Quantitative Trait Loci Mapping of Resistance to Sugarcane Mosaic Virus in Maize

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ABSTRACT

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Sugarcane mosaic virus (SCMV) is an important virus disease of maize (*Zea mays*) in Europe. In this study, we mapped and characterized quantitative trait loci (QTL) affecting resistance to SCMV in a maize population consisting of 219 F₃ or immortalized F₂ families from the cross of two European maize inbreds, D32 (resistant) × D145 (susceptible). Resistance was evaluated in replicated field trials across two environments under artificial inoculation. The method of composite interval mapping was employed for QTL detection with a linkage map based on 87 restriction

fragment length polymorphism and 7 mapped microsatellite markers. Genotypic and genotype × environment interaction variances for SCMV resistance were highly significant in the population. Heritabilities ranged from 0.77 to 0.94 for disease scores recorded on seven consecutive dates. Five QTL for SCMV resistance were identified on chromosomes 1, 3, 5, 6, and 10 in the joint analyses. Two major QTL on chromosomes 3 and 6 were detected consistently in both environments. Significant epistatic effects were found among some of these QTL. A simultaneous fit with all QTL in the joint analyses explained between 70 and 77% of the phenotypic variance observed at various stages of plant development. Resistance to SCMV was correlated with plant height and days to anthesis.

Sugarcane mosaic virus (SCMV) is one of the most important virus diseases of maize (*Zea mays* L.) and causes serious yield losses in susceptible cultivars (7). In Germany, SCMV is more prevalent than maize dwarf mosaic virus (MDMV) and causes increasing damage to maize (8), while MDMV is a widespread viral disease in the southern U.S. corn belt (24). Taxonomically, SCMV as well as MDMV and wheat streak mosaic virus (WSMV) belong to a group of related pathogenic potyviruses in maize (20). SCMV is naturally transmitted by aphids in a nonpersistent manner. It is not possible to control SCMV directly with chemical means. Likewise, control of the aphid vectors is not effective because of the nonpersistent mode of virus transmission. Therefore, cultivation of resistant maize varieties is the preferable way to control SCMV infections.

Studies on the resistance to MDMV, SCMV, and WSMV have been conducted mainly with U.S. maize germ plasm. In the maize inbred Oh7B, resistance to MDMV is controlled by a single dominant gene (48). McMullen and Louie (37) found a major gene, designated *Mdm1*, that confers resistance to MDMV in Pa405 and is located between restriction fragment length polymorphism (RFLP) markers *bnl6.29* and *umc85* on the short arm of chromosome 6 (6S). A more detailed study showed that *Mdm1* was perfectly cosegregating with the nucleolus organizer region (*nor*) (54). In the cross Pa405 × Oh28, three genes (*Wsm1*, *Wsm2*, and *Wsm3*) conferred resistance to WSMV (35,36). Additional virus resistance genes in maize were identified on chromosome 1 (*Msv1* against maize streak virus [MSV]) (61), chromosome 3 (*Mv1* against maize mosaic virus [MMV]) (43), and chromosome 6 (*HPV* against high plains virus) (33).

In a recent study with early-maturing European maize germ plasm, 122 inbreds were screened under both greenhouse and field conditions (21). Three lines (D21, D32, and FAP1360A) were found to be completely resistant to SCMV, MDMV, Johnson grass mosaic virus, and sorghum mosaic virus. Preliminary investigations on the inheritance

indicated that one to three genes conferred resistance to SCMV in different crosses. Two major resistance genes, *Scm1* and *Scm2*, mapped to chromosomes 6S and 3, respectively (40). Linkage analysis of 40 BC₅ progenies derived from the cross FAP1360A × F7 suggested that *Scm1* is closely linked with amplified fragment length polymorphism marker *E3M8-1* (0.0 centimorgan [cM]) and that *Scm2* is located distal of RFLP marker *umc102* (4.4 cM) (63). Because of the small population size employed by these authors, it was neither possible to investigate the gene action of *Scm1* and *Scm2* nor to identify minor resistance genes.

In this study, we mapped quantitative trait loci (QTL) for resistance to SCMV using a population of F₃ or corresponding immortalized F₂ (IF₂) families derived from the cross D32 × D145. Our objectives were to (i) estimate the number and chromosomal position of major and minor genes responsible for SCMV resistance in this cross, (ii) determine their gene action, (iii) identify molecular markers linked to these QTL, and (iv) examine associations between resistance to SCMV and other agronomic traits.

MATERIALS AND METHODS

Plant materials. Two early-maturing European inbred lines, D145 (flint) and D32 (dent), were crossed in both reciprocal forms to produce a random set of 220 F₃ families (population D32 × D145). A subset of 122 F₃ families had the cytoplasm of line D145, and the remaining 98 F₃ families had the cytoplasm of D32. For each F₃ family, a pool of 20 F₃ plants was chosen to generate an IF₂ (9) population by randomly crossing 10 F₃ plants as females and 10 F₃ plants as males. Line D32 is completely resistant to SCMV, whereas line D145 is highly susceptible (21).

Inoculum preparation and inoculation. Virus inoculum for testing resistance against SCMV isolate Seehausen was prepared as described by Fuchs and Grüntzig (7). Young leaves with typical mosaic symptoms of the SCMV-infected maize variety Bermasil were homogenized using 5 volumes of a 0.01-M phosphate buffer at pH 7.0. Carborundum was added to the sap. During sap preparation and mechanical inoculation, the inoculum was kept at +4°C. Plants at the three- to four-leaf stage were mechanically inoculated

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twice at a weekly interval by an airbrush technique with a tractor-mounted air compressor at a constant pressure of 800 kPa (8).

Agronomic trials. The F_3 population derived from the cross D32 $\times$ D145 was evaluated at Eckartsweier (located in the Upper Rhine Valley close to Straßburg, France) in 1996 and at Hohenheim (near Stuttgart, Germany) in 1997 for resistance against SCMV. The experiment included 220 F_3 or IF_2 families and both parent lines each included as quintuple entries. The 230 entries were grown in a 23×10 alpha lattice design with two replications (46). Plots consisted of single rows, 0.7 m apart and 3 m long. Plots were overplanted and later thinned to a final plant density of nine plants per m^2 with a total of 20 plants per row.

Resistance to SCMV was scored beginning 2 weeks after initial inoculation. Virus symptoms were recorded at weekly intervals on seven dates in 1996 and on nine dates in 1997. F_3 means from the fifth and eighth scorings in 1997 were not used for the combined analyses across environments because they were similar to the fourth and seventh scorings, respectively. The other seven scorings in 1997 were treated as the first to seventh scorings and used for the combined analyses across both years. These seven scorings on seven dates were designated VIR1 to VIR7. The susceptible parent D145 was, by mistake, substituted by the susceptible inbred D408 in the field experiment in 1996.

Plant height (PHT, in centimeters) was measured plotwise as the distance from soil level to the lowest tassel branch of a representative plant. Days to anthesis (DTA) refers to the number of days from sowing to anthesis for 50% of the plants in each plot.

Leaf collection and DNA extraction. Leaves were harvested from 20 plants per F_3 family after the second SCMV inoculation in 1996. Subsequently, equal amounts of leaf material from each plant were bulked to determine the marker genotype of the corre-

sponding parental F_2 plant. Harvested leaves were freeze-dried and ground to powder. DNA extraction was performed according to the cetyltrimethylammonium bromide method (11).

RFLP analyses. Genomic DNA was digested with restriction enzymes *EcoRI*, *HindIII*, *EcoRV*, *BamHI*, and *BglI*. The resulting DNA fragments were separated by electrophoresis on 0.8% agarose gels and vacuum-blotted onto nylon membranes. Hybridization was carried out by using a nonradioactive detection method described by Hoisington et al. (11). A total of 105 maize DNA probes from the standard probe collection available at the University of Missouri, Columbia, was employed for screening parents D32 and D145. The resulting 76 polymorphic RFLP probes were applied to the F_3 population.

Simple sequence repeat (SSR) analyses. Additionally, seven mapped microsatellite SSR markers were analyzed (Fig. 1). Sequences of these primers were obtained from the maize database (55,56) and synthesized by Amersham Pharmacia Biotech (Freiburg, Germany). Polymerase chain reaction amplification and MetaPhor gel electrophoresis (FMC BioProducts, Rockland, ME) were performed according to Lübberstedt et al. (26).

Segregation and linkage analyses. Segregation at each marker locus was checked for deviations from Mendelian segregation ratios (1:2:1 or 3:1) and allele frequency 0.5 by χ^2 tests (60). Taking into account that multiple tests were performed, appropriate type I error rates were determined by the sequentially rejective Bonferroni procedure described by Holm (12). Estimates of the proportion of parental genome for each individual were obtained according to Paterson et al. (45). Linkage analysis of RFLP and SSR markers was conducted with Mapmaker 3.0b (22) software using Haldane's mapping function. The threshold used for linkage analyses was set to a $\log_{10}$ odds ratio (LOD) value of 3.0. A total of 87 RFLP and 7 SSR

Fig. 1. Combined linkage map for 87 restriction fragment length polymorphism and 7 microsatellite markers based on 219 F_3 families derived from the cross D32 $\times$ D145 and approximate map position of five quantitative trait loci (QTL) (including *Scm1* and *Scm2*) for resistance to sugarcane mosaic virus (SCMV) found in this study and other resistance genes against various viral and fungal diseases of maize reported in the literature. Three minor QTL for resistance to SCMV are indicated by *QSCM*. † = Map positions not in agreement with those previously published (44). * = Genotype frequencies deviated significantly from the Mendelian expectations (1:2:1 or 3:1).

marker loci well distributed over the maize genome was used to genotype the parental F₂ plants of all 219 F₃ families (Fig. 1). One F₂ plant had a homozygosity level of 90.9% and was treated as an outlier based on a χ^2 test. Consequently, the corresponding F₃ family was dropped from the statistical, linkage, and QTL analyses.

Statistical analyses. The rate of infection for virus was determined as percentage values. The biometric analysis of phenotypic data and QTL mapping were performed as described in detail by Lübberstedt et al. (27,29) with IF₂ families treated as F₃ families (denoted by this term throughout the remainder of this paper). Briefly, adjusted entry means and effective error mean squares from each location were used to compute combined analyses of variances across environments. The sums of squares for entries were subdivided into orthogonal contrasts (i) among both parent lines D32 and D145, (ii) the mean of both parents ($\bar{P}$) and the mean of the 219 F₃ families ($\bar{F}_3$), (iii) the means of the two subgroups of F₃ families differing in the origin of their cytoplasm, and (iv) the variation among F₃ families within each subgroup. A corresponding subdivision was conducted on the entry $\times$ environment (E) interaction sums of squares. *F* tests on contrasts as well as estimates of variance components σ_g^2 (genotypic variance), σ_{ge}^2 (genotype $\times$ E interaction variance), and σ^2 (error variance) of F₃ families and their standard errors were calculated as described by Searle (52). Heritabilities (h^2) on an entry-mean basis were estimated as described by Hallauer and Miranda (10):

$$\hat{h}^2 = \frac{\hat{\sigma}_g^2}{\hat{\sigma}_g^2 + \hat{\sigma}_{ge}^2/e + \hat{\sigma}^2/re}$$

in which *e* = number of environments and *r* = number of replications. Exact 90% confidence intervals of $\hat{h}^2$ were calculated according to Knapp et al. (19). Phenotypic correlations ($\hat{r}_p$) among traits were calculated by applying standard procedures.

Fig. 2. Proportion of sugarcane mosaic virus-infected plants for the resistant (D32) and susceptible (D145) parent lines and mean of the 219 F₃ families on various scoring dates in 1997.

We employed the method of composite interval mapping (66) for mapping of QTL and estimation of their effects as described by Bohn et al. (1). A LOD threshold of 3.0 was chosen for declaring a putative QTL significant; this ensures a comparison-wise type I error $P_c < 0.0032$ or a genome-wise type I error $P_g = MP_c < 0.29$ ($M = 84$, being the total number of intervals tested). For the analysis of data from each environment and also for the joint analyses across environments, cofactors were selected by stepwise regression. Final selection was for the model that minimized Akaike's information criterion with penalty = 3.0 (15). The joint analyses of QTL for phenotypic traits were based on the average of the families across two environments. QTL positions were determined at the local maxima of the LOD ($\log_{10}$ odds ratio) plot curve in the regions under consideration. The proportion of the phenotypic variance ($\hat{\sigma}_p^2$) explained by a single QTL was calculated as the square of the partial correlation coefficient (41). Putative QTL were examined for the presence of QTL $\times$ E and digenic epistatic interactions as described by Lübberstedt et al. (30). Each QTL was represented by a 20-cM interval with the local LOD maximum as center. QTL with overlapping 20-cM intervals among different traits were considered as being in common, irrespective of the sign of additive or dominance effects. All computations were performed with PLABQTL (58) software.

RESULTS

Segregation and linkage of RFLPs and SSRs. Sixty RFLP and SSR markers showed a codominant segregation ratio (1:2:1), and 34 marker loci displayed a dominant segregation ratio (3:1). Allele frequencies did not deviate significantly ($P > 0.05$) from 0.5 at any marker locus. At 6 out of the 94 marker loci, the observed genotype frequencies deviated significantly ($P < 0.001$) from the Mendelian expectations (1:2:1 or 3:1) (Fig. 1). The proportion of the D32 genome among the 219 F₃ families followed a normal distribution and ranged from 31.9 to 67.2% with mean $\bar{x} = 49.3\%$ and standard deviation (SD) = 7.2%. The level of homozygosity in the parental F₂ plants displayed approximately a normal distribution and varied from 20.0 to 69.5% with $\bar{x} = 47.2\%$ and SD = 10.0%. Our linkage map for the cross D32 $\times$ D145 is in good

TABLE 2. Phenotypic correlations and number of common quantitative trait loci (QTL)* among resistance to sugarcane mosaic virus, plant height (PHT), and days to anthesis (DTA) measured in two environments, estimated from 219 F₃ families of the maize population D32 $\times$ D145

Trait ^y	VIR5	VIR7	PHT	DTA
VIR1	0.73** ^z (2)	0.70** (2)	-0.36** (1)	0.46** (1)
VIR5		0.99** (4)	-0.44** (1)	0.47** (1)
VIR7			-0.44** (1)	0.49** (1)

* The number of common QTL is in parentheses. Additive gene effects from all common QTL had the same sign.

^y The first, fifth, and seventh dates are denoted as VIR1, VIR5, and VIR7, respectively.

^z ** = Phenotypic correlation was significant at the 0.01 probability level.

TABLE 1. Estimates of means, variance components, and heritabilities for resistance to sugarcane mosaic virus in 219 F₃ families from maize population D32 $\times$ D145 measured in two environments

Trait ^w	Mean ($\bar{F}_3$)	Variance components			Heritability (F ₃ families)	
		$\hat{\sigma}_g^2$	$\hat{\sigma}_{ge}^2$	$\hat{\sigma}^2$	$\hat{h}^2$	90% C.I. on $\hat{h}^2$ ^x
VIR1	7.6 $\pm$ 0.7 ^y	79.1 $\pm$ 10.0** ^z	15.8 $\pm$ 4.8**	30.3 $\pm$ 2.0	0.77	(0.71, 0.83)
VIR2	22.1 $\pm$ 1.5	392.5 $\pm$ 46.7**	126.9 $\pm$ 17.4**	52.6 $\pm$ 3.5	0.81	(0.76, 0.86)
VIR3	36.5 $\pm$ 1.9	701.6 $\pm$ 72.0**	42.8 $\pm$ 10.9**	63.4 $\pm$ 4.2	0.93	(0.91, 0.95)
VIR5	48.4 $\pm$ 2.0	846.1 $\pm$ 85.7**	38.8 $\pm$ 11.1**	67.6 $\pm$ 4.5	0.94	(0.92, 0.96)
VIR7	54.1 $\pm$ 2.0	842.6 $\pm$ 86.1**	54.8 $\pm$ 12.3**	66.2 $\pm$ 4.4	0.93	(0.91, 0.95)

^w Scorings of sugarcane mosaic virus symptoms on the first, second, third, fifth, and seventh dates are denoted as VIR1, VIR2, VIR3, VIR5, and VIR7, respectively.

^x Confidence intervals (C.I.) of $\hat{h}^2$ were calculated by the method of Knapp et al. (19).

^y Values include $\pm$ standard deviation.

^z ** = Significant at the 0.01 probability level.

agreement with published maps for temperate maize germ plasm, except for five loci that mapped to different positions (Fig. 1). The 94 markers span a map distance of 1,799 cM with an average interval length of 21.4 cM. All loci were significantly linked ($LOD > 3.0$) to one of the 10 linkage groups.

Phenotypic data. Both parents D32 and D145 differed significantly ($P < 0.01$) for resistance to SCMV in 1997 (Fig. 2). D32 expressed almost complete resistance to SCMV with only 1% infected plants, while 100% of all plants of D145 were diseased at VIR7. The mean proportion of infected plants across all 219 F_3 families increased from 8% on the first scoring date (VIR1) to 54% on the final scoring date (VIR7) averaged across both environments (Table 1). The correlation among F_3 family means between both environments for VIR7 was 0.88. No significant ($P > 0.05$) difference existed between the mean of both parents ($\bar{P}$) and the mean of all 219 F_3 families ($\bar{F}_3$) for VIR7 in 1997. The contrast between the two subgroups of F_3 families differing in the reciprocal form of the parental F_1 cross revealed significant ($P < 0.05$) cytoplasmic effects only for PHT and DTA, but not for SCMV resistance (VIR1 to VIR7).

Genotypic variances ($\hat{\sigma}_g^2$) were highly significant ($P < 0.01$) for SCMV resistance on all scoring dates (Table 1). Estimates of $\hat{\sigma}_{ge}^2$ were also significant for these traits, but significantly smaller than $\hat{\sigma}_g^2$. Heritabilities for VIR1 to VIR7 ranged from 0.77 to 0.94.

Phenotypic correlations were highly significant ($P < 0.01$) between resistance to SCMV and various agronomic traits such as PHT and DTA (Table 2). Correlations were highest ($\hat{r}_p \geq 0.70$) among VIR1, VIR5, and VIR7. These traits also showed moderate correlations with PHT ($-0.44 \leq \hat{r}_p \leq -0.36$) and DTA ($0.46 \leq \hat{r}_p \leq 0.49$).

QTL analyses. In the joint analyses for SCMV resistance, four, eight, seven, nine, and nine markers were used as cofactors for VIR1, VIR2, VIR3, VIR5, and VIR7, respectively. A similar set of cofactors were applied to identify QTL in individual environments. Five QTL were identified on chromosomes 1, 3, 5, 6, and 10 with significant effects on resistance to SCMV in the joint analyses across both years (Table 3). All QTL alleles conferring resistance to SCMV were contributed by the resistant parent D32, except for the QTL detected on chromosome 10. The largest QTL detected at all stages of plant development was located on chromosome 6S and explained between 56 and 47% of $\hat{\sigma}_p^2$ for VIR1

TABLE 3. Parameters associated with putative quantitative trait loci (QTL) significantly affecting resistance to sugarcane mosaic virus on various scoring dates (VIR1, VIR2, VIR3, VIR5, and VIR7) in 219 F_3 families of the maize population D32 $\times$ D145 measured in two environments

Bin ¹	Parameter ^u	VIR1 ^v			VIR2			VIR3			VIR5			VIR7		
		EWE96 ^w	HOH97	Comb	EWE96	HOH97	Comb	EWE96	HOH97	Comb	EWE96	HOH97	Comb	EWE96	HOH97	Comb
1.08	a_1	1	0	0	2	1		3	2		1	0		-1	2	0
	d_1	-10***	-16**	-13**	-27**	-19**		-31**	-25**		-26**	-22**		-23**	-24**	-23**
	R^2_1	7	7	8	6	7		10	10		7	7		6	6	7
	$a_1 \times E$			ns		ns			ns			ns			ns	ns
	$d_1 \times E$			ns		*			ns			ns				ns
3.04/3.05	a_3				4*	10**	7**	14**	18**	16**	22**	20**	22**	24**	24**	24**
	d_3				3	-15**	-7	0	-7	-4	-9	-8	-7	-9	-10	-10
	R^2_3				7	7	7	18	17	18	25	20	27	27	22	28
	$a_3 \times E$						*			ns			ns			ns
	$d_3 \times E$						ns		ns				ns			ns
5.01	a_5										5*		4**	6**	4*	5**
	d_5										-4		-6	-6	-10	-8
	R^2_5										7		6	7	6	8
	$a_5 \times E$												ns			ns
	$d_5 \times E$												ns			ns
6.00	a_6	12**	14**	13**	22**	42**	32**	38**	39**	38**	42**	38**	40**	42**	35**	38*
	d_6	-10**	-16**	-13**	-12**	-40**	-27**	-18**	-25**	-22**	-17**	-20**	-19**	-15*	-19**	-17**
	R^2_6	47	27	56	42	50	55	53	42	50	52	39	52	49	35	47
	$a_6 \times E$			ns			**						**			**
	$d_6 \times E$			ns			**						ns			ns
10.05	a_{10}	-5**		-3**												
	d_{10}	-2		2												
	R^2_{10}	20		8												
	$a_{10} \times E$			**												
	$d_{10} \times E$			ns												
Total	R^{2y}	55	49	62	60	74	76	71	66	74	72	64	74	71	60	71
	Epistasis															
	$d_1 \times a_6$		-29**	-22**												
	$d_1 \times d_6$		36*						-78*			-87*			-93*	
	$a_3 \times a_6$						8*						-14**		-18**	-16**
	$a_3 \times d_6$				16*			27*			36**		32**	39**		37**
Total	$a_6 \times a_{10}$	-7**		-5**												
	R^{2z}	63	53	70	61	74	77	71	66	75	73	64	76	71	62	74

¹ Chromosomes dissected into Bin regions and QTL positions based on the marker linkage map (Fig. 1) and Neuffer et al. (44) and Gardiner et al. (9). A Bin is a chromosome region flanked by core markers. These core (RFLP) markers have been selected to cover most of the 10 maize chromosomes. The average distance of neighboring core markers is about 20 cM. These core markers serve as a kind of "reference marker" for comparison across different mapping studies.

^u a = Additive effect and d = dominance effect of the QTL. A positive sign of additive effects reflects that QTL alleles increasing resistance were contributed by parent D32. R^2 estimates the proportion of the phenotypic variance (%) explained by the detected QTL. $a \times E$ and $d \times E$ = Interaction between QTL and environment for the a and d effect, respectively.

^v All values are percentages.

^w EWE96 = Eckartsweier 1996, HOH97 = Hohenheim 1997, and Comb = combined analyses.

^x *, ** = Significant at the 0.05 and 0.01 probability level, respectively; ns = not significant ($P > 0.05$).

^y Estimates of total R^2 obtained by a simultaneous fit of all putative QTL affecting the respective trait using a model with additive and dominance effects.

^z Estimates of total R^2 obtained by a simultaneous fit of all putative QTL affecting the respective trait using a model including additive, dominance, and epistatic effects.

to VIR7. Another large QTL on chromosome 3 was identified from the second to seventh scoring (VIR2 to VIR7) with increasing R^2 values; it accounted for 28% of $\hat{\sigma}_p^2$ at VIR7. These two large QTL were detected consistently in both environments (Table 3). One minor QTL on chromosome 1 was detected on all SCMV scoring dates in the joint analyses and explained between 7 and 10% of $\hat{\sigma}_p^2$ at various stages of plant development. Another minor QTL on chromosome 5 was detected from the fifth to seventh scoring and explained 6 to 8% of $\hat{\sigma}_p^2$. One minor QTL on chromosome 10 was identified at the first scoring (VIR1) and explained 8% of $\hat{\sigma}_p^2$. The three minor QTL were only detected in one environment on some of the SCMV scoring dates. A simultaneous fit with all QTL, using a model with additive and dominance effects, explained between 62 and 76% of $\hat{\sigma}_p^2$ in the joint analyses. Extending this model for digenic epistatic effects increased the R^2 values only marginally, except for VIR1. Significant digenic epistatic effects were found between the two major QTL on chromosomes 6S and 3 for VIR2, VIR5, and VIR7 and between the major QTL on chromosome 6S and the two minor QTL on chromosomes 1 and 10 (Table 3). The major QTL on chromosomes 6S and 3 showed significant additive and dominance gene effects, whereas the two QTL on chromosomes 5 and 10 displayed only additive gene effects. The minor QTL on chromosome 1 displayed overdominance. Significant ($P < 0.05$) QTL $\times$ E interactions were found for four QTL on certain dates of SCMV scores (Table 3).

DISCUSSION

Genetic foundation of resistance to SCMV. A previous study of SCMV resistance in a F_2 population of the cross D145 $\times$ D32 gave evidence for two major resistance genes (*Scm1* and *Scm2*) on chromosomes 6S and 3 (40). However, the exact position of both resistance genes and their gene action could not be determined because of the limited population size and progeny type. Using 219 F_3 families in this study, two QTL with major effects were identified on chromosomes 6S and 3 adjacent to markers *phi075* and *umc102*, respectively (Table 3 and Fig. 1), confirming the presence of *Scm1* and *Scm2* in the resistant parent D32. *Scm1* explained the largest proportion of $\hat{\sigma}_p^2$ for all scoring dates and displayed partial dominance for resistance to SCMV based on the estimates of the additive and dominance effect of the QTL (Table 3). The second QTL with major effect (*Scm2*) was not detected at the first scoring, but was of increasing importance from VIR2 to VIR7 as reflected by the R^2 values (Table 3). *Scm2* also showed partial dominance for resistance to SCMV and explained a smaller proportion of $\hat{\sigma}_p^2$ than did *Scm1* at all scorings (Table 3).

In agreement with our study, several reports on resistance to SCMV, MDMV, and WSMV indicated an oligogenic type of inheritance (6,36,63). However, in most cases, the gene action of the resistance genes was dominant (6,24,36,48), whereas a partially dominant or additive mode of gene action of resistance genes against potyviruses in maize was rarely reported (23,63). Louie et al. (24, 25) discussed that the degree of resistance conferred by *Mdm1* was dependent on the susceptible parent and resistance to MDMV could be strongly influenced by environments. Oligogenic inheritance of virus resistance with partial dominance of resistance genes was also reported for another virus (MSV) in maize (61) and for potyviruses in other crops (4).

The resistance controlled by *Scm2* was influenced by plant age with an elevated resistance to SCMV at the adult stage. This kind of resistance to potyviruses was also described previously (42,53). Mikel et al. (42) reported that three genes were required for MDMV resistance at the adult stage in maize inbred B68, whereas only two resistance genes were sufficient at early ratings. Kegler and Friedt (18) concluded that resistance to potyvirus X in potato at the adult stage was related to a reduced content of ribosome and RNA in the leaves.

In addition to major genes, presence of minor genes conferring resistance to SCMV was conjectured for the three maize inbreds

D21, D32, and FAP1360A (40). In support of this hypothesis, we detected QTL on chromosomes 1, 5, and 10 that explained between 6 and 10% of $\hat{\sigma}_p^2$ (Table 3). Minor genes were also identified for resistance to MDMV (16,17). Caranta et al. (4) detected several minor QTL associated with resistance to various potyviruses in pepper, and Welz et al. (61) found three QTL with minor effects on resistance to MSV in maize. Kegler and Friedt (18) discussed the effects of minor genes for MDMV resistance that could reduce the rate of infection and virus concentration, prolong the incubation time, and inhibit the movement of viruses in the plant.

In the current study, 34 out of the 94 markers displayed a dominant (3:1) inheritance pattern. The influence of the large number of dominant markers on the results from QTL analyses was evaluated. Three out of the five QTL (on chromosomes 1, 6S, and 10) detected for SCMV resistance were flanked by codominant markers, whereas the other two QTL (on chromosomes 3 and 5) were flanked by one codominant and one dominant marker, respectively. When we deleted the flanking dominant markers, the estimates of additive and dominance effects of the two QTL on chromosomes 3 and 5 were hardly affected and there was no change for the other three QTL on chromosomes 1, 6S, and 10. Hence, the large number of dominant markers did not influence the estimated gene effects of the five detected QTL for resistance to SCMV. However, owing to the reduced power in QTL detection when using dominant instead of codominant markers, some minor QTL might not have been detected in our mapping population.

Epistatic effects. Classical genetic studies have uncovered various examples of epistatic effects on disease resistance (64). With the aid of QTL mapping, epistatic interactions can be related to individual resistance genes. Caranta et al. (5) found a digenic epistasis between two QTL influencing resistance to cucumber mosaic virus in pepper. Van Koeveering et al. (59) obtained evidence for aa epistatic effects between loci A and B for resistance to wheat spindle streak mosaic virus in wheat. In the current study, significant aa and ad epistatic effects were estimated for VIR7 between the two major QTL (Table 3), but there was only a minor increase in the R^2 values of this model. This apparent discrepancy can be explained by the high correlation between the coefficients of epistatic and additive or dominance effects in the regression model employed in the QTL analysis. Epistatic effects may arise from interrelated biochemical pathways or dependent physiological and morphological characters, which was confirmed by a detailed study of trichome-mediated insect resistance in potato (3).

Clustering of resistance genes in maize. Resistance genes against different pathogens are often clustered in the same chromosome regions of the maize genome (38). The resistance genes *Mdm1* (MDMV) (36), *Wsm1* (WSMV) (37), and *rhm1* (southern corn leaf blight) (65) were located on the short arm of chromosome 6 (6S). Recently, a resistance gene (*HPV*) against high plains virus and *Scm1* against SCMV were also mapped to this region (33,40). Presence of a major resistance gene against SCMV at the position of *Scm1* on chromosome 6S in the cross FAP1360A $\times$ F7 was corroborated by Xu et al. (63). It is unknown whether *Scm1*, *Mdm1*, and *Wsm1* are identical and display pleiotropic effects against different potyviruses or are closely linked genes. The U.S. inbred Pa405 as well as the three resistant European lines D21, D32, and FAP1360A conferred resistance against different maize pathogenic potyviruses (21). Furthermore, tests of allelism between Pa405 and D21, D32, or FAP1360A supported the hypothesis that *Scm1* and *Mdm1* or *Wsm1* are identical (40). However, the small sample size (200 plants for each F_2 population) was not sufficient to detect rare recombination events between closely linked loci resulting in susceptible plants. In agreement with the hypothesis of closely linked but different major resistance genes, Lübberstedt et al. (32) found a few susceptible plants in tests of allelism between the three European resistant inbreds. Furthermore, Xu et al. (63) identified three recombinants between the *nor* and the *Scm1* gene in a sample of 40 resistant BC5 individuals from the cross

FAP1360A × F7, whereas no recombinant was observed between *nor* and *Mdm1* in 7,650 F₂ individuals of the cross *pol1 yl* tester × Pa405 (54). Close linkage rather than pleiotropy of resistance genes has been frequently demonstrated including resistance gene clusters against common rust in maize (13), downy mildew in lettuce (14), and bacterial blight (*Pseudomonas syringae* pv.) in tomato (34,50). Linkage rather than pleiotropy also seems more likely for resistance genes acting against different pests such as SCMV and southern corn leaf blight.

The second major resistance gene in this study, *Scm2*, mapped closely to the centromere region of chromosome 3 (Fig. 1 and Table 3), harboring the major resistance genes *Rp3* (*Puccinia sorghi*) (51), *Mv1* (MMV) (43), *Wsm2* (WSMV) (37), and three QTL for resistance to European corn borer, Fusarium stalk rot (38), and common smut (28). *Wsm2* is different from *Mv1* because Pa405 is susceptible to MMV (43). However, it is unclear whether *Scm2* is identical to *Wsm2* or *Mv1*. Alternatively, the chromosome regions harboring *Scm2* and *Wsm2* (chromosome 3) as well as *Scm1*, *Mdm1*, and *Wsm1* (chromosome 6) might contain clusters of potyvirus resistance genes.

One QTL with minor effects on SCMV resistance clustered with *Wsm3* (WSMV) (37) located on chromosome 10. Hence, reverting to Robertson's (49) hypothesis about the relationship between major genes and QTL, the QTL for SCMV resistance identified in this study might indicate the potential presence of additional major *Scm* resistance genes in the maize germ plasm. Another QTL with minor effects was mapped on chromosome 5, clustering with one QTL for resistance against northern corn leaf blight (38) and one for common smut (28). The QTL on chromosome 1 mapped to the same position as a QTL for resistance to common smut (31). To resolve the relationship among clustered resistance genes, cloning of these genes is required.

Environmental influence on resistance and QTL × E interactions. Resistance against potyviruses in maize is strongly influenced by the environment. In maize inbreds tested for MDMV resistance, Louie et al. (25) found a considerably higher disease incidence in the greenhouse than in the field and hypothesized that the tender greenhouse plants are more easily infected after virus inoculation. In a study of SCMV resistance, a higher proportion of susceptible plants was also identified under greenhouse conditions than in field tests (40). Tu and Ford (57) found an increasing susceptibility of maize to MDMV with increasing temperature. These results suggested that resistance to potyviruses in maize might be a "threshold" character.

Although the two environments hardly differed in the F₃ population mean of infected plants in our study, estimates of $\hat{\sigma}_{ge}^2$ were highly significant ($P < 0.01$) for all dates of virus scoring and QTL × E interactions were highly significant ($P < 0.01$) for *Scm1* at VIR2, VIR5, and VIR7 (Table 3). QTL analyses showed that the additive or dominance effect values of two large QTL differed in two environments at certain SCMV scores and the three minor QTL were not expressed consistently across two environments (Table 3). These findings are in contrast to other QTL mapping studies of virus resistance (4,5,43,61), in which QTL × E interactions have not been detected. Possible explanations for these contrasting findings are (i) the different pathosystems investigated, (ii) the type of mapping populations evaluated (F₃ families versus F₂ plants or double-haploid lines), and above all, (iii) the biometric procedures and software employed in QTL mapping (39).

Associations between SCMV resistance and agronomic traits. Biotic and abiotic stresses can severely affect the development of the maize plant and cause dramatic physiological and phenotypic changes. In studies of drought stress on maize (2,47,62), PHT decreased under drought conditions. In this study, the reduction in PHT of infected plants was largely explained by one common QTL on chromosome 6S between PHT and VIR7 (Table 2) and one closely linked QTL on chromosome 3 (X. C. Xia, unpublished data). Significant phenotypic correlations were also found between VIR7

and DTA with one common QTL (Table 2). However, for all QTL in common between agronomic traits and SCMV resistance, it remains to be shown whether they are due to pleiotropy or close linkage.

Marker-assisted selection (MAS). Maize lines resistant against SCMV can be efficiently identified with conventional phenotypic selection because artificial inoculation and visual rating of disease symptoms are fairly rapid and highly reproducible. Nevertheless, MAS for improving SCMV resistance might be advantageous because of (i) the oligogenic inheritance of the target trait, (ii) the strong influence of the environment on the expression of SCMV resistance, and (iii) the high costs for resistance evaluation.

In our study, none of the 219 F₃ families was completely resistant across both years, corroborating the oligogenic inheritance of SCMV resistance in the cross D32 × D145. MAS could be used to incorporate all resistance genes, especially also the QTL with minor effects, into one or both maize inbreds to ascertain almost complete resistance of the resulting hybrid. However, a large population size and highly efficient marker system such as multiplexed SSRs would be required in a breeding program, given that the low frequency (0.25⁵) of an individual homozygous for the desired alleles at five unlinked loci in a F₂ population. Since yield reductions are mainly caused by early SCMV or MDMV infection, protection against these potyviruses is required especially at early stages of plant development. Consequently, the SCMV resistance genes located on chromosomes 1, 6, and 10, which are active at VIR1, might be most desirable for MAS. The resistance genes on chromosomes 1 and 6 especially seem to be rather useful for hybrid breeding given their overdominant (chromosome 1) or dominant (chromosome 6) gene action at VIR1 and VIR2 and their large gene effects compared with the resistance gene on chromosome 10. *Scm2* should be useful for suppression of SCMV infection at later stages of plant development.

In conclusion, QTL mapping and MAS might aid breeders to (i) identify QTL with large gene effects consistently expressed across environments; (ii) detect QTL alleles with dominant gene action, simplifying hybrid breeding; and (iii) pyramid these genes into one or both parents of a hybrid. Since MAS requires neither natural infection nor artificial inoculation with SCMV, it allows indirect selection in offseason nurseries before flowering and, hence, could substantially accelerate breeding programs for resistance to SCMV and other diseases in maize.

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