Mapping QTL contributing to SCMV resistance in tropical maize

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Souza, I. R. P. de, Schuelter, A. R., Guimarães, C. T., Schuster, I., Oliveira, E. de, and Redinbaugh, M. 2008. Clustering of QTL conferring SCMV resistance in tropical maize. — *Hereditas 145*: 167–173. Lund, Sweden. eISSN 1601-5223. Received April 25, 2007. Accepted June 12, 2008

Sugarcane mosaic virus (SCMV) has been increasing in importance as a maize disease in Brazil. In this study, we mapped and characterized quantitative trait loci (QTL) associated with resistance to SCMV in a maize population consisting of 150 $F_{2:3}$ families from the cross between two tropical maize inbred lines, L520 (resistant) and L19 (susceptible). F_2 individuals were genotyped with microsatellite (SSR) markers, and the derived $F_{2:3}$ families were evaluated for their response to artificial inoculation with SCMV under field conditions at Sete Lagoas, MG, Brazil, in 2001 and 2005. Multiple interval mapping was used for QTL detection with a linkage map based on 19 SSR markers. Three QTLs for SCMV resistance were identified with two QTLs (*Scm2a* and *Scm2b*) clustered on chromosome 3, bin 3.04, and one QTL (*Scm1*) on chromosome 6, bin 6.01, explaining 13.34, 41.85 and 7.66% of the phenotypic variation for SCMV resistance, respectively.

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In Brazil, maize mosaic disease, caused by the Potyvirus species Sugarcane mosaic virus (SCMV) has increased in economic importance in the past years. MELO (2000) concluded that maize mosaic in Brazil is caused by SCMV comparing nucleotide and amino acid sequences of the coat protein gene. This virus is transmitted in a non-persistent manner by several aphid species, mainly by *Rhopalosiphum maidis* (SHUKLA et al. 1994), also in Brazil, and it can reduce grain production by up to 50% (WAQUIL et al. 1996). The utilization of maize hybrids with resistance to SCMV is the most economical and efficient method to control this virus disease.

Studies on maize resistance to MDMV (Maize dwarf mosaic virus) and to SCMV have been conducted mainly in United States (FINDLEY et al. 1973; MCMULLEN and LOUIE 1989; ROANE et al. 1989; SIMCOX et al. 1995) and in Germany (XIA et al. 1999; YUAN et al. 2003) with temperate germplasm. MDMV resistance has been frequently reported as controlled by a single gene, while examples of oligogenic virus resistance are scarce in addition of being difficult to distinguish from monogenic resistance affected by genotype-specific modifiers, genotype-environment interactions (FRASER 1986, 1987), and different symptom classification systems (MCMULLEN and LOUIE 1989). Initial symptom response to MDMV infection was controlled by a single major dominant gene, *Mdm1* that mapped to chromosome 6 in Pa405 (MCMULLEN and LOUIE 1989; SIMCOX et al. 1995). Recently, resistance to MDMV was associated with chromosome 6 in 42 of 43 tropical and Corn belt inbred lines (JONES et al. 2007).

In field trials, resistance to SCMV in the European lines, D21, D32 and FAP1360A was associated with one to three genes depending on the resistance source (MELCHINGER et al. 1998). Linkage mapping and bulk segregant analysis identified two major genes, *Scmv1*, on the short arm of chromosome 6 and *Scmv2* near the centromere of chromosome 3 that controlled resistance to SCMV in maize (MELCHINGER et al. 1998; XIA et al. 1999; XU et al. 1999; DUSSLE et al. 2000). The presence of both genes was necessary for full resistance to SCMV. *Scmv1* suppressed symptom expression throughout development at high level, whereas *Scmv2* was expressed at later stages of infection and development (XIA et al. 1999; DUSSLE et al. 2000). DUSSLE et al. (2000), using a total of 121 F_3 lines from the cross F7 (susceptible) × FAP1360A (resistant), confirmed that these two QTL were responsible for SCMV resistance and explained together from 15% (first score) to 62% (final score) of the phenotypic variance for the virus resistance at various stages of the plant development. Gene action was additive for the *Scmv2* region but completely dominant for the *Scmv1* region, and significant

DOI: 10.1111/j.2008.0018-0661.02006.x

epistatic effects were identified between these QTL in the population D145 × D32 (Xia et al. 1999). Besides chromosomes 3 and 6, Xia et al. (1999) and Li and Zhang (2004) identified additional resistance QTL on chromosome 1, 5 and 10.

Identification and mapping of genes or quantitative trait loci (QTL) for virus resistance provides information on the number of genes or regions that must be transferred by breeding programs and aids identification of sources for resistance. Also, virus resistance must first be mapped to specific regions of the maize genome to allow the use of marker-assisted selection.

In the present work, we investigated the nature and chromosomal locations of loci for resistance to SCMV in the Brazilian maize population composed of 150 $F_{2:3}$ families derived from the cross L520 (resistant) × L19 (susceptible). The objectives were to (i) estimate the number and chromosomal position of QTLs conferring resistance to SCMV, (ii) determine the gene action, and (iii) identify molecular markers flanking these QTLs.

MATERIAL AND METHODS

Plant material

Two tropical maize inbred lines, contrasting in their resistance to SCMV, L520 (resistant) and L19 (susceptible), were crossed and the F_1 generation was subsequently selfed to produce a set of 150 F_2 individuals. The F_2 individuals were tagged at the field level and were subsequently selfed to produce the $F_{2:3}$ families. These inbred lines were developed by researchers of the breeding program at Embrapa Maize and Sorghum, Brazil. L520 and L19 were derived from the BR105 and BR106 populations, respectively. BR105 was obtained after 8 cycles of recurrent selection in the Swan-*Dmr* (Thailand) and BR106 is a composite obtained from the recombination of four Tuxpeño open pollinated varieties with 13 cycles of recurrent selection in Brazil (Parentoni et al. 2001).

DNA extraction

Genomic DNA was extracted from leaves of each V_8 (eight leaves) physiological stage F_2 individual. Harvested leaves were frozen in liquid nitrogen and stored at -80^{o}C. Five g of leaves were ground to a fine powder in liquid N_2 and DNA was extracted according to Saghai-maroof et al. (1984). DNA was quantified using the spectrophotometric readings at A_{260} and A_{280}, and concentration was calculated according to Sambrook et al. (1989).

Simple sequence repeat (SSR) analyses

SSR markers sequences were obtained from the Maize Genetic and Genomic Database (www.maizegdb.org/ssr.php) and synthesized by WMed (São Paulo, Brazil). Primers that were polymorphic in the parental inbred lines L520 and L19, and for which the heterozygote could be identified in F_1 plants were chosen as markers, and used to genotype the F_2 individuals. Polymerase chain reaction (PCR) was carried out according to Ninamango-Cárdenas et al. (2003) and amplified fragments were electrophoretically separated on 4% agarose (Invitrogen, Carlsbad, CA, USA) gels in Tris-borate buffer (Sambrook et al. 1989).

Phenotypic evaluation

For the phenotypic evaluation, a randomized block experimental design was used with two replications in 2001 and one replication in 2005. Each $F_{2:3}$ family was represented in each block by one 4 m row. Rows were 0.8 m apart and overplanted and later thinned to 20 plants per row.

Inoculum was prepared by macerating leaves from 30 day-old susceptible maize cultivars, previously inoculated with SCMV and expressing strong symptoms, in 10 mM phosphate buffer at pH 7.0 at a ratio of 1:5 (w/v). Plants were inoculated at the three-leaf stage by gently rubbing the two youngest leaves with ca 1 ml of inoculum and 600 mesh Carborundum. Leaves were subsequently rinsed with sterile water (Almeida et al. 2000). The plants were inoculated two times, with a one week interval. Plants were evaluated for the presence (susceptible) or absence (resistant) of mosaic symptoms 30 days after inoculation. A resistance score was calculated for each family: resistance score = (number of resistant plants/total number of plants in the row) × 100. The mean resistance score of $F_{2:3}$ families across three replicates in two years was used for QTL mapping.

Linkage mapping

Markers were scored as 2 (homozygous for the resistant parent allele), 0 (homozygous for the susceptible parent allele) and 1 (heterozygous). Segregation of each locus was checked for deviation from a Mendelian segregation ratio (1:2:1) using a χ^2 test for goodness of fit ($p < 0.05$). Linkage analysis of SSR markers was conducted using the Kosambi (1944) mapping function with a minimum $\log_{10}$ odds ratio (LOD) of 3.0 and maximum recombination frequency of 0.4 performed by MapMaker/EXP 3.0 (Lander et al. 1987).

Statistical analyses

For the phenotypic evaluation, a randomized block experimental design was used with three replications,

two in 2001 and one in 2005. ANOVA, heritability based on family means (h^2_m), coefficients of genetic and experimental variation and correlation among replicates were estimated using Genes software (Cruz 2001).

QTL analyses

Single and multiple regression analyses were carried out using the software Jump ver. 3.1.6.2 (Sas Inst. Inc. 1995) and associations between markers and resistance were considered with a significant if the coefficient of determination (R^2). QTL mapping was performed using multiple interval mapping (MIM) (Kao et al. 1999) with QTL Cartographer 2.5 for Windows (Wang et al. 2006). Each independent likelihood ratio (LR) peak indicated the presence of one QTL using the significant threshold calculated by Bayesian information criteria (BIC-M0). Multiple interval mapping was used to estimate the percentage of the phenotypic variance explained by individual and by all QTL and their effects.

RESULTS

Phenotypic data

Variance analyses detected a significant genetic variability of SCMV resistance in the $F_{2:3}$ maize population evaluated in two years, showing high heritability value, 86.78, based on family means, and a good correlation among block, 68.79%. These data indicate that the resistance to SCMV score in these families was consistent between years. Frequency distribution of resistance to SCMV scores of the $F_{2:3}$ families followed the normal distribution according to Lilliefors test at 5% of significance (Fig. 1).

Segregation and linkage of SSR markers

Nineteen codominant SSR markers showed Mendelian segregation (1:2:1) and were used in the linkage map analysis, 11 that map to chromosome 3, and eight to chromosome 6 (Fig. 2). Genotypic analysis of the 150 F_2 individuals produced two linkage groups spanning 137.18 cM with an average distance between markers of 7.6 cM. Marker order was similar to the IBM2 2004 neighbor genetic map (http://www.maizegdb.org/map.php), except that phi374118 and bnlg1350, which mapped to bin 3.02 and 3.08, respectively, in the IBM2 2004 map, mapped to bin 3.04 and bins 3.06/3.07.

Mapping SCMV resistance QTLs in tropical maize

Multiple regression analysis identified three SSRs markers, umc1030, umc2002 and umc1018, associated with resistance to SCMV explaining 55.79% of the phenotypic variation (Table 1). These results are in agreement with multiple interval mapping (Fig. 2), which detected two QTLs on chromosome 3, and one QTL on chromosome 6.

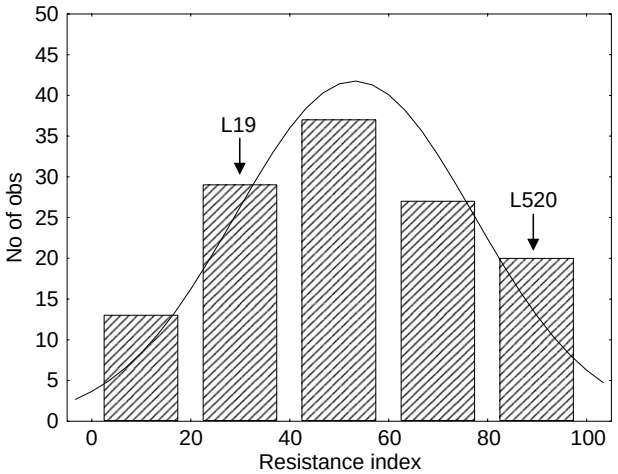

Fig. 1. Frequency distribution of the resistance to SCMV in a maize $F_{2:3}$ population. Solid line indicates the expected normal curve and the arrows indicate the resistance score of the parental lines.

On chromosome 3 the two QTLs were flanked by the markers nc030 and umc2002, and the QTL on chromosome 6, flanked by umc1018 (Table 2). *Scm2a* (bin3.04), *Scm2b* (bin 3.04) and *Scm1* (bin 6.01) explained 13.34, 41.85 and 7.66% of the phenotypic variation for resistance to SCMV. *Scm2a* and *Scm1* showed over-dominant gene effect and *Scm2b* displayed an additive effect.

The QTLs on chromosome 3 (*Scm2a* and *Scm2b*) were derived from the resistant maize inbred line; while the QTL on chromosome 6 (*Scm1*) was derived from the susceptible parental line. The QTL derived from the susceptible parent was not associated with the resistance by single regression analysis but it was detected by multiple regression model (Table 1) and multiple interval mapping (Table 2). This result was confirmed by further linear regression analysis showing that *Scm1* was significantly ($p < 0.05$) associated with resistance only in the presence of *Scm2a* or *Scm2b* (Table 3), indicating the occurrence of epitasis.

DISCUSSION

Clusters of maize virus resistance QTLs are located on chromosomes 3, 6, and 10. The cluster on chromosome 6 (bin 6.01) confers resistance to three members of the Potyviridae (MDMV, SCMV and WSMV). Loci on chromosome 3 (bin 3.05) and 10 (bin 10.05) carry the resistance to phylogenetically diverse viruses as well as to bacterial and fungal pathogens (Redinbaugh et al. 2004). Resistance genes that

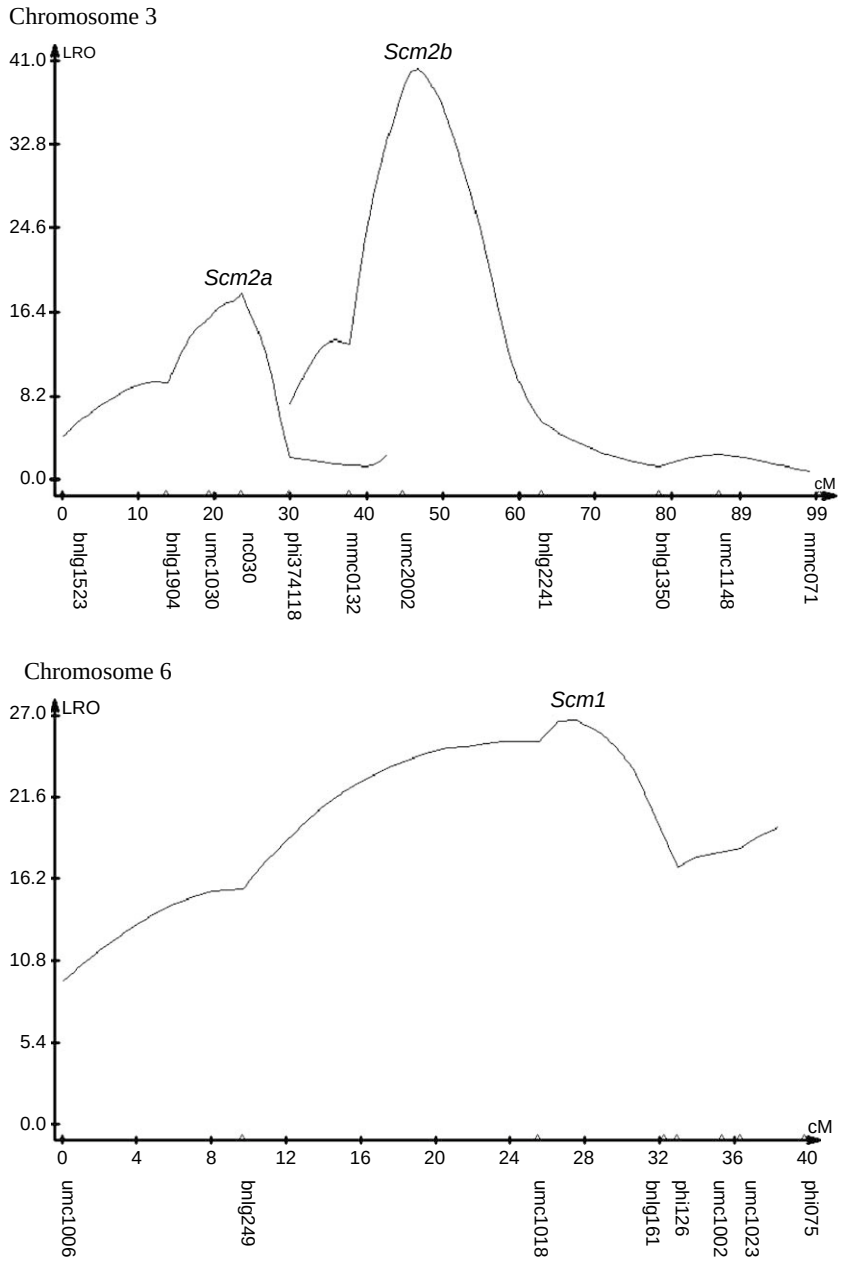

Fig. 2. Quantitative trait loci for SCMV resistance on maize chromosomes 3 and 6. Each independent LR peak indicates the presence of one QTL significant using Bayesian information criteria BIC – M0 according to QTL Cartographer 2.5. Map distances are given in centiMorgans (cM).

were also localized to the centromeric region of maize chromosome 3 include: *mv1*, a QTL for resistance to Maize mosaic virus (MMV) (Ming et al. 1997); *Wsm2* a dominant gene for resistance to Wheat streak mosaic virus (WSMV) (McMullen et al. 1994); *mcd1*, a QTL for resistance to Maize chlorotic dwarf virus (Jones et al. 2004); and, *rp3*, a gene for resistance to *Puccinia sorghi* (Sanz-Alferez et al. 1995). These studies support the notion that there is a cluster of pathogen resistance genes in this region (McMullen and Simcox 1995).

In our study, two clustered QTLs (*Scm2a* and *Scm2b*) were identified on chromosome 3 (bin 3.04) and one QTL on chromosome 6 (*Scm1*) (bin 6.01) (Table 2, Fig. 2). Similar results for SCMV resistance were found by Li and Zhang (2004), using $F_{2:3}$ families derived from the cross Huangzao × Ye 107. The authors mapped one major QTL on chromosome 3 (bin 3.04) showing significant additive effects. They also identified one minor QTL on chromosome 6 (bin 6.01). Furthermore, two major QTLs, *Scmv1* and *Scmv2,* conferring resistance to SCMV were mapped

Table 1. *SSR markers associated with SCMV resistance detected by multiple regression marker analysis at $p < 0.001$.*

Locus	Bin[1]	R^2 adjusted (%)[2]	Effect ± SD	F ratio	Prob > F[3]
umc1030	3.04	37.03	22.65 ± 2.62	74.51	<0.0001
umc2002	3.04	42.44	23.56 ± 2.44	93.18	<0.0001
umc1018	6.01	4.31	−7.65 ± 2.97	6.64	0.0112
R_t^2 adjusted (%)[4]		55.79			

[1]chromosomal position of the marker; [2]the coefficient of determination detected by single regression analysis; [3]association probability detected by single regression analysis; [4]percentage of the total phenotypic variation explained by all markers detected by multiple regression analysis.

to maize chromosome arms 6S and 3L, respectively, in the cross D145 × D32 (Xia et al. 1999) and in the cross F7 × FAP1360A using bulk segregant analysis (Xu et al. 1999) and by QTL mapping (Dussle et al. 2000). Dussle et al. (2003) using 131 SSRs mapped *Scmv2* and *Scmv1* to the regions 3.04–05 and 6.00–01, respectively. These results confirm the oligogenic inheritance of SCMV resistance in different crosses between temperate maize inbred lines.

For full resistance to SCMV, the presence of both *Scmv1* and *Scmv2* QTLs was essential, with *Scmv1* suppressing the expression of symptoms throughout all developmental stages at high level, whereas *Scmv2* was expressed at later stages of infection (Xia et al. 1999; Dussle et al. 2000). These two QTLs together explained among 15% to 62% of the phenotypic variance, depending on stage of plant development, being more informative at later phenotypic evaluation. Using RFLP and SSR markers, *Scmv1* was mapped on the short arm of chromosome 6, between the nucleolus organizer region (*nor*) and the RFLP marker bnlg6.29, while *Scmv2* was mapped to an interval of 26.8 cM flanked by the RFLP markers umc92 and umc102 near the centromeric region of chromosome 3 (Xu et al. 1999). Melchinger et al. (1998) also mapped *Scmv2* at the same region on chromosome 3 by using one SSR and four RFLP markers. In our study (Fig. 2, Table 2), the QTL *Scm1* was flanked by the marker umc1018 at chromosome 6, which is located 12.50 cM from phi075. Xia et al. (1999), using 219 F_3 families, identified one QTL with major effect (*Scmv1*) adjacent to marker phi075. Also on the short arm of chromosome 6 was mapped one major Maize dwarf mosaic virus (MDMV) resistance gene, *Mdm1*, to the 1-cM interval between the RFLP markers umc85 and bnlg6.29a (McMullen and Louie 1989; Simcox et al. 1995). Close linkage between *Scmv1* and *Mdm1* is supported by the finding of Xu et al. (1999) that found three recombinants between the *nor* and *Scmv1* gene among 40 BC_5R individuals from the cross FAP1360A × F7. However, no recombinant was observed between *nor* and *Mdm1* in 7650 plants of *cros pol y1* tester × Pa405 (Simcox et al. 1995).

In the current study, significant epistatic effect were found among the major QTLs (*Scm2a, Scm2b*) on chromosome 3 and the minor QTL (*Scm1*) on chromosome 6 derived from the susceptible parent (Table 3). Also, epistatic effects were identified between the major QTL *Scm1* and *Scm2* in population D145 × D32 (Xia et al (1999). Xu et al. (1999) suggested that *Scm1* is not sufficient for resistance without additional resistance genes. Quint et al. (2002) suggested in a comparison of the recombination frequencies of cross F7 × FAP1360A and D21 × D408 that further resistance genes are required for complete resistance to SCMV.

Table 2. *Sugarcane mosaic virus resistance QTLs in tropical maize population detected by multiple interval mapping.*

QTL	Bin[1]	Position (cM)[2]	Flanking marker	LR[3]	R^2 (%)[4]	Gene effects[5]			Gene action
						d	a	[d/a]	
Scm2a	3.04	23.5	nc030	16.1	13.34	−10.78	8.35	1.3	OD
Scm2b	3.04	46.6	umc2002	38.3	41.85	3.34	21.43	0.2	A
Scm1	6.01	27.5	umc1018	24.5	7.66	−10.69	−1.98	5.4	OD
Total					66.20				

[1]chromosomal position of the marker; [2]distance from the beginning of the linkage group, in cM; [3]LR is the likelihood of ratio (ratio between the likelihood considering a QTL in the interval, and a likelihood considering no QTL in the interval); [4]R^2 is the proportion of the phenotypic variation explained by each QTL; [5]a is the estimative of additive effects, d is the estimative of dominant effects and [d/a] is the degree of dominance.

Table 3. *Interactions among SCMV resistance QTLs on maize chromosomes 3 and 6 evaluated by single regression analysis. Presence of Scm2a and Scm2b (3.04) indicates that QTLs were derived from the resistant line and presence of Scm1 (6.01) indicates the origin from the susceptible line.*

Presence of QTLs	Regression of *Scm1*		Absence of QTLs	Regression of *Scm1*	
	R^2	prob		R^2	prob
Scm2a	0.2175	0.019	*Scm2a*	0.0104	0.621
Scm2b	0.1455	0.044	*Scm2b*	0.0361	0.333
	Regression of *Scm2a*			Regression of *Scm2a*	
Scm1	0.47107	<0.0001	*Scm1*	0.1484	0.036
Scm1	Regression of *Scm2b*		*Scm1*	Regression of *Scm2b*	
	0.446488	<0.0001		0.2097	0.011

The gene action of the two major QTLs (*Scm2a* and *Scm2b*) on chromosome 3, was additive (*Scm2b*) and overdominant (*Scm2a*), and the gene action of the minor QTL *Scm1* on chromosome 6 were also overdominant. Additive gene action was also detected in the *Scmv2* identified by Dussle et al. (2000) on chromosome 3, explaining 15% of the phenotypic variation. Larger additive gene effect of *Scmv1* than that of *Scmv2* was detected in the F_3 lines from the cross D32 × D145 (Xia et al. 1999). The effect of the *Scm2* region was much smaller than that for the *Scm1* region in both populations D32 × D145 (Xia et al. 1999) and F7 × FAP1360A. Xu et al. (2000) suggested the presence of different resistant loci in the *Scm2* region of the three European inbred lines FAP1360A, D21 and D32. A clustering of resistance genes in the *Scm2* region has also been reported by Xu et al. (2000).

Yuan et al. (2003) using a different maize population consisting of 121 F_3 lines from cross F7 (susceptible) × FAP1360 (resistant), found that the *Scmv1* region harbored two QTLs rather than one as identified in previous studies. Dussle et al. (2002) mentioned that in different studies a reason for the lack of resistance-allele-specific co-segregating markers could be the presence of more than one SCMV resistance gene in the *Scmv1* region. However, we found clustered QTLs (*Scm2a* and *Scm2b*) for SCMV resistance at chromosome 3 (bin 3.04).

Our results showed that the two regions, on chromosome 3 and 6 explained together 66.20% of the phenotypic variance, and chromosome 3 harbors a cluster of linked genes and not allelic variation for one single resistance gene. It is suggested that different genetic systems control resistance to the various virus strains of the SCMV complex; however they may have some genes in common. Studies with populations originated from the cross with other resistant inbred lines are currently under way, and also the development and testing by near isogenic lines (NILs) carrying these regions will be important to verify the QTLs.

Acknowledgements – The current work was supported by grants from FAPEMIG (Fundação de Amparo à Pesquisa do Estado de Minas Gerais) and Embrapa (Brazilian Agricultural Research Corporation). We thank Célio Ramos das Neves for help conducting field trials.

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