</td>
<td>35.718</td>
<td>-0.967</td>
<td>-0.0464</td>
</tr>
<tr>
<td>DR at milk stage in 2010</td>
<td>2.453</td>
<td>0.955-3.935</td>
<td>0.874</td>
<td>35.624</td>
<td>-0.977</td>
<td>-0.0494</td>
</tr>
</tbody>
</table>

Figure 1. SSR genetic linkage map and QTL mapping for resistance to SLB. Note: Bars and whiskers indicate 1-LOD and 2-LOD QTL likelihood intervals, respectively.
Table 2. QTLs for resistance to SCLB detected at two stages in 2009.

<table>
<thead>
<tr>
<th>Stage</th>
<th>QTL</th>
<th>Chromosome</th>
<th>Location</th>
<th>Flanking marker</th>
<th>Additive</th>
<th>Dominance</th>
<th>LOD(threshold)</th>
<th>R² (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.4-1</td>
<td>Chr.4</td>
<td>78.80</td>
<td>umc2287-bnlg2162</td>
<td>-0.619</td>
<td>-0.608</td>
<td>10.70(2.87)</td>
<td>20.1</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.6-1</td>
<td>Chr.6</td>
<td>27.46</td>
<td>umc1753-bnlg426</td>
<td>-0.363</td>
<td>-0.357</td>
<td>4.31(2.87)</td>
<td>7.55</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.6-2</td>
<td>Chr.6</td>
<td>44.80</td>
<td>umc1133-bnlg2097</td>
<td>-0.648</td>
<td>-0.636</td>
<td>11.46(2.87)</td>
<td>23.9</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.3-1</td>
<td>Chr.3</td>
<td>107.83</td>
<td>umc2275-umc2008</td>
<td>0.276</td>
<td>0.285</td>
<td>2.63(2.56)</td>
<td>4.02</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.4-2</td>
<td>Chr.4</td>
<td>92.68</td>
<td>bnlg2162-umc1051</td>
<td>-0.839</td>
<td>-0.869</td>
<td>19.74(2.56)</td>
<td>35.4</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.4-3</td>
<td>Chr.4</td>
<td>106.06</td>
<td>umc1856-umc2286</td>
<td>-0.352</td>
<td>-0.365</td>
<td>3.55(2.56)</td>
<td>6.50</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.9-1</td>
<td>Chr.9</td>
<td>79.13</td>
<td>umc1571-umc1170</td>
<td>-0.456</td>
<td>-0.472</td>
<td>8.71(2.56)</td>
<td>10.6</td>
</tr>
</tbody>
</table>

**QTL for SLB**

Eighteen putative QTL associated with SLB at the seedling and adult stage was detected in 2009 and 2010 using composite interval mapping. These QTL, which were detected on chromosomes 3, 4, 6, 8, 9 and 10, explained between 3.50 and 35.4% of the total phenotypic variation. Nine QTL associated with SLB were clustered on chromosome 4 and explained most of the total phenotypic variations, these QTL explained a combined 62.0% phenotypic variations in 2009 and 38.35% in 2010 (Tables 2 and 3).

**QTL mapping of SLB resistance in 2009**

Three QTL for SLB resistance at the seedling stage were detected on chromosome 4 and 6 (Table 2, Figure 1). The first QTL, *qDR-ch.4-1*, was found between markers umc2287 and bnlg2162 on chromosome 4 and explained 20.1% of the variation. The other two QTL, *qDR-ch.6-1* and *qDR-ch.6-2*, were found on chromosome 6 in the marker intervals umc1753-bnlg426 and umc1133-bnlg2097 and explained 7.55 and 23.9% of the phenotypic variance.

Four SLB resistance QTL identified at the milk stage were detected on chromosomes 3, 4 and 9 (Table 2, Figure 1). The first QTL, *qDR-ch.3-1*, was found in the marker interval umc2275 to umc2008 on chromosome 3 and accounted for 4.02% of the phenotypic variance. There were two QTL, *qDR-ch.4-2* and *qDR-ch.4-3*, detected on chromosome 4 within the marker intervals bnlg2162-umc1051 and umc1856-umc2286 that accounted for 35.4 and 6.50% of the phenotypic variance. The other QTL, *qDR-ch.9-1*, was found between umc1571 and umc1170 on chromosome 9 and controlled 10.6% of the variation.

Two major QTL, *qDR-ch.4-1* and *qDR-ch.4-2*, were detected on chromosome 4 and explained 20.1% and 35.4% of the total phenotypic variation seen at the seedling and at the milk stage, respectively. These two QTL were found adjacent to each other in the umc2287-umc1051 interval. Six QTL alleles came from the resistant parent (T14) with a negative additive effect.

The QTL *qDR-ch.3-1* was the exception, and this resistance QTL allele was derived from the susceptible parent T4. Overall, most resistance alleles came from the resistant parent.

**QTL mapping of SLB resistance in 2010**

Six QTL for SLB resistance at the seedling stage were detected on chromosomes 3, 4, 6 and 8 (Table 3, Figure 1). The first QTL, *qDR-ch.3-2*, was located between umc2275 and umc2208 on chromosome 3 and controlled 11.12% of the variation. The other two QTL, *qDR-ch.4-4* and *qDR-ch.4-5*, were found on chromosome 4 in the marker intervals umc2148-umc1757 and umc2287-bnlg2162 and explained 5.31 and 6.50% of the phenotypic variance. One QTL, *qDR-ch.6-3*, was found in the marker interval umc1723-umc2318 on chromosome 6 and accounted for 3.50% of phenotypic variance. The other two QTL, *qDR-ch.8-1* and *qDR-ch.8-2*, were detected on chromosome 8 in the marker intervals phi119-bnlg1056 and bnlg1056-umc1304 and explained 9.45 and 8.83% of the phenotypic variance.

Five QTL for SLB resistance at the milk stage were detected on chromosomes 4, 9 and 10 (Table 3, Figure 1). The first three QTL, *qDR-ch.4-6*, *qDR-ch.4-7* and *qDR-ch.4-8*, were found on chromosome 4 in the marker intervals nc005-umc1294, bnlg1189-umc2009 and umc2009-umc1808, respectively, and explained 6.88, 13.65 and 6.01% of the phenotypic variance, respectively. The other two QTL, *qDR-ch.9-2* and *qDR-ch.10-1*, were detected on chromosome 9 and 10 in the marker intervals umc1170-bnlg1714 and umc1432-umc1785, respectively, and accounted for 4.50% and 3.78% of the phenotypic variance.

Two major QTL, *qDR-ch.3-2* and *qDR-ch.4-7*, were identified on chromosome 3 and 4, accounted for 11.12 and 13.65% of the phenotypic variance at the seedling and milk stage, respectively. Two QTL, *qDR-ch.8-1* and *qDR-ch.8-2*, were found near the marker bnlg1056 on chromosome 8. Two QTL, *qDR-ch.4-7* and *qDR-ch.4-8*, were associated with the marker umc2009 located near the interval of bnlg1189-umc1808. All QTL came from the...
resistant parent with a positive additive effect. The common QTL were detected in two years and two stages Two QTL, which were located on chromosome 3 and 4, were detected in both years. One QTL located between umc2275-umc2008 on chromosome 3, was detected at both developmental stages. Two stable SLB resistance QTL were detected in 2009 and 2010. qDR-ch.4-1 and qDR-ch.4-5 both mapped to the region umc2287-bnlg2162 in 2009 and 2010 suggesting that these two QTL might be the same stable QTL. qDR-ch.3-1 and qDR-ch.3-2 were both detected in marker interval umc2275-umc2008 at milk stage in 2009 and seedling stage in 2010.

During the experimental study period, a resistance QTL was detected both at the seedling and at the milk stage. qDR-ch.3-1, a QTL from the seedling stage, and qDR-ch.3-2, a QTL from the milk stage, both mapped to the marker region between umc2275 and umc2208, suggesting the presence of a single stable resistance QTL.

### DISCUSSION

SLB is frequently found in hot, humid maize-growing areas worldwide. This study was to identify loci contributing to SLB resistance at two developmental stages in two years. One QTL were detected existing in both seedling stage and milk stage in two years, this suggested the QTL might be the stable QTL. It could be used as candidate gene of quantitative trait gene. This result was beneficial for realizing the genetic basis of SLB resistance and developing marker-assisted selection in maize breeding project.

Although many studies on QTL identification for SLB resistance were reported in maize (Balint-Kurti et al., 2006, 2008; Bubeck et al., 1991; Carson et al., 2004; Jiang et al., 1999). Compared to previous studies, our result differed greatly from the previous, including QTL number, position and genetic effects, few of the QTL identified in this study co-localize with QTL found in Balint-Kurti et al. (2006-2008) and Carson et al. (2004); which could result from different parental lines, environmental design, genetic maps, markers used and statistical methods or developmental stages and ecological conditions. Moreover, it is worthy of note that, some QTL reported by different researches were located on same chromosomes, but their chromosomal bin loci were different from each other. For example, the QTL on chromosome 3 identified by Balint-Kurti et al. (2008) was within bin 3.04 and were detected within bin 3.08 and 3.09 in this study.

Furthermore, in this study, there were similar findings in several respects to the previous study. We detected QTL near the 3.08-3.09 and 4.03-4.04 region; QTL have previously been detected in these regions by Balint-Kurti et al. (2006, 2008). The QTL qDR-ch.9-1 identified in this study at 9.02 to 9.04 was adjacent to a QTL region (9.03 to 9.04) detected by Balint-Kurti et al. (2006-2008). In this study, three QTL (qDR-ch.4-1, qDR-ch.4-2 and qDR-ch.4-5) clustered together at region 4.08 and the identification of this region as an SLB resistance QTL location is consistent with previous studies (Balint-Kurti et al., 2006). This suggests that these QTL regions might contain the same resistance genes or related members in a disease resistance gene family. Quantitative trait loci (QTL) for SLB resistance were identified in bins 6.01 (two QTL) is consistent with previous studies (Balint-Kurti et al., 2008). A comparison of the results of all published SLB resistance QTL suggested that bins 6.01 are ‘hotspots’ for SLB resistance QTL (Thompson et al., 1984; Zaitlin et al., 1993; Chang et al., 1995; Balint-Kurti et al., 2008).

Tanksley (1993) reported that QTL that explain more than 10% of the phenotypic variation are major QTL. In this two year study, six major SLB resistance QTL that explained more than 10% of the phenotypic variation and 12 minor QTL were detected either in seedlings or in mature plants. These results are consistent with the high stable resistance of T14 identified in the field over many years (2005 to 2008). The three major QTL (qDR-ch.4-2,

### Table 3. QTLs for resistance to SCLB detected at two stages in 2010.

<table>
<thead>
<tr>
<th>Stage</th>
<th>QTL</th>
<th>Chromosome</th>
<th>Location</th>
<th>Flanking Marker</th>
<th>Additive</th>
<th>Dominance</th>
<th>LOD(threshold)</th>
<th>R² (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.3-2</td>
<td>Chr.3</td>
<td>109.83</td>
<td>umc2275-umc2008</td>
<td>-0.471</td>
<td>0.148</td>
<td>5.71(2.61)</td>
<td>11.12</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.4-4</td>
<td>Chr.4</td>
<td>0.100</td>
<td>umc2148-umc1757</td>
<td>-0.293</td>
<td>0.198</td>
<td>4.32(2.61)</td>
<td>5.31</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.4-5</td>
<td>Chr.4</td>
<td>82.81</td>
<td>umc2287-bnlg2162</td>
<td>-0.354</td>
<td>0.150</td>
<td>2.91(2.61)</td>
<td>6.50</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.6-3</td>
<td>Chr.6</td>
<td>75.00</td>
<td>umc1723-umc2318</td>
<td>-0.010</td>
<td>0.365</td>
<td>2.85(2.61)</td>
<td>3.50</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.8-1</td>
<td>Chr.8</td>
<td>18.16</td>
<td>phi119-bnlg1056</td>
<td>-0.462</td>
<td>0.097</td>
<td>6.41(2.61)</td>
<td>9.45</td>
</tr>
<tr>
<td>Seedling stage</td>
<td>qDR-ch.8-2</td>
<td>Chr.8</td>
<td>25.80</td>
<td>bnlg1056-umc1304</td>
<td>-0.440</td>
<td>0.150</td>
<td>6.14(2.61)</td>
<td>8.83</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.3-6</td>
<td>Chr.4</td>
<td>42.76</td>
<td>nc005-umc1294</td>
<td>-0.358</td>
<td>0.037</td>
<td>3.97(2.64)</td>
<td>6.88</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.4-7</td>
<td>Chr.4</td>
<td>56.79</td>
<td>bnlg1189-umc2009</td>
<td>-0.494</td>
<td>0.077</td>
<td>7.72(2.64)</td>
<td>13.65</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.4-8</td>
<td>Chr.4</td>
<td>63.94</td>
<td>umc2009-umc1808</td>
<td>-0.329</td>
<td>0.039</td>
<td>2.94(2.64)</td>
<td>6.01</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.9-2</td>
<td>Chr.9</td>
<td>92.88</td>
<td>umc1170-bnlg1714</td>
<td>-0.242</td>
<td>0.165</td>
<td>3.89(2.64)</td>
<td>4.50</td>
</tr>
<tr>
<td>Milk stage</td>
<td>qDR-ch.10-1</td>
<td>Chr.10</td>
<td>23.26</td>
<td>umc1432-umc1785</td>
<td>-0.223</td>
<td>-0.116</td>
<td>3.29(2.64)</td>
<td>3.78</td>
</tr>
</tbody>
</table>
qDR-ch.9.1 and qDR-ch.4-7) detected within 4 cm of adjacent markers can provide a basis for molecular marker-assisted selection, isolation and cloning of resistance genes and breeding of resistance lines.

Different QTL can be detected under different circumstances because of interactions between the genotype and the environmental conditions. QTL consistently identified under different environmental conditions are ideal for molecular marker-assisted selection. In this study, QTL located between the umc2275 to umc2008 interval on chromosome 3 and between the umc2287 to bnlg2162 interval on chromosome 4 can be used in QTL-assisted selection. The other QTL were not detected in both years, and this might be due to genotype x environment interactions. QTL on chromosome 4 explained about 62% and 38.35% of the total phenotype variation in the first and second year of cultivation, respectively.

Genetic studies have shown that the strict temporal scheduling of gene expression exists in both prokaryotes and eukaryotes and is controlled by an in vivo gene regulatory system. Different genes have different regulatory systems, particularly in complex higher organisms (Liu et al., 2008). By studying the dynamic changes in plant height, Yan et al. (2003) indicated that the QTL have the same temporal and spatial expression. Wu (1987) also showed that plant height, grain weight, leaf number and other traits were controlled by different genes at different developmental stages. In this study, QTL associated with SLB resistance at the seedling stage were quite different from the QTL identified at plant milk. Among the 18 resistance QTL, only one was detected at both the seeding and adult stage. This QTL, which was located between umc2275 to umc2008 on chromosome 3, will be a focus of future studies including maize breeding efforts for disease resistance to SLB. The remaining 17 QTL were only expressed at either the seeding stage or at milk stage, which indicated that resistance to SLB was related to the developmental stage. Most of the resistance QTL was only detected at a specific growth stage, which suggests that the dynamic expression of traits at different developmental stages is the result of ordered selective gene expression.

In this study, two SLB resistance QTL were detected at the seedling stage in 2009 and 2010, and both were located between the umc2287 and bnlg2162 markers on chromosome 4. Whether they are the same or two different QTL, it remains to be determined on a more saturated linkage map. This region should be the focus of further study for multi-point investigations for many years.

Many studies have shown that QTL are not randomly distributed on chromosomes but clustered together (Mcmullen et al., 1995; Saghai-Maroo et al., 1996). In this study, QTL for resistance SLB clustered together on chromosome 4. qDR-ch.4-1 and qDR-ch.4-5 were in the same region, and the linkage distance of qDR-ch.4-7 and qDR-ch.4-8 was very small, both near the marker umc2009. This chromosome will be a focus of SLB resistance QTL mapping in further studies. Therefore, these results may provide the useful information for marker-assisted selection (MAS) and further genetic and breeding studies on maize SLB.

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