Genetic Components of Resistance to Stalk Tunneling by the European Corn Borer in Maize

M. D. Krakowsky, M. J. Brinkman, W. L. Woodman-Clikeman, and M. Lee*

ABSTRACT

Identification of the genes conferring resistance to European corn borer (ECB) (Ostrinia nubilalis (Hübner)) is an important step in understanding how resistance is expressed and whether different sources of maize (Zea mays L.) germplasm can be combined to enhance protection. The locations of genes for resistance to ECB tunneling have been reported but are inconsistent across studies. The objectives of this study were to map and characterize quantitative trait loci (QTL) for resistance to tunneling in De811 and compare these with related studies and with QTL for anthesis and ear height. Inbred De811 (resistant) was crossed to susceptible inbred B73 to produce a population of 147 F1 lines. The population was artificially infested and evaluated in three environments. The F1 lines were genotyped at 88 restriction fragment length polymorphism (RFLP) loci to facilitate QTL mapping with composite interval mapping (CIM). Seven QTL for ECB tunneling were detected on chromosomes 1, 3, 4, 5, and 8, associated with 42% of the phenotypic variation. The F1 exhibits partial dominance for resistance but only one QTL with dominant gene action was observed. An F1 population of B73 × B52 that was evaluated in the same environments facilitated comparisons of genetic heterogeneity between inbreds De811 and B52. Only one QTL for tunneling was common between the populations, indicating that the two inbreds may contribute different genes for resistance in crosses with B73. This information could be useful for combining the favorable alleles of De811 and B52.

E CB IS A MAJOR PEST of temperate maize, with yield losses and control measures exceeding $1000 million annually (Mason et al., 1996). In temperate zones, the larvae of two or more sexual generations feed on leaf, sheath, and collar tissues and pollen and tunnel into the stalk and ear shank (Pesho et al., 1965; Guthrie et al., 1970; Mason et al., 1996). The stalk tunneling reduces grain yield (Pesho et al., 1965; Klenke et al., 1986b; Mason et al., 1996) by interfering with physiological processes, physically weakening the stalk and ear shoot (Lynch, 1980; Klenke et al., 1986b) and by providing points of entry for pathogens associated with stalk rot (Mason et al., 1996).

Adapted inbred lines with elevated levels of resistance to stalk tunneling by ECB have been identified (e.g., B52, De811, Mo47; Russell et al., 1971; Hawk, 1985; Barry et al., 1995). Knowledge of the inheritance and the genetic basis of resistance to tunneling could facilitate development of germplasm with enhanced levels of resistance and desired agronomic traits. High heritabilities (0.63–0.78) for resistance to tunneling have been reported (Schön et al., 1993; Jampatong, 1999; Cardinal et al., 2001). Identification of genetic components of resistance to tunneling has been hindered by environmental variation, a laborious and lengthy screening process, and the polygenic nature of the trait; however, linkage analysis has provided estimates of gene locations for inbred lines B52 and Mo47 (Onukogu et al., 1978; Schön et al., 1993; Jampatong, 1999; Cardinal et al., 2001).

The inbred line De811 is resistant to ECB stalk tunneling (Hawk, 1985) and shows partial dominance for resistance in the F1 of crosses to susceptible inbreds (e.g., A619, B73, C131A; Guthrie et al., 1989). The effects and positions of genes for resistance to tunneling in De811 have not been previously reported. Such information could be useful for breeding with De811 and other germplasm. In this study, 147 F1 lines of B73 × De811 were genotyped at RFLP loci and evaluated for tunneling and two other traits that could potentially confound assessment of tunneling: anthesis and ear height (Dicke, 1954; Coors, 1987). This population and F1 lines of B73 × B52 (Schön et al., 1993) were grown in the same environments. The common environments and susceptible parent provide an opportunity to assess genetic heterogeneity for resistance. The objectives of this study were (i) to assess genetic and environmental components of resistance to ECB tunneling in the F1 generation of B73 × De811; (ii) to determine the genotypic correlations between ECB tunneling and ear height and anthesis; (iii) to map genetic factors for resistance, anthesis, and ear height; and (iv) to evaluate the relative importance of additive and dominance gene effects on resistance.

MATERIALS AND METHODS

Plant Materials

Random F1 plants from a cross between inbred lines B73 and De811 were self-pollinated to produce 150 F2 lines. Inbred B73 is widely used in temperate maize breeding programs but is highly susceptible to stalk tunneling by ECB (Table 1). Inbred De811 and the F1 (B73 × De811) exhibit high levels of resistance to ECB tunneling in the stalk (Table 1).

Field Experiments

The experiments were planted at two locations: the Agronomy and Agricultural Engineering Research Center (AAÆRC) near Ames, IA, and the Iowa State University Research Farm near Ankeny, IA, on 11 May and 25 April, respectively, in

Abbreviations: AIC, Aikake information criteria; CI, confidence interval; CIM, composite interval mapping; cM, centimorgan; ECB, European corn borer; GDD, growing-degree days; LOD, log of the odds ratio; QTL, quantitative trait loci; RFLP, restriction fragment length polymorphism; SCB, sugarcane borer; SWCB, Southwestern corn borer.
1989, and at the AAERC on 29 May 1990. Each location–year was considered a separate environment. Soil fertilization, weed control, and cultivation practices were consistent with optimum maize production for this region. The entries in each experiment consisted of the 150 F3 lines and two entries each of B73, De811, and the F1. Entries were evaluated in hill plots consisting of two hills spaced on centers of 0.76 m at Ames and 1.02 m at Ankeny, and were arranged in a 12 × 13 simple lattice design with two replications per environment. Plots were overplanted and thinned to three plants per hill (i.e., six plants per plot; Guthrie et al., 1985).

### Trait Evaluation

All plants in each plot were artificially infested with ECB larvae when 50% of the entries in the experiment had reached anthesis. Anthesis was defined as three of the six plants in a plot shedding pollen. Newly hatched larvae were obtained from the USDA Corn Insect Laboratory, Ames, IA. The larvae were applied at four infestation points: the primary leaf axil, the first and second leaf axils above the primary ear, and stalk tunneling. Stalk tunneling was recorded to the nearest centimeter from the soil level to the highest ear-bearing node. GDD were calculated from planting to anthesis, according to the formula [(max. °C + min. °C)/2] – 10°C, where 10°C was used for the minimum temperature and 30°C was used for the maximum temperature if the actual temperatures exceeded those limits (Cross and Zuber, 1972). Ear height was measured on all plants in the plot as the distance (to the nearest 5 cm) from the soil level to the highest ear-bearing node. Anthesis was only measured at the Ames environments, while ear height was measured at all environments.

### Analysis of Phenotypic Data

For each trait and entry, least square means (lsmeans) were calculated with complete and incomplete blocks as random effects and entries as fixed effects for each environment (Cardinal et al., 2001). Environments were also treated as random effects when calculating lsmeans for the mean environment. These means were used for the QTL analysis. Means of the two parental lines and the F1 were calculated as the average of the lsmeans of the two entries in each environment. Genotype × environment, and error variance were calculated with environments, complete and incomplete blocks, and entries and the entry × environment interaction as random effects (Cardinal et al., 2001). Broad-sense heritabilities on an entry-mean basis and their exact confidence intervals were calculated according to established procedures (Knapp et al., 1985; Fehr, 1987). Genetic correlations ($r_e$) were calculated by means of PROC GLM considering entries and environments as random effects (SAS Institute, Inc., 1990). Box’s test (Milliken and Johnson, 1992) was used to test for homogeneity of error variances between environments.

### Detection of QTL

The protocols for DNA isolation, Southern hybridization, and collection of segregation data at RFLP loci have been described (Veldboom et al., 1994). Ninety-four genomic and cDNA probes detected 103 RFLP loci. One hundred forty-four F3 lines were split from the soil level to the first node above the primary ear. Anthesis was only measured at the Ames environments, while ear height was measured at all environments. Anthesis was considered a separate environment. Soil fertilization, weed control, and cultivation practices were consistent with optimum maize production for this region. The entries in each experiment consisted of the 150 F3 lines and two entries each of B73, De811, and the F1. Entries were evaluated in hill plots consisting of two hills spaced on centers of 0.76 m at Ames and 1.02 m at Ankeny, and were arranged in a 12 × 13 simple lattice design with two replications per environment. Plots were overplanted and thinned to three plants per hill (i.e., six plants per plot; Guthrie et al., 1985).

### Phenotypic data for the parents, F1, and F3 lines of B73 × De811.

<table>
<thead>
<tr>
<th>Trait</th>
<th>Environment</th>
<th>B73 mean</th>
<th>De811 mean</th>
<th>F1 mean</th>
<th>F3 lines</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stalk tunneling</td>
<td>Ames 1989</td>
<td>80</td>
<td>35</td>
<td>38</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>Ankeny 1989</td>
<td>65</td>
<td>25</td>
<td>40</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>Ames 1990</td>
<td>29</td>
<td>19</td>
<td>9</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>58</td>
<td>26</td>
<td>31</td>
<td>37</td>
</tr>
<tr>
<td>Anthesis</td>
<td>Ames 1989</td>
<td>1017</td>
<td>1058</td>
<td>1008</td>
<td>1018</td>
</tr>
<tr>
<td></td>
<td>Ankeny 1989</td>
<td>892</td>
<td>919</td>
<td>893</td>
<td>890</td>
</tr>
<tr>
<td></td>
<td>Ames 1990</td>
<td>957</td>
<td>985</td>
<td>951</td>
<td>954</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>91</td>
<td>93</td>
<td>102</td>
<td>91</td>
</tr>
<tr>
<td>Ear height</td>
<td>Ames 1989</td>
<td>91</td>
<td>93</td>
<td>102</td>
<td>91</td>
</tr>
<tr>
<td></td>
<td>Ankeny 1989</td>
<td>957</td>
<td>985</td>
<td>951</td>
<td>954</td>
</tr>
</tbody>
</table>

§ LSD for Ear Height: A = 11, B = 11, C = 10.

A = comparison among F1 lines, B = comparison between F1 lines and parental inbreds, C = comparison among parental inbreds.
RESULTS

In all environments, B73 and De811 differed significantly for ECB tunneling, and there were significant differences among the F2 lines (Table 1). The F2 values for ECB tunneling were close to De811 and differed significantly from B73. The highest levels of ECB tunneling in the parents and F2 lines and the greatest amount of genotypic variation were observed in Ames 1989. The genotypic variation for tunneling was almost 10 times greater for 1989 than 1990 at Ames. The combined precipitation in July and August at Ames was lower in 1989 (11 cm) and higher in 1990 (30 cm) than the 40-yr average (21 cm). Precipitation in July and August at Ankeny in 1989 (22 cm) was similar to the 40-yr average (20 cm). The excessive precipitation at Ames 1990 likely increased larval mortality. The variance for genotype (36) and genotype × environment [16, 95% confidence interval (95% CI) = 9–38] in the mean environment were not significantly different from the F2 lines of B52 × B73 (69 and 10, respectively), while the error variance herein was lower (85 and 141, respectively; Schön et al., 1993). For both experiments the error variance accounts for a large fraction of the phenotypic variance, illustrating the complications with assessing resistance to tunneling. The broad-sense heritability for tunneling was 0.65 (95% CI = 0.55–0.71), which is comparable to previous studies (0.63–0.78; Sadegdel-Moghaddam et al., 1983; Schön et al., 1993; Cardinal et al., 2001).

Seven QTL for ECB tunneling were detected on chromosomes 1, 3, 4, 5, and 8 in the mean environment (Fig. 1 and Table 2). The QTL were associated with 42% of the phenotypic variation, and all exhibited significant additive effects. Significant dominance effects were evident for one QTL (chromosome 1, umc13). Alleles from De811 were associated with decreased tunneling at five QTL. Epistatic effects were not detected.

QTL for ECB tunneling in the individual environments differed from those in the mean environment (data not shown). Only the QTL on chromosome 5 (umc68-umc51) was detected in all environments, while the QTL on chromosome 3 (umc102) was detected in both 1989 environments and the mean environment. Five and four QTL were observed for Ankeny 1989 and Ames 1990, respectively, but only those on chromosomes 3 (umc102) and 5 (umc68-umc51) were also observed in the mean environment. The QTL on chromosome 1 (umc13) was detected in Ames 1990 at LOD 4.8, but was excluded from the model because it did not increase the AIC by at least 2.0. Detection of that QTL was dependent on a cofactor (bnl12.06a), indicating that there may be a QTL in the region between bnl12.06a and bnl7.08.

Anthesis date and ear height are potentially confounding effects on the assessment of resistance to ECB tunneling (Dicke, 1954; Coors, 1987) because larval survival is affected by availability of pollen, and the length of the stalk may determine the amount of tunneling observed. Significant differences for anthesis and ear height were observed among F2 lines in individual environments and the mean environment (Table 1). The broad-sense heritabilities for those traits were 0.68 (95% CI = 0.55–0.76) and 0.87 (95% CI = 0.83–0.89), respectively. The genetic correlations (rG) between anthesis date and ear height and ECB tunneling were −0.36 (P < 0.001) and 0.35 (P < 0.05), respectively.

Seven QTL for anthesis were detected on chromosomes 1, 3, 5, and 7 (Fig. 1 and Table 3), and 11 QTL for ear height were observed on chromosomes 1, 2, 3, 4, 5, 6, and 9 (Fig. 1 and Table 4) in the mean environment. The anther QTL on chromosomes 1 (umc11) and 3 (umc92) are within 15 cM of QTL for tunneling. On chromosome 1, later anthesis was linked with reduced tunneling, while on chromosome 3, delayed anthesis was linked with increased tunneling. The ear height QTL on chromosomes 1 (umc11), 4 (umc31), and 5 (umc68) are within 10 cM of QTL for ECB tunneling. In those regions, decreased ear height is linked to decreased ECB tunneling.
DISCUSSION

Four studies of QTL for ECB tunneling have been performed with different resistant parents crossed to the same susceptible parent (B73; Schön et al., 1993; Jampatong, 1999; Cardinal et al., 2001). Comparisons of QTL across populations, while complicated by sampling variation and differences in environments and methodology and limited by the number of common genetic loci, can provide an opportunity to assess genetic heterogeneity of a phenotype (van Ooijen, 1992; Jansen and...
Table 2. QTL for ECB stalk tunneling in the F3 maize population of B73 × De811 in the mean environment.

<table>
<thead>
<tr>
<th>Chrom.</th>
<th>Genetic locus† (GL)</th>
<th>Distance from GL</th>
<th>Additive</th>
<th>Dominance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Effect‡ (cm)</td>
<td>Partial $R^2$§</td>
</tr>
<tr>
<td>1</td>
<td>umc157</td>
<td>15</td>
<td>−3.3**</td>
<td>5.4</td>
</tr>
<tr>
<td>1</td>
<td>umc11</td>
<td>7</td>
<td>3.7**</td>
<td>8.2</td>
</tr>
<tr>
<td>3</td>
<td>umc102</td>
<td>0</td>
<td>−4.3**</td>
<td>24.7</td>
</tr>
<tr>
<td>4</td>
<td>bnl5.46</td>
<td>1</td>
<td>2.9**</td>
<td>12.6</td>
</tr>
<tr>
<td>5</td>
<td>umc166</td>
<td>0</td>
<td>−2.7**</td>
<td>9.0</td>
</tr>
<tr>
<td>5</td>
<td>umc68</td>
<td>6</td>
<td>−4.4**</td>
<td>21.3</td>
</tr>
<tr>
<td>8</td>
<td>bnl8.26</td>
<td>0</td>
<td>−2.7**</td>
<td>9.4</td>
</tr>
</tbody>
</table>

Total adjusted $R^2$| 42%

* Effect is significant at $P < 0.05$.
** Effect is significant at $P < 0.01$.
† The locus defining the interval that contains the QTL that is proximal to the telomere of the short arm of the chromosome.
‡ Allele from De811 is associated with an increase (+) or decrease (−) in the value of the trait.
§ Percentage of phenotypic variation explained by the QTL, maintaining all other QTL effects fixed.
¶ Percentage of phenotypic variation explained by a model including all QTL as main effects and adjusted for the number of parameters in the model.

Table 3. QTL for anthesis for the F3 lines of B73 × De811 in the mean environment

<table>
<thead>
<tr>
<th>Chrom.</th>
<th>Genetic locus† (GL)</th>
<th>Distance from GL</th>
<th>Additive</th>
<th>Dominance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Effect‡ (cm)</td>
<td>Partial $R^2$§</td>
</tr>
<tr>
<td>1</td>
<td>umc11</td>
<td>6</td>
<td>6.3**</td>
<td>10.7</td>
</tr>
<tr>
<td>1</td>
<td>umc83</td>
<td>7</td>
<td>−6.7**</td>
<td>11.1</td>
</tr>
<tr>
<td>3</td>
<td>umc92</td>
<td>0</td>
<td>6.1**</td>
<td>10.7</td>
</tr>
<tr>
<td>3</td>
<td>bnl3.18</td>
<td>0</td>
<td>9.0**</td>
<td>21.8</td>
</tr>
<tr>
<td>5</td>
<td>bnl7.71</td>
<td>0</td>
<td>7.7**</td>
<td>14.0</td>
</tr>
<tr>
<td>7</td>
<td>umc56</td>
<td>4</td>
<td>−7.2**</td>
<td>8.0</td>
</tr>
<tr>
<td>7</td>
<td>umc80</td>
<td>9</td>
<td>7.9**</td>
<td>7.7</td>
</tr>
</tbody>
</table>

Total adjusted $R^2$| 51%

* Effect is significant at $P < 0.05$.
** Effect is significant at $P < 0.01$.
† The locus defining the interval that contains the QTL that is proximal to the telomere of the short arm of the chromosome.
‡ Allele from De811 is associated with an increase (+) or decrease (−) in the value of the trait.
§ Percentage of phenotypic variation explained by the QTL, maintaining all other QTL effects fixed.
¶ Percentage of phenotypic variation explained by a model including all QTL as main effects and adjusted for the number of parameters in the model.

Table 4. QTL for ear height for the F3 lines of B73 × De811 in the mean environment

<table>
<thead>
<tr>
<th>Chrom.</th>
<th>Genetic locus† (GL)</th>
<th>Distance from GL</th>
<th>Additive</th>
<th>Dominance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Effect‡ (cm)</td>
<td>Partial $R^2$§</td>
</tr>
<tr>
<td>1</td>
<td>umc13</td>
<td>0</td>
<td>6.5**</td>
<td>33.5</td>
</tr>
<tr>
<td>1</td>
<td>umc83</td>
<td>5</td>
<td>−1.6</td>
<td>0.9</td>
</tr>
<tr>
<td>2</td>
<td>umc5</td>
<td>9</td>
<td>−3.6**</td>
<td>11.0</td>
</tr>
<tr>
<td>3</td>
<td>umc121</td>
<td>41</td>
<td>−3.5**</td>
<td>11.9</td>
</tr>
<tr>
<td>4</td>
<td>umc31</td>
<td>0</td>
<td>3.2**</td>
<td>8.1</td>
</tr>
<tr>
<td>4</td>
<td>umc23b</td>
<td>8</td>
<td>−5.0**</td>
<td>9.9</td>
</tr>
<tr>
<td>4</td>
<td>bnl7.65</td>
<td>1</td>
<td>5.1**</td>
<td>14.0</td>
</tr>
<tr>
<td>5</td>
<td>umc68</td>
<td>11</td>
<td>−3.7**</td>
<td>14.0</td>
</tr>
<tr>
<td>6</td>
<td>npi235</td>
<td>0</td>
<td>−3.9**</td>
<td>14.8</td>
</tr>
<tr>
<td>6</td>
<td>umc38a</td>
<td>1</td>
<td>−2.5**</td>
<td>7.7</td>
</tr>
<tr>
<td>9</td>
<td>umc81</td>
<td>4</td>
<td>6.0**</td>
<td>29.3</td>
</tr>
<tr>
<td>9</td>
<td>bnl5.09</td>
<td>1</td>
<td>−3.4**</td>
<td>12.0</td>
</tr>
</tbody>
</table>

Total adjusted $R^2$| 58%

* Effect is significant at $P < 0.05$.
** Effect is significant at $P < 0.01$.
† The locus defining the interval that contains the QTL that is proximal to the telomere of the short arm of the chromosome.
‡ Allele from De811 is associated with an increase (+) or decrease (−) in the value of the trait.
§ Percentage of phenotypic variation explained by the QTL, maintaining all other QTL effects fixed.
¶ Percentage of phenotypic variation explained by a model including all QTL as main effects and adjusted for the number of parameters in the model.

Seven QTL were detected for ECB tunneling in the F3 lines of B73 × B52. Herein, the QTL on chromosome 3 (umc102) is in the same region (i.e., within 25 cM), and the alleles from the resistant parent (B52 and De811) were associated with a decrease in tunneling. The QTL on chromosomes 3 (umc102) and 5 (umc68) are in the same regions as QTL detected in recombinant

Stam, 1994; Zeng, 1994; Visscher et al., 1996). Herein, all comparisons will be made between QTL for ECB tunneling detected in the mean environment and will be based on common marker loci. The F3 lines of B73 × B52 were evaluated in the same environments in 1989 as the population herein, and this should enhance comparisons of QTL detected in those two populations.
inbred lines (RILs) of B73 × B52 (Cardinal et al., 2001),
and the QTL on chromosome 5 is also in the same
region as one detected in F1 lines of B73 × Mo47 (Jampatong, 1999).
In all populations, alleles from the resis-
tant parent (De811, B52, and Mo47) were associated
with decreased tunneling.

On the basis of common genetic loci, four QTL for
ECB tunneling herein are in the same regions as QTL
for resistance to leaf feeding by the southwestern corn
borer (SWCB, Diatraea grandisella Dyar) and the sugar-
cane borer (SCB, Diatraea saccharalis Fabricius) in two
populations of tropical maize. The QTL on chromo-
somes 1 (umc157 and umc11), 5 (umc68), and 8
(bnl8.26) are in the same regions (i.e., within 25 cm) as
those for resistance to leaf feeding by SWCB, SCB, or
both (Groh et al., 1998). The linkage between resistance
to leaf feeding by SWCB and SCB and ECB tunneling
was unexpected because resistance to leaf feeding
and tunneling by ECB in temperate maize has a low geno-
tic basis (Hawk, 1985; Lee et al., 1990; Barry
et al., 1995; Senior et al., 1998). The detection of differ-
ent QTL in crosses of B73 to B52, De811, and Mo47
may be due to genetic heterogeneity among the resis-
tant inbreds. Some QTL for ECB tunneling were also
detected in studies of resistance to leaf feeding by
SWCB and SCB, which suggests that there may be
common mechanisms of resistance to these different species
and feeding stages. The evidence of genetic heterogeneity
among the inbreds, specifically B52 and De811, suggests
that breeding could combine these sources of resistance
to produce germplasm with higher levels of resistance
to ECB tunneling.

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