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Origin of *Scm1* and *Scm2* – two loci conferring resistance to sugarcane mosaic virus (SCMV) in maize

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Abstract Sugarcane mosaic virus (SCMV) causes serious losses of grain and forage yield of maize (*Zea mays* L.) in Europe. Two dominant genes, *Scm1* and *Scm2*, have been identified to confer resistance to SCMV. *Scm1* is located on the short arm of chromosome 6 and *Scm2* near the centromere region of chromosome 3. In the present study, resistant, partially resistant, and susceptible maize inbred lines, together with their ancestral lines, were evaluated with molecular markers to trace back the origin of *Scm1* and *Scm2*. The banding patterns indicated that the *Scm1* region, originally identified in resistant European line FAP1360A, was derived from its ancestral line FAP954A. The other two resistant European lines, D21 and D32, most likely carry the same *Scm1* region, which originated from their common ancestral line A632. This *Scm1* region was also present in three partially resistant lines, D09, FAP1396A and FAP693A, but not in the resistant U.S. inbred Pa405. Apart from FAP954A and A632, none of the remaining ancestral lines and none of the susceptible lines harbored the *Scm1* region. The *Scm2* region present in FAP1360A was obviously transmitted from its ancestral line Co125. However, the presence of the respective *Scm2* region was not confirmed in the other three resistant lines (D21, D32 and Pa405), the remaining ancestral lines, and all partially resistant lines by using closely linked markers.

Key words *Zea mays* L · Maize · Sugarcane mosaic virus · SCMV · *Scm1* · *Scm2* · AFLP · RFLP · SSR · Pedigree relationship

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Introduction

Sugarcane mosaic virus (SCMV) and maize dwarf mosaic virus (MDMV) are two important pathogens of maize and related crops, causing yield losses, chlorosis and stunting (Shukla 1989; Fuchs and Grüntzig 1995). The most intensively studied inbred line, Pa405, is resistant to both viruses in field and greenhouse tests (Louie et al. 1991). Segregation analyses suggested that 1–5 genes were involved in the resistance of Pa405 to both viruses. The major MDMV resistance gene, *Mdm1*, was mapped to Chromosome 6 (McMullen et al. 1989; Louie et al. 1991; Simcox et al. 1995).

Kuntze et al. (1997) surveyed 122 early maturing European maize inbred lines for resistance to SCMV under both field and greenhouse conditions. Three lines (D21, D32 and FAP1360A) were completely resistant to SCMV, four lines (D06, D09, R2306 and FAP1396A) showed delayed expression of SCMV symptoms, while the majority of lines were highly susceptible to SCMV. Segregation analyses suggested 1–3 genes conferring resistance to SCMV in different crosses (Melchinger et al. 1998). By employing RFLP and SSR markers, these authors mapped two resistance genes, *Scm1* and *Scm2*, to the short arm of chromosome 6 and near the centromere region of chromosome 3, respectively. AFLP-based high-resolution mapping restricted the *Scm1* region to an interval of 5.8 cM between AFLP markers *E2M7–1* and *E6M6–2*, and the *Scm2* region to an interval of 12.8 cM within the AFLP marker *E1M8–1* and SSR marker *phi053* (Xu et al. 1999). One AFLP marker *E3M8–1* even co-segregated with the *Scm1* locus.

Based on pedigree records, two resistant European lines (D21 and D32) are closely related by descent. In contrast, the third resistant European maize inbred FAP1360A and U.S. resistant inbred Pa405 were developed independently (Kuntze et al. 1995). For successful long-term resistance management (e.g., pyramidization), as many different resistance genes as possible need to be identified. Therefore, it is an important question whether all of the maize inbreds resistant to SCMV harbor the same or different, possibly closely linked, resistance genes.

The objective of the present study was to trace back the origin of the SCMV resistance genes *Scm1* and *Scm2* in resistant, or partially resistant, lines by using markers closely linked to them according to a previous study (Xu et al. 1999).

Materials and methods

Plant materials

Twenty maize inbreds were analyzed including (1) four resistant lines D21, D32, FAP1360A (Europe) and Pa405 (United States), (2) direct ancestral lines of the three resistant European lines: Iodent, A632, Co125, Co158, V3, W401, WD, W59E, FAP594A and FAP493B, (3) three partially resistant European lines D06, FAP1396A, and FAP693A and (4) three highly susceptible European lines F7, D408 and D145 (see Table 1). Pedigree relationships of the four resistant lines are outlined in Fig 1 and based on personal communication (D. Klein, University of Hohenheim; K.-H. Camp, Delley Seeds and Plants AG) and Gerdes et al. (1993).

SCMV inoculation and scoring

Evaluation of inbred lines for resistance to SCMV in 1993 at Hohenheim and in 1994 at Hohenheim as well as Eckartsweier was reported by Kuntze et al. (1997), including 11 out of the 20 inbreds (see Table 1) employed in the present study. Apart from the resistant U.S. line Pa405 and three partially resistant lines, the remaining 16 lines were further evaluated for their resistance to SCMV under field or greenhouse conditions in 1998. In two independent field trials conducted in 1998 at Hohenheim, each line was planted in one-row plots of 25 plants with two replications. SCMV inoculation was carried out twice by the airbrush technique (Fuchs et al. 1996) at the 3–4 leaf stage and at a 1-week interval. Mosaic symptoms were scored four times at 2-week intervals. Final scoring was conducted at flowering stage about 7 weeks after the second inoculation. The final scoring in previous experiments (Kuntze et al. 1997) was conducted 4 (1993) or 7 (1994) weeks after the second inoculation.

DNA isolation, RFLP, SSR, and AFLP analyses

Leaf material was collected, freeze dried and ground to powder for each of the 20 inbred lines. High-quality genomic DNA was obtained by the CTAB method (Hoisington et al. 1994) with two extra purification steps using chloroform/isoamyl alcohol. Molecular markers closely linked to two SCMV resistance loci, *Scm1* and *Scm2*, identified in the study of Xu et al. (1999) were selected to assay the 20 genotypes. Markers mapping to the *Scm1* region included two RFLP markers [*bnl6.29* and the nucleolus organizer region (*nor*)], two SSR markers (*phi126* and *phi077*), and 12 AFLP markers (*E1M4–1*, *E2M7–1*, *E3M8–1*, *E6M6–2*, *E4M6–1*, *E4M2–1*, *E8M7–1*, *E4M2–2*, *E6M6–1*, *E6M1–5*, *E1M5–1*, and *E4M3–1*). In the *Scm2* region, two SSR markers (*phi036* and *phi053*) and 11 AFLP markers (*E2M7–2*, *E1M8–1*, *E6M1–1*, *E6M1–3*, *E8M5–2*, *E8M7–3*, *E6M1–2*, *E1M4–2*, *E6M1–4*, *E8M5–1* and *E8M7–2*) were employed.

For RFLP analyses, genomic DNA was digested with *Eco*RI, separated on 1% agarose gels, and vacuum-blotted onto nylon membranes. Hybridization was carried out with the Digoxigenin-dUTP (Boehringer, Mannheim, Germany) labelled RFLP marker *bnl6.29* and the intergenic spacer region of rDNA cloned in the plasmid pZms1 (McMullen et al. 1986). For SSR analyses, the sequences of four SSR primers (*phi126*, *phi077*, *phi036 and phi053*) were obtained from the maize database (http://teosinte.agron.missouri.edu/Coop/SSR-Probes/SSR1.htm) and synthesized by Amersham Pharmacia Biotech (Freiburg, Germany). PCR amplification was performed as described by Xu et al. (1999). AFLP analyses were carried out by using the AFLP™ Analysis System Kit (GibcoBRL, Life Technologies) with P^{33}-labelled oligonucleotides according to the supplier's instructions and as described by Vos et al. (1995). Twelve informative primer combinations corresponding to 23 linked AFLP markers were employed in our analysis (see Fig. 2).

Data collection

All closely linked markers used in the present study were identified in a BC5 population of the cross FAP1360A (resistant)×F7 (susceptible) (Xu et al. 1999). Each of the linked markers was, therefore, polymorphic between the parental lines FAP1360A and F7. For evaluation of all 20 genotypes, a marker band was scored as positive (+), if it was identical with the band present in the resistant line FAP1360A, and negative (–) otherwise. Linked markers for both the *Scm1* and *Scm2* region were arranged according to their map positions (see Tables 2 and 3).

As illustrated in Table 2, 16 closely linked markers were distributed within 14.5 cM of the *Scm1* region. Frequently, several markers were mapped at the same chromosomal location. As a consequence, eight chromosomal locations were defined within the *Scm1* region. Identity of marker alleles was assumed, if two lines carried the same positive marker band (+/+). In contrast, simultaneous absence of the FAP1360A marker band for two lines (–/–) is less informative, because it is neither a clear indication of identity nor of difference between two lines. Different alleles (+/–, –/+) unambiguously proved non-identity of two lines at a given marker locus. Identity of a chromosomal region between two lines is supported by identity of all or most marker bands within a chromosomal region.

Results and discussion

SCMV resistance of the 20 inbred lines

In a survey of resistance to SCMV (Table 1), resistance of the three European inbreds D21, D32 and FAP1360A (Kuntze et al. 1997), as well as susceptibility of the three European inbreds F7, D408 and D145, was confirmed. Out of the ten direct ancestral lines, Co125 was partially resistant to SCMV (Table 1). A632 displayed complete resistance to SCMV under greenhouse conditions and partial resistance under field conditions. FAP594A was tested only in the greenhouse and displayed complete resistance to SCMV. FAP493B showed partial resistance under greenhouse conditions. The remaining six ancestral lines, Iodent, Co158, V3, W401, WD and W59E, were highly susceptible to SCMV under both field and greenhouse conditions.

Pedigree analysis of the *Scm1* region

By comparing the banding patterns between FAP1360A and its ancestral lines W59E, Co125, FAP493B and FAP954A, only FAP954A showed identical positive alleles for each of the 16 markers. In contrast, no positive marker band was found in the ancestral line FAP493B, and only a few positive marker alleles appeared in the remaining two ancestral lines Co125 and W59E (Table 2). In accordance with the marker results, FAP954A was the major ancestor of FAP1360A according to the pedigree records (Fig. 1), and completely resistant to SCMV

Table 1 Maize inbred lines, their origin and reaction to SCMV infection in field and greenhouse trials[a]

Inbred lines	Origin	Heterotic group	Field trial 1		Field trial 2		Greenhouse trial		Published data[d]	
			R[b]	S[c]	R	S	R	S	1993	1994
Ancestral lines										
Iodent							0	18		
A632	USA	Dent	18	25	5	3	3	0		
Co125	Canada	Dent	48	1	13	0	7	0	0	75
Co158	Canada	Dent	0	46	0	5	2	8		
V3	USA	Flint	0	44	0	5	0	7		
V401	USA	Dent	0	45	0	6	0	2		
WD	USA	Dent	0	48	0	7	0	7		
W59E						0	9			
FAP954A	Switzerland	Dent				8	0			
FAP493B	Switzerland	Dent				1	2			
Resistant										
D21	Germany	Dent	40	0	22	0			0	0
D32	Germany	Dent	52	0	17	0			0	0
FAP1360A	Switzerland	Dent	39	0	22	0			0	0
Pa405	USA	Dent							0	0
Susceptible										
F7	France	Flint	0	21	0	21				
D408	Germany	Dent	0	43	0	21			100	100
D145	Germany	Flint	0	40	0	22			100	100
Partial resistant									100	100
D09	Germany	Dent								
FAP1396A	Switzerland	Dent							0	26
FAP693A	Switzerland	Dent							0	29
									0	58

[a] All data were recorded from final scoring of resistance to SCMV, for details see Materials and methods
[b] Number of resistant plants
[c] Number of susceptible plants
[d] Percentage of susceptible plants published by Kuntze et al. (1997)

in the greenhouse trial (Table 1). Therefore, it is very likely that the entire *Scm1* region of 14.5 cM in FAP1360 A was derived from FAP954 A.

By comparing FAP1360A with the other two European resistant lines D21 and D32 (Fig. 2), positive marker bands were found to cover, without interruption, the region between the *nor* and *E8M7–1* (Table 2). Therefore, it is most likely that all three lines carry the same chromosomal region of 7.2 cM harboring the *Scm1* locus. Accordingly, *Scm1* has also been mapped to the short arm of chromosome 6 in the cross D32×D145 (Xia et al. 1999). Likewise, in the cross D21×D408 one major gene on chromosome 6 S was found to confer resistance in D21 to SCMV (Melchinger et al. 1998). Hence, it is most likely that these three European resistant lines share the same resistance gene at this locus. In contrast, the resistant U.S. line Pa405 displayed a rather different banding pattern for the markers in the *Scm1* region. Even AFLP marker band *E3M8–1*, which cosegregates with *Scm1* in FAP1360A, was absent in Pa405. Therefore, it seems rather unlikely that the *Mdm1* gene identified in Pa405 has the same origin as the *Scm1* gene in the three resistant European lines.

In order to determine the origin of *Scm1* for both lines D21 and D32, all of their seven ancestral lines were evaluated with each of the 16 linked markers. As shown in Table 2, one ancestral line (A632) had almost the same banding profile as FAP1360A except for the two most distant markers *phi126* and *E4M3–1*. A632 shared the same chromosomal region of 8.7 cM (from chromosomal location 1 to 5) with D32, and of 7.2 cM (from chromosomal location 2 to 5) with D21. Another ancestral line V3 had only the two AFLP marker bands *E3M8–1* and *E6M6–2* closest to *Scm1* in common with FAP1360A, D21 and D32, but was different at the other marker loci. The remaining five ancestral lines, Iodent, Co125, Co158, W401 and WD, displayed largely different banding patterns, especially for the co-segregating marker band *E3M8–1*. A632 and Co125 were partially resistant to SCMV, while the other ancestors were highly susceptible to SCMV. Therefore, the *Scm1* region for both lines D21 and D32 seems to be derived from A632.

A pedigree relationship among D21, D32 and FAP1360A is given by their common ancestor line Co125. Since Co125 was classified as partially resistant to SCMV, one would have speculated without prior knowledge of the marker data that *Scm1* originated from Co125 for all three lines. Although the possibility of a double-crossover within a very short region around *Scm1* can not be ruled out entirely, the marker data strongly support the hypothesis of FAP954A and A632 being the origin of the *Scm1* region in FAP1360A, D21 and D32, respectively. There is another pedigree link between FAP1360A with D21 and D32 by synthetic Minnesota #13 (Fig. 1): D21/D32–A632–Minnesota #13–FAP493–FAP1360A. However,

Table 2 Survey of marker bands present or absent in 20 maize inbred lines using RFLP, AFLP and SSR markers flanking *Scm1*

Inbred lines	Proximal to *Scm1*						Distal to *Scm1*									
	CL1[a]			CL2	CL3	CL4	CL5				CL6			CL7		CL8
	phi126 4.4[b]	E1M4–1 4.4	nor 4.4	E2M7–1 2.9	E3M8–1 0.0	E6M6–2 2.9	bnl6.29 4.3	E4M6–1 4.3	E4M2–1 4.3	E8M7–1 4.3	E4M2–2 5.8	E6M6–1 5.8	E6M1–5 5.8	phi077 7.2	E1M5–1 7.2	E4M3–1 10.1
Ancestral lines																
Iodent	–	+	+	–	–	+	–	–	+	+	–	–	–	–	+	+
A632	–	+	+	+	+	+	+	+	+	+	+	+	+	+	+	–
Co125	+	–	+	–	–	+	–	+	–	+	–	–	–	–	–	–
Co158	–	–	–	–	–	+	–	+	–	–	+	–	–	–	+	+
V3	–	–	–	–	+	+	–	+	+	–	–	–	–	+	+	+
W401	–	+	–	–	–	+	–	–	–	–	–	–	–	+	–	+
WD	+	–	–	–	–	+	–	–	–	–	–	–	–	–	+	–
W59E	–	–	–	–	–	+	–	–	–	–	–	–	–	–	+	+
FAP954A	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
FAP493B	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Resistant																
D21	–	–	+	+	+	+	+	+	+	+	–	–	–	+	+	+
D32	–	+	+	+	+	+	+	+	+	+	–	–	–	–	+	+
FAP1360A	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Pa405	–	–	+	–	–	+	–	–	–	+	–	–	–	–	–	–
Susceptible																
F7	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
D145	+	+	+	–	–	–	–	+	–	–	–	+	–	–	–	–
D408	–	+	–	–	+	+	–	–	+	–	–	–	–	+	+	+
Partial resistant																
D09	–	+	+	+	+	+	+	+	+	+	–	–	–	+	+	+
FAP1396A	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
FAP693A	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	–

[a] CL: chromosomal location
[b] The genetic distance in cM between the markers and *Scm1* was taken from Xu et al. (1999). All markers are arranged according to their map position

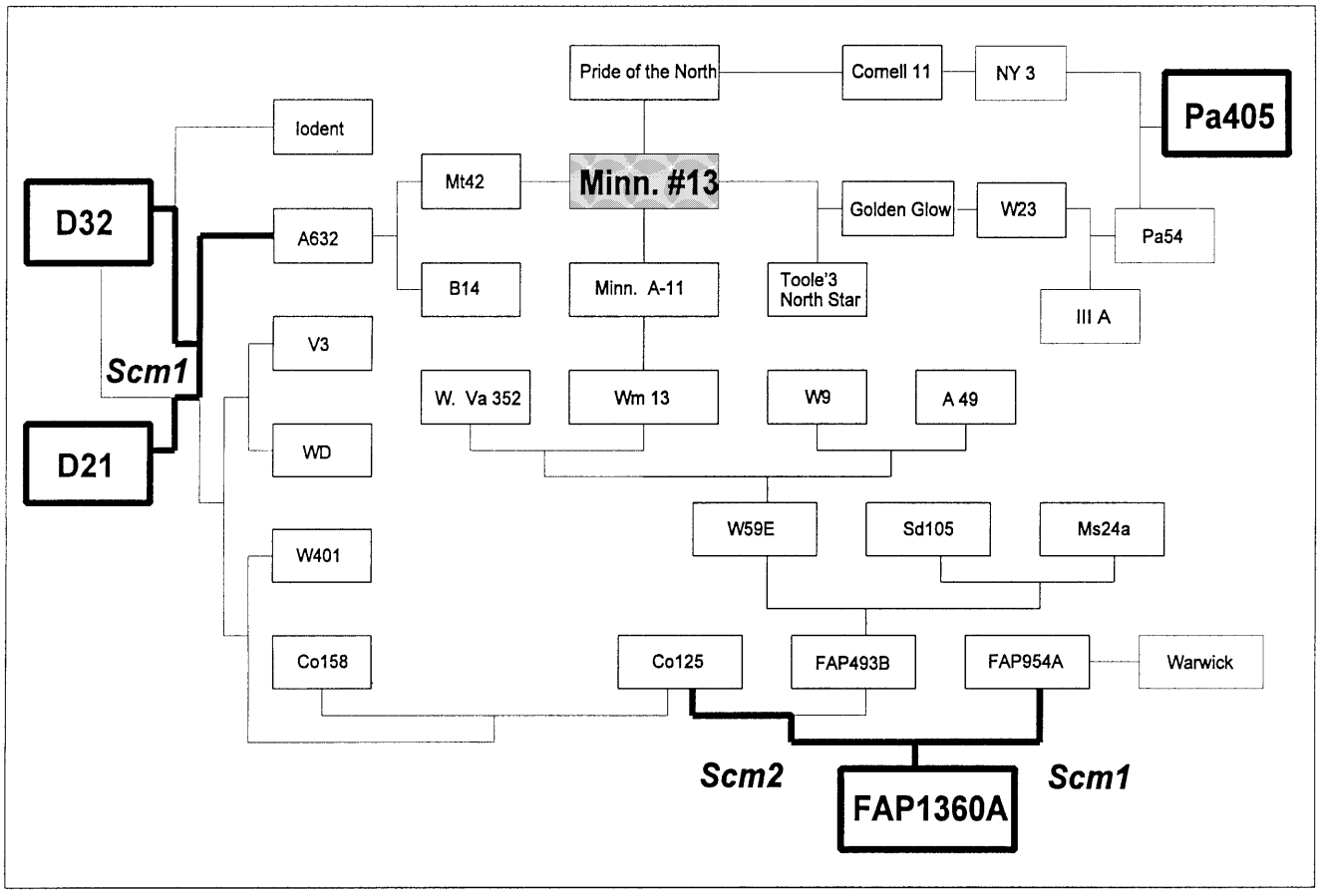

Fig. 1 Pedigree relationships of maize inbreds D21, D32, FAP1360A and Pa405

FAP493 can not be the donor of the *Scm1* region based on the marker data. Thus, Minnesota #13 was less likely to be the original ancestor for the *Scm1* region of all three lines. A pedigree relationship between A632 and FAP1360A or its ancestor FAP954A is not known, but possibly A632 was used to build up the Warwick synthetic from which FAP954A was extracted (Fig. 1). Therefore, molecular markers can clarify, or at least disprove, the pedigree relationships of individual chromosome segments directly at the level of DNA.

Within the three partially resistant lines, FAP1396A shared an identical banding profile with FAP1360A for the *Scm1* region. FAP693A also had the same banding pattern with FAP1360A except for a distant marker band *E4M3–1* (distal to *Scm1*). D09 displayed the same banding pattern as FAP1360A for an interval of 7.2 cM from chromosomal location 2 to 5. Thus, it is very likely that the *Scm1* region of FAP1360A was also present in all three partially resistant lines.

As expected, banding patterns of the three highly susceptible lines differed distinctly from those in FAP1360A. All linked markers were different for F7, and only a few marker bands were positive for the other two lines D408 and D145. Thus, it can be concluded that the *Scm1* region was absent in these three susceptible lines.

Pedigree analysis of the *Scm2* region

Among the four ancestral lines of FAP1360A (W59E, Co125, FAP493B and FAP954A), Co125 exhibited an identical banding pattern with FAP1360A within an interval of 16.9 cM from chromosomal location 2 to 5, including the *Scm2* locus (Table 3). Moreover, Co125 was found to be partially resistant to SCMV under both field and greenhouse tests (Table 1), and related to FAP1360A with an co-ancestry coefficient (Malécot 1948) f=0.25 (Fig. 1). The other ancestral lines showed banding patterns in the *Scm2* region rather different from FAP1360A. Hence, the *Scm2* region in FAP1360A most likely originated from Co125.

In a survey of the other three resistant lines, D21, D32 and Pa405, remarkable differences in banding patterns were observed between these three lines and FAP1360A (Table 2). As shown in Fig. 1, Co125, the likely donor of the *Scm2* region for FAP1360A, was also one of the ancestors for both D21 (f=0.09) and D32 (f=0.06). However, the *Scm2* region of Co125 was rather different from that in D21 and D32 according to closely linked markers (Table 2). One possibility might be that Co125 transmitted a very short *Scm2* segment to D21 and D32, too short to be detected by the two markers flanking *Scm2*. Other more likely possibilities are: (1) different sources of *Scm2* for FAP1360A and D21 or D32, and (2) absence of *Scm2* at least in D21, where the *Scm2* region did not contribute to SCMV resistance in cross D21×D408 (Melchinger et al. 1998).

Table 3 Survey of marker bands present or absent in 20 maize inbred lines using RFLP, AFLP and SSR markers flanking *Scm2*

Inbred lines	Proximal to *Scm2*			Distal to *Scm2*									
	CL1[a]	CL2	CL3	CL4		CL5		CL6			CL7	CL8	CL9
	phi036 28.5[b]	E2M7–2 10.9	E1M8–1 9.4	phi053 3.4	E6M1–1 3.4	E6M1–3 6.0	E8M5–2 6.0	E8M7–3 7.6	E6M1–2 7.6	E1M4–2 7.6	E6M1–4 9.2	E8M5–1 19.4	E8M7–2 23.1
Ancestral lines													
Iodent	–	–	–	–	+	–	+	–	–	–	–	–	–
A632	+	–	–	+	–	–	+	+	–	–	–	–	+
Co125	–	+	+	+	+	+	+	+	+	–	+	–	+
Co158	–	–	–	–	–	–	+	–	+	–	–	–	+
V3	–	+	–	–	–	–	–	–	–	+	+	+	–
W401	–	–	–	+	–	–	–	+	+	+	–	+	–
WD	+	+	–	+	+	–	+	–	–	+	–	+	+
W59E	–	–	–	+	+	–	–	+	–	–	–	+	+
FAP954A	–	–	–	+	–	–	+	+	–	+	–	–	+
FAP493B	–	–	–	–	+	–	–	+	–	–	–	+	+
Resistant													
D21	–	–	–	–	–	–	+	–	+	+	–	–	–
D32	+	–	+	–	–	–	+	+	+	–	–	–	–
FAP1360A	+	+	+	+	+	+	+	+	+	+	+	+	+
Pa405	–	–	–	–	+	–	–	+	–	+	–	–	+
Susceptible													
F7	–	–	–	–	–	–	–	–	–	–	–	–	–
D145	–	–	–	–	–	–	–	–	–	–	+	–	+
D408	–	+	–	–	+	+	+	+	–	+	–	–	+
Partial resistant													
D09	+	–	+	–	–	–	+	+	+	–	–	–	+
FAP1396A	–	+	–	–	–	–	–	–	+	+	–	+	+
FAP693A	–	–	–	–	–	–	+	+	–	–	–	+	+

[a] CL: chromosomal location
[b] The genetic distance in cM between the markers and *Scm1* was taken from Xu et al. (1999). All markers are arranged according to their map position

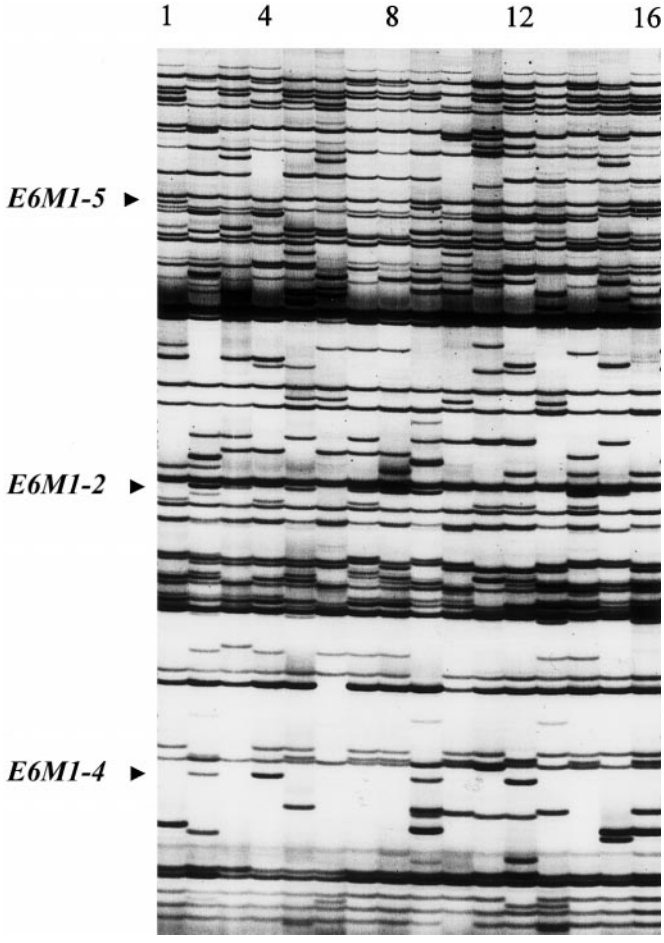

Fig. 2 AFLP profiles of 16 maize inbreds including AFLP markers (indicated by *arrows*) *E6M1–2*, *E6M1–4* and *E6M1–5* (Xu et al. 1999). *Lanes 1–16* correspond from left to right to maize inbreds A632, Co125, Co158, V3, W401, WD, D21, D32, FAP1360A, Pa405, F7, D145, D408, D09, FAP1396A and FAP693A

The three partially resistant lines also revealed banding profiles different from those in FAP1360A, suggesting that the *Scm2* locus was not present in these three lines. This is in agreement with our previous studies (Xia et al. 1999; Xu et al. 1999) in that *Scm1* confers resistance, but it is not sufficient for complete resistance without additional resistance genes. As expected, banding patterns differed considerably between FAP1360A and the highly susceptible lines.

Conclusions

The genetic basis of SCMV resistance in European elite maize materials seems to be rather narrow with respect to the major resistance gene *Scm1*. All completely and partially resistant European inbreds evaluated in this study harbored the same genomic region flanking *Scm1* and, therefore, very likely *Scm1* itself. Broadening the genetic basis for major SCMV resistance genes is desirable and might be achieved by introgression of the respective chromosome segments from Pa405 or other independent sources of SCMV resistance into European maize materials.

Scm1 was found to be insufficient for complete resistance to SCMV (Xia et al. 1999; Xu et al. 1999). Interestingly, the marker genotypes of the three completely resistant lines were diverse for the *Scm2* region, indicating the presence of different SCMV resistance loci in this or other genomic regions. Hence, pyramidization of SCMV resistance genes within European germplasm seems to be more promising for resistance loci different from and complementing *Scm1*. However, *Scm2* was mapped in two different crosses including SCMV-resistant lines D32 and FAP1360A (Xia et al. 1999; Xu et al. 1999). The question of identity versus non-identity of "*Scm2*" in both lines needs to be clarified by high-resolution mapping of this genomic region.

Marker-based evaluation of pedigree records is of immediate value for breeders. Molecular markers help to identify relevant chromosome segments directly, as well as donors of valuable genes. Using the same AFLP or RFLP markers employed in this study, breeders can screen their materials tracing back to A632, FAP954A or Co125 for the presence of *Scm1* or *Scm2* in modern elite materials, and this information can help them to design efficient resistance breeding programs.

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