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Generation-means analysis and quantitative trait locus mapping of anthracnose stalk rot genes in maize

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Abstract A generation-means analysis was performed on two maize populations, each segregating for genes conferring resistance to anthracnose stalk rot (ASR). The populations were derived from a cross of DE811ASR × DE811 and of DE811ASR × LH132. The resistant parent, DE811ASR, was obtained through introgression with MP305 as the donor and DE811 as the recurrent parent. The analysis revealed significant additive effects in both populations and a significant additive × dominant effect in the DE811ASR × DE811 population. Quantitative trait locus (QTL) mapping, using restriction fragment length polymorphism (RFLP)-based molecular markers, indicated a significant QTL on linkage group 4 in both populations. The QTL analysis confirmed additive inheritance in both populations. This work demonstrates a close correspondence between generation-means analysis and discrete observations using molecular markers. Linkage of a genetic marker to genes conferring resistance to ASR will be useful for the introgression of resistance into elite germplasm.

Key words Restriction fragment length polymorphism Linkage mapping · Multiple regression · Anthracnose Maize

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Introduction

Anthracnose in maize (*Zea mays* L.), caused by the fungus *Colletotrichum graminicola* (Ces.) Wils., has recently become a major concern among breeders in the United States (Warren et al. 1973; Hooker 1976; Hooker and White 1976). The most common phases of infection by C. graminicola in maize are anthracnose leaf blight and anthracnose stalk rot (ASR). ASR is of more economic concern due to a significant yield reduction caused by stalk lodging, premature death, reduced grain weight (Smith 1976; Perkins and Hooker 1979; White et al. 1979), and in some cases crop failure (Warren et al. 1973). The disease occurs throughout the eastern part of the United States and extends north to New York and west to Illinois (Wheeler et al. 1974; Hooker and White 1976; White et al. 1979; Smith 1988).

At present the only economically-feasible means to control ASR are genetic resistance and modified cultural practices that reduce disease incidence (Hooker 1976; White et al. 1979). This includes crop rotation and tillage to avoid overwintering of the pathogen in infected tissue and the reduction of second-generation European corn borer (ECB) (*Ostrinia nubilalis* Hübner) infestation which can exacerbate ASR (Bergstrom et al. 1983; Keller et al. 1986). Determining the mode of inheritance and the chromosomal location of genes involved in resistance to ASR would facilitate the incorporation of ASR resistance into breeding lines.

Reports indicate that inheritance is often additive (Lim and White 1978; Carson and Hooker 1981), with dominance (Carson 1981; Badu-Apraku et al. 1987; Toman 1991) important in some populations. Reciprocal-translocation testcross analysis of an inbred, A556, indicated that genes for resistance were located on chromosomes 1, 4, 6

or 8 (Carson and Hooker 1982). The inbred line LB31 has been reported to carry a single dominant gene conferring resistance (Badu-Apraku et al. 1987), while MP305 was reported to have two resistance genes acting in a dominant fashion in crosses with A632 (Carson 1981). In a recent study, ASR resistance was primarily inherited in an additive fashion in crosses with DE811ASR, an inbred obtained using MP305 as a source of resistance (Weldekidan and Hawk 1993).

In view of the literature supporting a simply-inherited trait acting in an additive or perhaps dominant mode of inheritance, a directed approach to the mapping of ASR resistance would be the most appropriate. Near-isogenic lines (Martin et al 1991) can be used to identify the location of the genetic elements controlling the trait using molecular markers whose genetic position is known. By screening markers and identifying those showing polymorphism, the putative location of the resistance gene(s) can be determined. The use of molecular markers also allows one to proceed with a quantitative trait locus (QTL) analysis (Paterson et al. 1988) to prove that the region conditions resistance. This is done by genotyping and phenotyping a segregating population, then looking for associations between genotypes and the desired phenotype (resistance). Markers linked to genes conditioning resistance should show association with the trait. The inclusion of progeny testing in the analysis offers more opportunity to analyze the trait because phenotyping a family of individuals provides a more accurate estimate of the phenotypic value of the trait being measured (Paterson et al. 1991). The use of a map-based approach, such as QTL analysis, also enables one to look at other parts of the genome in an effort to find other regions conditioning resistance.

The use of classical phenotype analysis in the case of ASR would also provide more information on the mode of inheritance of resistance to ASR in this cross. Because of this, it was decided that a generation-means analysis would be appropriate (Mather and Jinks 1971). It was thought that the simultaneous use of a QTL and a generation-means analysis would provide a link between quantitative genetic theory and discrete molecular-marker analysis.

Materials and methods

Plant material and inoculations

Two populations of maize, each segregating for resistance to ASR, were developed for use in this study. Three backcrosses under selection for resistance were used to obtain an ASR-resistant line using MP305 as the donor parent and DE811 as the recurrent parent (Weldekidan and Hawk 1993). This line, DE811ASR (P_1), was then crossed to DE811 (P_2) and LH132 (also P_2) to develop F_1, F_2, F_3, BCP_1 (the first backcross to P_1), and BCP_2 (the first backcross to P_2), generations for the generation-means analysis.

In 1990, two rows of P_1, P_2 and F_1, six rows of F_2 and four rows of BCP_1 and BCP_2 generations were planted at the University of Delaware Experiment Station in Newark, Delaware, in a randomized complete block design with four replications. The DE811ASR × DE811 and DE811ASR × LH132 crosses hereafter will be denoted "DE811c" and "LH132c", respectively. Two rows each of inbreds MP305, LB31 and Pioneer hybrid 3241 (P3241) were planted as resistant checks and the Agway hybrid 788 (AG788) was planted as a susceptible check. The plots were 3.1 m long double rows with 76-cM spacing between rows. Irrigation was provided by overhead sprinklers for both years.

In 1991 all generations were planted as in 1990 with the addition of 200 and 172 F_3 families of ten plants each for the DE811c and LH132c populations, respectively, in a randomized complete block design with three replications. Two rows each of a resistant check, DE811ASR × LH51, and a susceptible check, Cargill hybrid 7877 (C7877), were included. Some F_3 families in the DE811c population had poor germination, so marker plants (Purple Genter) were sown to ensure adequate plant competition.

A mixture of six virulent *C. graminicola* isolates were used for the inoculations (White and Humy 1976; Weldekidan and Hawk 1993). Disease ratings were taken 4 weeks after inoculation by counting the total number of internodes discolored and the number of internodes greater than 75% discolored. The ratings were taken by visual inspection after splitting the stalks longitudinally from ear height to ground level (Carson and Hooker 1981).

Generation-means analysis

The generation-means analysis was performed by gathering the plot mean ASR rating for each generation. These data were then subjected to a weighted least squares regression using the following equation: $Y = m + a_1d + a_2h + a_3i + a_4j + a_5l$; where Y is the mean of a given generation, m is the midpoint, d is the pooled additive genetic effect, h is the pooled dominance genetic effect, i is the additive × additive genetic effect, j is the additive × dominance genetic effect, l is the dominance × dominance genetic effect, and a_1–a_5 are the respective coefficients of these effects in the equations of expectation of generation means (Mather and Jinks 1971; Carson and Hooker 1981). This regression may be curtailed to eliminate one or all of i, j, and l, depending upon how well the model fits as determined by R^2. The regression was performed with the SAS statistical package (v 6.06 SAS institute, Cary, N. C.) using PROC GLM and PROC REG.

Molecular-marker and QTL analysis

Seedling tissue from each inbred parent was collected, lyophilized, and stored at –20°C. DNA was isolated (Dellaporta et al. 1985) and restricted with one of three restriction endonucleases (*Eco*RI, *Eco*RV or *Hin*dIII, Boehringer Mannheim, Indianapolis IN), electrophoresed for 16 h at 30 V in TPE (0.08 M Tris phosphate, 0.002 M EDTA) buffer, blotted in 10 × SSPE (1.5 M NaCl, 0.1 M sodium phosphate, 0.01 M EDTA) buffer onto Immobilon-N (Millipore, Bedford Mass.) nylon membranes, and probed with ^{32}P-labeled probes prepared by the random primers method (Feinberg and Vogelstein 1983) using a random primers labeling kit (Gibco BRL, Gaithersburg Md.). Autoradiographs were made by exposing X-Omat AR film (Eastman Kodak, Rochester N.Y.) to the membranes at –70°C for 48 h in X-ray cassettes with one Cronex lightning plus intensifying screen (DuPont – NEN, Billerica Mass.) to reveal polymorphisms between the parents.

Molecular-marker analysis was begun by surveying the parents for polymorphisms using the University of Missouri public RFLP clone set (Gardiner et al. 1990). A subset of 113 markers spaced at 20-cM intervals throughout the genome (as per the UMC map) was surveyed for polymorphisms between DE811, LH132, MP305 and DE811ASR. Once polymorphisms were identified, DNA from 249 F_2 individuals for the DE811c population, and 231 F_2 individuals from the LH132c population, was isolated from tissue collected 2 weeks before pollination in 1990. F_2 progeny blots were made using one of the above enzymes, depending upon which enzyme-probe combination revealed polymorphisms between the parents in the cross. The progeny blots were probed and scored in a codominant fashion, and the scores were used for input into MAPMAKER v 1.9 (Lander et al. 1987) and MAPMAKER Macintosh v 1.0 to calculate

a genetic map with each marker on a group linked with a minimum LOD of 3.0 and a recombination fraction (ϕ) of 0.40. The order of the markers was established using ripple analysis to a LOD of 3.0.

Genotypes from the linkage map and phenotype ratings from F_2 individuals, and F_3 families derived from the F_2 individuals, were used for input into MAPMAKER QTL v. 0.9. The F_2 and F_3 data for each disease rating were analyzed separately using the method of interval mapping (Lander and Botstein 1989) and LOD scores. The LOD score is an expression of QTL likelihood, based on the $\log_{10}$ of the odds ratio, which is the likelihood of the data arising given a QTL at the locus divided by the likelihood of the data arising given no QTL at the locus (Paterson et al. 1988). A LOD of above 2.4 indicates that the presence of a QTL can be inferred. The F_3 family data were pooled from all plants in each family across the three replications. Families that had less than 25 plants from the three replications were not used. For the DE811c and LH132c populations, 158 and 151 F_3 families were used respectively. Gene action was also analyzed with MAPMAKER QTL using both F_2 and F_3 data for both populations.

Results and discussion

Generation-means analysis

Disease development was good in both 1990 and 1991 as indicated by the disease ratings for the P_2 and BCP_2 generations, which were significantly higher than the P_1 and BCP_1 generations (Table 1). The F_1, F_2 and F_3 generations had significantly-lower disease development than the susceptible parents. In 1990, MP305 and LB31 were resistant while P3241 had more disease development than the other resistant checks. The susceptible check AG788 had less disease development than the susceptible parents, so different check genotypes were used in 1991. The susceptible check C7877 had significantly-higher disease development in 1991 than the resistant parent and disease ratings for the resistant check DE811ASR×LH51 were not significantly different from the resistant parent.

In 1990 and 1991, generation-means analysis of the DE811c population indicated that 98 and 80%, respectively, of the variation for the total-number-of-internodes-discolored could be explained by additive effects with 9.5% of the variation explained by additive × dominant interactions in 1991 (Table 2). Analysis for the total number of internodes discolored rating in the LH132c population in 1990 and 1991 indicated that 70 and 99%, respectively, of the variation was explained by additive effects with no other effects being significant. The results of the analysis for the number of internodes with a greater than 75% discolored rating were similar to the total-number-of-internodes-discolored rating for both populations in 1990 and 1991 (Table 2).

The generation-means analysis indicates that inheritance of resistance to ASR is largely additive in both the DE811c and LH132c populations. There were significant additive effects for the 2 years in both populations for each rating. The additive × dominant interaction in the DE811c population in 1991 is consistent with a recent diallel study in which DE811ASR had a significant specific-combining-ability effect when crossed with OF9 (Weldekidan and Hawk 1993). This effect was only present in the DE811c population in 1991.

Molecular marker and QTL analysis

Of the 113 probes surveyed in DE811 and DE811ASR, seven detected polymorphisms. These alleles were shared by DE811ASR and MP305 indicating that they are probably introgressed regions from MP305. Forty probes were polymorphic between DE811ASR and LH132, including the seven mentioned above. All polymorphic probes in each cross were placed on the F_2 progeny blots with the exception of one in the LH132c population which was not scoreable.

MAPMAKER analysis of the DE811c population showed that the seven polymorphic markers fell into three linkage groups corresponding to chromosomes 3 and 9 on

Table 1 Mean rating for the total number of internodes discolored and the number of internodes greater than 75% discolored for each generation in the DE811c and LH132c populations in 1990 and 1991 at Newark, Delaware

Generation	Total number of internodes discolored				Number of internodes greater than 75% discolored			
	DE811		LH132		DE811		LH132	
	1990	1991	1990	1991	1990	1991	1990	1991
P_1	1.8	1.8	2.1	1.3	0.4	1.2	0.9	0.9
P_2	3.9	3.5	3.0	3.0	2.9	3.0	1.8	2.0
F_1	2.9	2.3	2.0	2.2	1.6	1.6	0.7	1.8
F_2	3.1	2.2	2.3	2.4	2.1	1.5	1.1	1.7
F_3	–	2.3	–	2.1	–	1.6	–	1.4
BCP_1	2.3	1.3	2.1	1.7	1.0	0.6	0.9	1.0
BCP_2	3.4	3.0	3.1	2.6	2.5	2.4	1.8	1.8
MP305	1.7	–	1.4	–	0.6	–	0.3	–
LB31	2.1	–	2.3	–	0.7	–	1.1	–
P3241	2.7	–	2.9	–	1.2	–	1.7	–
AG788	2.1	–	0.7	–	2.6	–	1.4	–
DE811ASR × LH51	–	1.4	–	1.6	–	0.9	–	0.7
C7877	–	4.4	–	3.1	–	4.0	–	2.5
LSD (0.05)	0.6	0.8	0.6	0.5	0.6	0.9	0.7	0.5

Table 2 Generation-means analysis for the total number of internodes discolored and the number-of-internodes greater-than-75%-discolored rating in the DE811c and LH132c populations in 1990 and 1991 at Newark, Delaware

Genetic effect	Variation explained for each effect							
	Total number of internodes discolored				Number of internodes greater than 75% discolored			
	DE811		LH132		DE811		LH132	
	1990	1991	1990	1991	1990	1991	1990	1991
d[a]	98.2*	80.0*	70.2*	99.3*	95.8*	78.3*	63.4*	94.5*
h	0.2	4.3	14.1	0.3	0.2	5.9	21.5	1.3
i	1.1	3.9	0.7	0.1	3.5	4.8	1.2	1.3
j	0.1	9.5*	9.1	0.1	0.2	9.2*	8.4	2.1
l	0.3	1.9	5.7	0.1	0.3	1.3	5.3	0.8

[a] d is the pooled additive genetic effect; h is the pooled dominance genetic effect; i is the pooled additive × additive genetic effect; j is the pooled additive × dominance genetic effect; and l is the pooled dominance × dominance genetic effect

* Significant at the P=0.05 level of probability

Table 3 MAPMAKER QTL analysis of the genetics of the putative QTL for ASR resistance when constrained by different modes of inheritance

Data	Genetics									
	Total number of internodes discolored					Number of internodes greater than 75% discolored				
	Free		Add.	Dom.	Rec.	Free		Add.	Dom.	Rec.
	LOD	%Var	ΔLOD	ΔLOD	ΔLOD	LOD	%Var	ΔLOD	ΔLOD	ΔLOD
DE811 F_2[a]	13.0	21.8	−2.2	−10.2	−0.6	15.6	25.5	−1.3	−10.5	−1.9
DE811 F_3[a]	45.1	73.2	−2.6	−32.9	−13.1	42.5	71.1	−3.9	−32.5	-9.7
LH132 F_2[b]	8.2	16.3	−1.5	−6.1	−0.3	14.8	27.6	−4.9	−12.8	−0.0
LH132 F_3[b]	31.5	64.9	−2.8	−21.3	−7.3	24.6	55.4	−2.4	−16.5	−4.7

[a] Data generated at the UMC15 locus

[b] Data generated at the point between UMC15 and UMC66 with the largest LOD score in each rating analysis

the UMC map (Gardiner et al. 1990), and with a single marker on chromosome 4. Some markers mapped to different locations from the published map, indicating that a different locus was mapped in this population. For example, UMC140 cosegregated with the markers on chromosome 9 here, but was previously published on chromosome 1 (Gardiner et al. 1990). When the data were analyzed with MAPMAKER QTL, none of the markers on linkage groups 3 and 9 showed a significant correlation to the phenotypes. However, using both F_2 and F_3 data, UMC15 (the single marker on chromosome 4) showed a significant association with the trait. This was found by forcing MAPMAKER to consider the single marker as two by duplicating the genotypes of UMC15 and giving the duplicate marker another name. When this is done, MAPMAKER performs what is essentially an analysis of variance in an interval of 0 cM between the marker and its duplicate scores.

In the F_2 data, a LOD score of 13 was found at UMC15 explaining 22% of the variation for the total number-of-internodes discolored rating. For the F_3 data using the total-number-of-internodes-discolored rating, a LOD of 45 was found with 73% of the variation explained. Similar LOD scores were found for the number of internodes with a greater than 75% discolored rating for the F_2 and F_3 data (Table 3). The LOD score of 45 means that the data are roughly 10^{45} more likely to have arisen given a QTL versus the data arising given no QTL at the locus. A LOD score of greater than 2.4 indicates that the presence of a QTL can be inferred.

The 39 polymorphic markers in the LH132c population came from regions corresponding to every linkage group except chromosome 2 on the UMC map, with some groups better represented than others. An estimated 75% of the genome was within 30 cM of a marker, indicating that most of the genome was assayed. These markers were used in an effort to find regions contributing to resistance, other than those derived from MP305, segregating in the DE811c population. LH132 and DE811ASR are 55% similar according to a germplasm survey conducted at DuPont (unpublished data), so a placement of many more markers on this population would not necessarily reveal more polymorphisms. A multipoint map was calculated from segregation data generated with these markers and is presented in the QTL scan (Fig. 1). Two markers, BNL6.32 and UMC98 from chromosomes 1 and 2, respectively, on the UMC map did not show linkage. These markers were included in the QTL analysis by duplicating their scores as

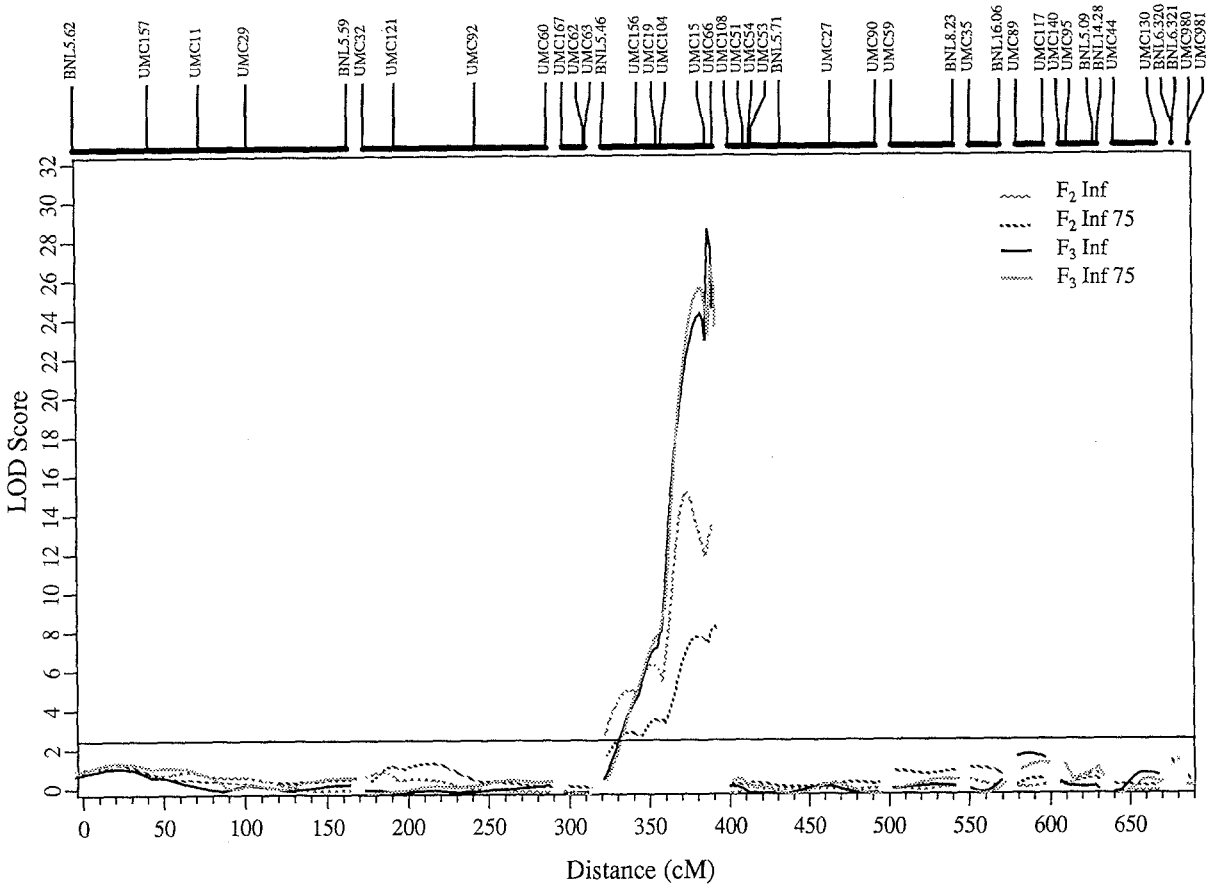

Fig. 1 ASR QTL likelihood plot for F_2 individuals and F_3 families in the DE811ASR × LH132 population. The total number of internodes discolored for the 1990 F_2 data is indicated by "F_2 Inf", the number of internodes greater than 75% discolored is indicated by "F_2 Inf 75". The F_3 familiy data for 1991 are labelled similarly. The *dark bars* at the top represent linkage groups from a MAPMAKER calculated map. These groups are not identical to those on the published map (Gardiner et al. 1990) since other loci were mapped here. The unlinked loci are at the right and distances on this map are shown on the x-axis. The LOD score (y-axis), which is an expression of the odds ratio (Paterson et al. 1988), is from a MAPMAKER QTL scan treating the four traits separately. A LOD score above 2.4 indicates the presence of a QTL

mentioned above. The results from analysis of the LH132c F_2 population show a QTL on chromosome 4 between UMC15 and UMC66 (Fig. 1). In the F_2 data a LOD score of 8 was found at this QTL explaining 16% of the variation for the total-number-of-internodes-discolored rating. For the F_3 data using the total-number-of-internodes-discolored rating, a LOD of 31 was found with 65% of the variation explained. Similar results were found for the number of internodes with a greater than 75% discolored rating (Table 3).

The QTL mapping results provided strong evidence for an ASR-resistance QTL on linkage group 4. None of the other linkage groups show evidence above the LOD 2.4 threshold for a QTL to be inferred (Fig. 1). The same region shows a QTL in both populations for phenotypic data collected in 1990 F_2 individuals and 1991 F_3 families. The entire region coding for resistance may be less than 12 cM, because the markers UMC52 (6.8 cM away) and UMC133 (5.2 cM away) flanking UMC15 do not show polymorphism even when many enzyme-probe combinations are used (data not shown). This is consistent with the assumption that the disease-resistance locus is a small region, with one or a few genes present, as has been shown for other disease-resistance loci (Hulbert and Michelmore 1985). The other introgressed regions from MP305 did not contribute to resistance; evidently they were not lost during backcrossing since only three backcrosses were used in the introgression to create DE811ASR. This is not a surprising result if the only genes involved are at the locus on chromosome 4. The existence of other important loci is unlikely, because they presumably would have been selected in the creation of DE811ASR. The analysis of the 39 markers polymorphic in the LH132c population did not uncover other regions coding for resistance in addition to the regions derived from MP305. There may be other loci with minor effects coming from the LH132 background that were too small to detect with the population sizes used here (about 230 F_2 and about 150 F_3 individuals). Loci with detectable effects not included in the 75% of the genome assayed here could also be present.

MAPMAKER QTL was also used to determine whether the data were likely to fit an additive, dominant, or recessive model of inheritance. The analysis was performed by comparing the likelihood that the data would arise assuming a particular mode of inheritance with the likelihood

that the data would arise given no assumptions about the mode of inheritance (the most-likely or "free" model). The results of this analysis are presented by MAPMAKER as ΔLOD scores, with a large negative ΔLOD indicating that a particular mode of inheritance is not as likely, since its ΔLOD deviated the most from the "free" model with no inheritance assumptions (Table 3).

The results of the inheritance analysis for the DE811c population indicated recessive gene action for the F_2 and additive inheritance for the F_3 data (Table 3). Additive inheritance did not deviate significantly from the "free" model for the F_2 data and actually explained the data better for the number of internodes with a greater than 75% discolored rating. For the LH132c population, recessive or additive gene action was again most likely in the F_2 data and additive inheritance was most probable in the F_3 data (Table 3). These data confirm the results of the generation-means analysis for the additive effects seen in both populations and are also consistent with the recent diallel study of DE811ASR (Weldekidan and Hawk 1993). The close correspondence between the results of the generation means analysis and QTL analyses is reassuring in terms of this trait as well as the relationship between classical quantitative genetic theory and molecular-marker analysis.

The linkage of UMC15 and UMC66 to a major QTL for ASR resistance using MP305 as a source of resistance should enable breeders to use marker-aided selection for faster introgressions. This type of strategy was used in 1992 to select for the MP305 allele at UMC15 and against the other introgressed regions from MP305 in DE811ASR in order to retain the DE811 background as a source of resistance to the European Corn Borer (unpublished data). An inbred line with high levels of ECB and ASR resistance would be valuable to maize breeders.

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