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# QTLs for resistance to *Setosphaeria turcica* in an early maturing Dent×Flint maize population

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**Abstract** Quantitative trait loci (QTLs) for resistance to the fungal pathogen *Setosphaeria turcica*, the cause of northern corn leaf blight (NCLB), were mapped in a population of 220 $F_3$ families derived from a cross between two moderately resistant European inbred lines, D32 (dent) and D145 (flint). The population was genotyped with 87 RFLP and 7 SSR markers. Trials were conducted in the field in Switzerland, and in the greenhouse with selected $F_3$ families in Germany. The $F_3$ population segregated widely for resistance with transgression of the parents. By composite interval mapping, a total of 13 QTLs were detected with two disease ratings (0 and 3 weeks after flowering). Together these QTLs explained 48% and 62% of the phenotypic variation. Gene action at most QTLs was partially dominant. Eight out of the 13 QTL alleles for resistance were contributed by the more-resistant parent, D145. On chromosomes 3, 5 and 8, QTLs were located in the same chromosomal regions as QTLs in tropical and U.S. Corn Belt germplasm. Some QTLs affected NCLB, head smut and common rust at the same time, with alleles at these loci acting isodirectionally.

**Key words** Corn · *Exserohilum turcicum* · Northern corn leaf blight · Quantitative resistance · QTL mapping · *Setosphaeria turcica* · *Zea mays*

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## Introduction

The fungus *Setosphaeria turcica* Leonard & Suggs with its conidial state *Exserohilum turcicum* Leonard & Suggs causes northern corn leaf blight (NCLB), a foliar disease of maize that occurs in many environments. NCLB is particularly damaging in the tropics but awareness of the disease has also grown in western Europe (Welz et al. 1996, 1998). Cultivars with quantitative resistance are widely used to control NCLB and diverse sources of resistance have been described (Adipala et al. 1993; Smith and Kinsey 1993; Carson and Van Dyke 1994; Lipps et al. 1997; Schechert et al. 1997; Welz et al. 1998).

Quantitative trait loci (QTLs) affecting resistance to *S. turcica* were discovered by means of molecular markers in U.S. Corn Belt germplasm (Freymark et al. 1993,1994; Dingerdissen et al. 1996) and in tropical African maize (Schechert et al. 1999). In the present study, we mapped such QTLs in a European Dent×Flint population that was tested in a temperate environment. Our objectives were to: (1) assess the genomic position of QTLs for resistance to *S. turcica*, (2) estimate the magnitude of gene effects and the mode of gene action at the QTLs detected, (3) compare the QTL positions with those found in American and African maize germplasm, and (4) compare the positions of QTLs for NCLB resistance with those of QTLs for resistance to other fungal diseases, mapped previously in the same population (Lübberstedt et al. 1999).

## Materials and methods

### Mapping population

The early maturing European inbred lines D32 (dent) and D145 (flint), proprietary to the University of Hohenheim, were crossed in both directions to produce a random set of 220 $F_3$ families. This set was balanced such that an about-equal number of $F_3$ families occurred in the cytoplasm of D32 and D145, respectively. Seed of each $F_3$ family was increased by randomly crossing ten female and ten male $F_3$ plants (see Lübberstedt et al. 1999). The parent lines both have a moderate level of NCLB resistance (Welz et al. 1998).

### Field trial

The mapping population was tested in one field trial at Cadenazzo, near Bellinzona at Lago Maggiore, Switzerland, in 1998. The same field had been used successfully in previous, naturally infected NCLB trials (Welz et al. 1998). The experiment included 230 entries planted in a 23×10 alpha design (Patterson and Williams 1976) with two replicates. Entries comprised 220 $F_3$ families, two of each of the parent lines D32 and D145, two of each of their $F_1$ hybrids, two resistant check inbreds (CML202 and D164), and two susceptible check inbreds (Lo951 and KW3231). The check inbreds were described by Welz et al. (1998) and Schechert et al. (1999). Plots consisted of single rows, 5-m long, spaced 75-cm apart. They were planted on May 6th at a density of 30 plants per row, i.e. 8 plants $m^{-2}$.

The trial was inoculated with a conidial suspension on June 4th, at the 4–6-leaf stage, by means of a repetitive pipette (see Schechert et al. 1999). One 100-µl drop containing 1000 conidia was placed in the leaf whorl of each plant. The inoculum was prepared from dried infected leaves which had been collected in the same and adjacent fields in 1997, and were incubated for 3 days in the laboratory. The disease severity was scored twice, on July 22nd and on August 14th (DS 1 and 2). Ten consecutive plants in a row were rated individually for the percentage of leaf area affected by NCLB to derive the plot mean (see Schechert et al. 1999).

Plant height was measured on August 19th as the distance between the stem base and the top leaf insertion on one representative plant per row.

### Greenhouse trial

The parental lines, the five most-resistant $F_3$ families, the five most-susceptible $F_3$ families, plus cv Helix as a susceptible check, were subsequently tested in a greenhouse pot trial at the University of Hohenheim. The trial was planted in randomized complete blocks with two replicates on August 5th, 1998. Each plot consisted of ten plants in 2-l pots. The trial was inoculated on September 9th using inoculum produced from the same source as for the field trial. It was scored once on October 11th, when most test entries were flowering. The incubation period of NCLB was also recorded on a single-plant basis, as the number of days between inoculation and the first appearance of wilted lesions (Welz et al. 1998).

## Molecular-marker analysis

The 220 $F_3$ families were genotyped with 87 RFLP and 7 SSR markers by Xia et al. (1999) who mapped QTLs for virus resistance in the same population. The linkage map had a total length of 1799 cM with an average interval length of 21.4 cM and was in good agreement with published maize maps (Xia et al. 1999).

### Data analysis

Phenotypic field trial data were analysed using the software package PLABSTAT (Utz 1993). Lattice-adjusted entry means of the 220 $F_3$ families were subjected to an analysis of variance (ANOVA) treating genotypes as a random variable. Estimates of variance components for the experimental error ($\hat{\sigma}^2$) and genotypes ($\hat{\sigma}^2_g$), and the repeatability ($\hat{h}^2$) of the traits DS 1, DS 2 and plant height, as well as phenotypic correlations among traits ($\hat{r}_p$), were computed. The means of parent lines (P1 and P2), midparent ($\overline{P}$), and $F_3$ families ($\overline{F}_3$) were compared by orthogonal contrasts (Snedecor and Cochran 1980, p 96). The greenhouse trial data were analysed with an ANOVA treating genotypes as fixed effects. Means were compared by least-significant differences (LSD, 5% level), and the phenotypic correlation between incubation period and blight severity was estimated.

To detect QTLs and estimate their gene effects, composite interval mapping (Zeng 1994) was employed using the software PLABQTL (Utz and Melchinger 1996) as described by Bohn et al. (1996). Basically, this method is a multiple regression analysis with markers as co-factors. Co-factors were selected by stepwise regression, in order to minimize Akaike's information criterion with a penalty of 3.0 (Jansen 1993). The significance threshold to postulate the presence of a QTL at a given genomic position was set at LOD=2.5. Two QTL positions on the same chromosome were regarded as different when they were separated by at least two markers and a minimum distance of 20 cM. The proportion of the phenotypic variance ($\hat{\sigma}^2_p$) explained by an individual QTL was given by the square of the partial regression coefficient (Melchinger et al. 1998). The proportion of the phenotypic variance explained by all detected QTLs was finally determined in a simultaneous fit.

## Results

Plant stands in the field trial developed well and the artificial infection was successful. The highly susceptible check inbred KW3231 was almost entirely killed by NCLB before flowering (DS 1, Table 1). The less-susceptible check inbred Lo951 from Italy was much less attacked by *S. turcica*. Check inbred D164 confirmed its

**Table 1** Means, variance components and repeatabilities of two disease severity ratings (DS 1 and 2) and plant height of the mapping population ($\overline{F}_3$), its parent lines (P1, P2), their $F_1$ hybrid and four check inbreds, in the field trial

| Parameter | DS 1 (%) | DS 2 (%) | Plant height (cm) |
|---|---|---|---|
| *Means of checks* | | | |
| CML202 | 0 | 0 | 236.6 |
| D164 | 10.5 | 37.7 | 121.1 |
| Lo951 | 32.3 | 57.0 | 191.9 |
| KW3231 | 90.9 | 100 | 135.7 |
| *Means of test entries* | | | |
| D32 (P1) | 36.9 | 69.4 | 171.5 |
| D145 (P2) | 24.1 | 47.7 | 149.6 |
| $\overline{P}$ | 30.5 | 58.6 | 160.6 |
| $F_1$ | 22.7 | 54.2 | 213.2 |
| $\overline{F}_3$ | 27.9 | 53.3 | 179.6 |
| *Standard error* (*all entries*) | 3.1 | 5.0 | 7.4 |
| *LSD-5%* (*all entries*) | 8.6 | 14.0 | 20.6 |
| *Variance components* | | | |
| $\hat{\sigma}^2_g$ | 94.1** | 201.3** | 282.5** |
| $\hat{\sigma}^2$ | 18.0 | 45.2 | 96.1 |
| *Repeatability* ($\hat{h}^2$) | 0.83 | 0.80 | 0.72 |

** Significant at $P = 0.01$

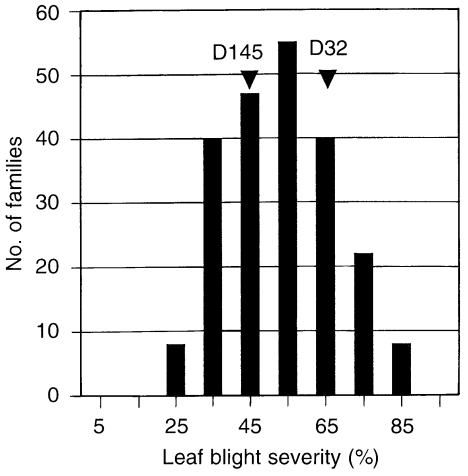


**Fig. 1** Frequency distribution of leaf-blight severity (DS 2) in a population of 220 $F_3$ families derived from the cross D32×D145, in a field trial inoculated with *S. turcica*, at Cadenazzo, Switzerland. Triangles indicate parental means assigned to histogram classes. Standard error of adjusted entry means=5.0% blight severity

moderate level of resistance (Welz et al. 1998) and CML202 proved its outstanding polygenic resistance (Schechert et al. 1999). The African CIMMYT inbred showed only one small lesion at the site of inoculation but no secondary infection.

The parental inbreds both showed a moderate level of resistance (Table 1), D145 being significantly (P=0.05) more resistant than D32, as in previous trials (Welz et al. 1998). The mapping population segregated transgressively (Fig. 1) and the genotypic variance components were highly significant (Table 1). Repeatabilities were high for both scorings. The $F_1$ hybrid and the $\bar{F}_3$ were more resistant than the midparent ($\bar{P}$) but these differences were not significant ($P$=0.05). The mid-parent heterosis for plant height in the $F_1$ generation amounted to 33% (Table 1). Plant height was weakly correlated with the severity of NCLB and other diseases ($\hat{r}_p$ values for DS 1:–0.16*, DS 2: –0.27**, common rust: –0.07, head smut: –0.22**, common smut: –0.13; */** indicating significance at $P$=0.05 or 0.01).

In the greenhouse experiment, the parent lines performed accordingly, D145 again being slightly more resistant than D32. Nine of the ten selected susceptible and resistant $F_3$ families reacted as in the field trial. Only inbred #52 behaved as a resistant outlier in the group of susceptible families. The phenotypic correlation between incubation period and disease severity was $\hat{r}_p$=–0.90 ($P$=0.01). For both traits the means among $F_3$ families were significantly different (ANOVA: F≥8.60, $df$=9, $P$<0.01). Assuming that the mean of the ten selected entries equalled the population mean, slight heterosis for resistance was also expressed by the $F_3$ families in the greenhouse (Table 2). The phenotypic correlation between disease severity in the greenhouse and in the field (DS 2) was $\hat{r}_p$=0.75 ($n$=12 entries).

A total of 13 QTLs were detected with the field data. Thirteen and 18 markers were used as co-factors in the multiple regression for DS 1 and DS 2, respectively. QTLs were identified on all chromosomes except 7 and 10 (Table 3). Six QTLs were significant for DS 1. The same six plus seven additional QTLs were significant for DS 2. Accordingly, the total proportion of the phenotypic variance explained by QTLs was higher for DS 2 than for DS 1 ($\hat{\sigma}^2_p$=62% vs 48%). For both scores a dominance model of inheritance gave the best fit to the data although the QTL-specific dominance effects were only significant in a few cases (Table 3). The average dominance ratio DR of the QTL (DR = $1/i \sum|d_i/a_i|$, for

**Table 2** Mean incubation period (IP) and disease severity (DS) of northern corn leaf blight in a controlled greenhouse trial with selected resistant and susceptible $F_3$ families of the mapping population $F_3$ (D32×D145), its parental inbreds and a susceptible check hybrid (cv Helix)

| Entry | IP (days) | DS (%) |
|---|---|---|
| *Check hybrid* ('Helix') | 12.9 | 23.0 |
| D32 (P1) | 14.1 | 13.5 |
| D145 (P2) | 14.8 | 12.8 |
| $\bar{P}$ | 14.4 | 13.2 |
| *Susceptible $F_3$s* | | |
| #25 | 14.4 | 13.7 |
| #30 | 14.8 | 18.1 |
| #52 | 17.0 | 10.7 |
| #59 | 13.8 | 18.5 |
| #180 | 12.7 | 32.5 |
| Mean | 14.5 | 18.7 |
| *Resistant $F_3$s* | | |
| #70 | 17.5 | 4.1 |
| #97 | 17.5 | 6.8 |
| #120 | 17.0 | 4.6 |
| #127 | 15.2 | 10.4 |
| #150 | 16.3 | 6.1 |
| Mean | 16.7 | 6.4 |
| $\bar{F}_3$ (10 entries) | 15.6 | 12.6 |
| *Standard error ($F_3$s only)* | 0.6 | 2.2 |
| *LSD-5% ($F_3$s only)* | 1.8 | 7.0 |

**Table 3** Genomic location of QTLs for northern corn leaf blight resistance and their additive (a) and dominant (d) gene effects, given as percent affected leaf area, in a population of 220 $F_3$ families derived from the cross D32×D145

| Chromosome | | | | DS 1 | | | | DS 2 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| No. | Bin[b] | Position (cM) | Flanking markers | a (%) | d (%) | $R^2$ (%) | LOD | a (%) | d (%) | $R^2$ (%) | LOD |
| 1 | 1.06/08 | 134/166 | *csu61b–dup12* | 2.33** | 2.52 | 5.8 | 2.8 | 2.56*[,cb] | –64 | 6.7 | 3.3 |
| 2 | 2.06 | 94 | *umc255–umc5a* | | | | | –3.64** | –46 | 7.5 | 3.7 |
| 3 | 3.01 | 14 | *umc32a–umc121* | | | | | –3.34** | –2.70 | 5.2 | 2.5 |
| | 3.03[d] | 42 | *umc38b–asg24* | | | | | 5.09** | –1.74 | 9.3 | 4.7 |
| | 3.07 | 168 | *umc3b–umc17a* | –2.53** | –1.01 | 8.4 | 4.2 | –5.13** | –2.78 | 11.8 | 6.0 |
| 4 | 4.03 | 16/28 | *phi21–csu253b* | –6.12** | –1.80 | 23.9 | 13.0 | –8.96** | 8.00 | 20.9 | 11.1 |
| | 4.06 | 78 | *umc47–umc66a* | | | | | –4.87** | 1.16 | 9.6 | 4.8 |
| 5 | 5.03 | 86 | *umc27a–umc43* | | | | | –3.43* | 6.32 | 6.5 | 3.2 |
| | 5.04 | 112 | *csu36a–bnl7.71* | –6.47** | –6.49** | 13.4 | 6.8 | –5.32** | –5.58 | 7.3 | 3.6 |
| 6 | 6.05/07 | 102/132 | *umc21–asg7* | –4.27** | 3.28 | 10.6 | 5.3 | –5.53** | 2.14 | 10.8 | 4.7 |
| 8 | 8.02/03 | 2 | *umc103a–bngl669* | | | | | 4.29** | 2.98 | 7.8 | 3.9 |
| | 8.06 | 74 | *umc17b–npi268a* | 3.88** | –0.62 | 10.1 | 5.1 | 7.19** | 1.42 | 19.2 | 10.1 |
| 9 | 9.02 | 56 | *umc105a–umc114* | | | | | 3.71** | 9.44* | 5.8 | 2.8 |
| Total | | | | | | 47.6 | 30.7 | | | 61.5 | 45.4 |

**, * QTL effect significant at $P$ =0.01 or 0.05, respectively
[a] Disease severity rating
[b] QTLs were assigned to bins according to reference maps (Maize Data Base 1999). A bin is defined as the interval between two fixed core marker loci and numbered by a code designating the linkage group and the location within the linkage group (Gardiner et al. 1993)
[c] Positive effect means that the QTL allele for resistance was contributed by the more-susceptible parent, D32
[d] Marker *umc38b* was previously mapped to bin 8.05 (Maize Data Base 1999)

**Table 4** Genetic relationship between diverse resistances in the mapping population $F_3$ (D32×D145). Above the diagonal: number of shared QTLs for resistance (in parentheses: number of QTLs with an equal additive effect sign) to *S. turcica* (NCLB ratings DS 1 and 2), *Sporisorium reiliana* (head smut), *Ustilago maydis* (common smut), and *Puccinia sorghi* (common rust). On the diagonal (bold face): number of QTLs detected for these traits. Below the diagonal: phenotypic correlation coefficient

| Item | NCLB 1 | NCLB 2 | Head smut[a] | Common smut[b] | Common rust[c] |
|---|---|---|---|---|---|
| NCLB 1 | **6** | 4[d] (4) | 3 (3) | 0 | 1 (1) |
| NCLB 2 | 0.84** | **13** | 4 (3) | 2 (1) | 3 (3) |
| Head smut | 0.37** | 0.29** | **8** | 3 (1) | 1 (1) |
| Common smut | 0.09 | 0.14* | 0.04 | **9** | 2 (0) |
| Common rust | 0.24** | 0.29** | 0.21** | 0.01 | **8** |

**, * Correlation coefficient significant at $P$=0.01 or $P$=0.05, respectively
[a] Assessed at Gongzhuling, China, in 1998 (see Lübberstedt et al. 1999)
[b, c] Assessed at Eckartsweier, Germany, in 1996 (see Lübberstedt et al. 1999)
[d] QTL positions were considered identical when the QTL likelihood curve peaks were ≤20 cM apart and separated by at least two markers

*i* QTLs) was 0.45 for DS 1 and 0.39 for DS 2. Stuber et al. (1992) defined a value between 0.2 and 0.8 as indicative of partial dominance. Eight out of the 13 QTL alleles for resistance were contributed by the more-resistant parent line, D145. Five alleles for resistance came from D32, the less-resistant parent.

The QTLs in bins 1.06/07, 3.07, 4.03, 5.04, 6.05/06 and 8.06 were significant for both leaf-blight ratings. The most highly significant QTL for DS 1, in bin 4.03, also carried the highest LOD score for the second disease rating. Repulsion-phase linkage of QTLs occurred on chromosome 3 (Table 3). We detected no digenic epistatic interaction effects among the QTLs for DS 1 ($P$=0.05). Epistasis could not be tested for DS 2 because the large number of QTLs could not be accomodated by the PLABQTL software.

QTLs for resistance to head smut, common smut and common rust were previously mapped in the same maize population (Lübberstedt et al. 1999). Some of these QTLs resided at the same chromosomal locations (Table 4). The three QTL alleles that provided for resistance to both head smut and NCLB rating 2, even attained exactly the same position in the two studies: chromosome 4–78 cM, 5–112 cM and 8–74 cM. One QTL for common rust resistance mapped to chromosome 5–114 cM. In most cases, the additive effects of various resistance genes at a given QTL had the same sign, i.e. the resistance alleles originated from the same parent line. In particular, the alleles for resistances to NCLB, head smut and common rust were genetically associated. The coefficients of phenotypic correlation among these three disease resistances were positive and significant, though only moderately (Table 4). Resistance to common smut

was phenotypically high independent from resistance to NCLB, head smut or common rust, and was apparently inherited by different genes.

## Discussion

As in previous trials (Welz et al. 1998), the parent lines D32 (dent) and D145 (flint) both had a moderate level of resistance, in the field and in the greenhouse. Their ranking in terms of resistance was also consistent across trials, D32 always being the more-susceptible line. According to pedigree information, line D145 has 25% of its genome from Mo17 (Messmer et al. 1992) which may explain some of its resistance (Carson 1995). Resistance phenotypes were characterized by a prolonged incubation period as in many other sources of quantitative resistance (Smith and Kinsey 1993; Carson 1995; Schechert et al. 1997, 1999), but otherwise showed normal lesion development. The mapping population segregated widely and, in either direction, a substantial portion of $F_3$ families transgressed the resistance of the parent lines (Fig. 1). This was probably due to the fact that the parents carried a similar number of resistance genes, most of which were either unlinked or linked in coupling, allowing for transgressive progeny to occur among the 220 $F_3$ families. However, even the most highly resistant $F_3$ family was significantly more diseased than CML202, a highly resistant African inbred line (Schechert et al. 1999), underscoring the breeding value of this tropical line.

In the greenhouse, but not in the field, $F_3$ family #52 showed extensive chlorotic spotting on the foliage. These symptoms were not due to fungal infection – they also appeared on uninfected new leaves – but most likely due to recessive detrimental genes with pleiotropic effects. Lesion-mimic genes could be involved. These mutant genes induce flecks and lesions of varying size, also in the absence of infection. In one study, the dominant lesion-mimic gene *Les1* promoted the hyphal growth of *S. turcica* and other fungi, resulting in larger lesions on infected plants (Echt and Trese 1989). In another study, lesion-mimic genes were associated with an enhanced expression of genes involved in systemically acquired resistance (Morris et al. 1998). There are at least 23, mostly dominant lesion-mimic genes located on several maize chromosomes (Neuffer et al. 1997). Since more than one of the selected $F_3$ families showed the fleck reaction, either of the two parent lines might have carried an undetected recessive lesion-mimic gene.

More QTLs were detected for DS 2, recorded about 3 weeks after flowering, than for DS 1, which was taken during the flowering period. The repeatability of DS 2 was also slightly higher than for DS 1. In a tropical maize population, most of the major QTLs were already detected by the incubation period at an early stage of plant development, and the least number of QTLs were detected after flowering (Welz et al. 1999). This different pattern may reflect the different adaptation of the germplasm used in the two studies, but the tight correlation between incubation period and disease severity in our greenhouse trial suggests that, also in this temperate maize population, most resistance genes were already expressed in the juvenile plant stage. This is confirmed by the similar shape of the QTL likelihood curves for DS 1 and 2 (data not shown).

A low level of partial dominance of the quantitative resistance was signified by the phenotypic generation means and by the average dominance ratio of QTL effects. In previous studies, the significance of dominance varied with the genetic material tested. Additive gene action prevailed (Jenkins and Robert 1952; Hughes and Hooker 1971; Sigulas et al. 1988; Carson 1995; Schechert et al. 1999), but in some crosses the heterosis was substantial (Schechert et al. 1997).

The magnitudes of individual gene effects were quite homogeneous, conforming with earlier QTL mapping studies (Dingerdissen et al. 1996; Schechert et al. 1999). The QTL with the largest effect for both disease ratings was located on the short arm of chromosome 4, in bin 4.03. This same genomic region was unimportant for NCLB resistance in tropical maize (Schechert et al. 1999) but carried a minor QTL in U.S. Corn Belt maize (Freymark et al. 1993). McMullen and Simcox (1995) speculated that variation at *bx1*, a locus on chromosome arm 4 S harboring genes for the production of DIMBOA (2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one), a preformed antimicrobial compound that acts against *S. turcica* (Couture et al. 1971), might be the underlying cause. However, in the reference linkage map of maize (Maize Data Base 1999), *bx1* and other *bx* loci were assigned to bin 4.01 rather than 4.02/03 (McMullen and Simcox 1995), making this explanation less likely. A biochemical assessment of DIMBOA production in genotypes grouped into opposite marker classes at the putative resistance QTL might help to clarify this issue.

The QTLs in chromosomal bins 3.07, 5.03/04 and 8.05/06 are also of special interest. These regions were significant for both disease ratings, DS 1 and 2, in this population and also in two other populations viz $F_3$ lines of the crosses Lo951×CML202 and B52×Mo17, having a largely different genetic background (Welz 1998; Schechert et al. 1999). Resistance alleles in bins 5.03/04 and 8.05/06 were shared by Mo17 and D145 and may be identical by descent in these lines. The universal importance of these QTLs may be due to duplication events because DNA probes from bin 8.05 also hybridized to DNA from bins 3.07/08 and 5.06/07 (Helentjaris 1995). Here, the marker *umc38b*, assigned to bin 8.05 in a reference map of maize (Maize Data Base 1999), resided in chromosome bin 3.03. Interestingly, two race-specific major genes, *Ht2* and *Htn1*, also lie in bins 8.05/06 (Simcox and Bennetzen 1993).

Since some genomic regions in maize are associated with resistance to various pests and diseases, there may be ”clusters” of closely linked resistance genes, or else gene action at the QTLs may be pleiotropic, either through shared physiological pathways or through multi-

functional gene products (McMullen and Simcox 1995). The compound DIMBOA, for example, is toxic for *S. turcica* and the European corn borer *Ostrinia nubilalis* (Molot and Anglade 1968). Our study uncovered QTLs for resistance to *S. turcica* (NCLB), *Sporisorium reiliana* (head smut) and *Puccinia sorghi* (common rust) that were either very closely linked or allelic. Again, the chromosomal bins 5.03/04 and 8.05/06 were involved. More detailed genetic studies are clearly needed to clarify the fine structure of these genomic regions.

Our results confirm that early maturing European inbred lines contain useful genes for quantitative resistance to *S. turcica*. Recombining moderately resistant inbred lines may lead to more-highly resistant progeny, as in this population. Broad-scale application of marker-assisted selection for NCLB resistance is certainly feasible, though not necessarily economic (Schechert et al. 1999), regarding the high heritability of quantitative NCLB resistance and the simplicity of conducting field tests. But marker-assisted conversion of proven elite inbreds may be a viable option to enhance valuable commercial hybrids that lack NCLB resistance. Their resistance to head smut or common rust could be simultaneously improved if donor genotypes and QTLs are carefully selected.

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