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Two chromosome segments confer multiple potyvirus resistance in maize

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With 2 tables

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Abstract

Potyviruses cause serious yield losses in maize production worldwide. While the maize dwarf mosaic virus (MDMV) predominates in the USA, sugarcane mosaic virus (SCMV) is a major pathogen in China and Germany. In previous studies, inbred FAP1360A revealed complete resistance against both MDMV and SCMV. Two major SCMV resistance genes, *Scmv1* and *Scmv2*, were located on chromosomes 6 and 3, respectively, in populations derived from crosses with the susceptible inbred line F7. For validation of these results obtained in segregating backcross- or $F_{2:3}$-populations, near-isogenic lines to F7 have been produced after one initial cross to FAP1360A by repeated backcrossing to F7, phenotypic selection for SCMV resistance, and marker-assisted selection for the *Scmv1* and *Scmv2* regions from FAP1360A. The near-isogenic line F7R has been studied in detail both at the genomic level and for resistance to different potyviruses. Based on 112 polymorphic simple sequence repeat markers, F7R received genomic segments introgressed from FAP1360A exclusively in the *Scmv1* and *Scmv2* chromosomal regions. F7R conferred complete resistance to SCMV and MDMV, but also to zea mosaic virus and to systemic infection by wheat streak mosaic virus. FAP1360A, F7, F7R were not systemically infected by high plains virus. Thus, introgression of *Scmv1* and *Scmv2* from FAP1360A into F7 was sufficient to generate the first potyvirus multiresistant European Flint line reported so far.

Key words: *Zea mays* — potyvirus resistance — sugarcane mosaic virus — maize dwarf mosaic virus — wheat streak mosaic virus — zea mosaic virus

The closely related potyviruses sugarcane mosaic virus (SCMV) and maize dwarf mosaic virus (MDMV) are amongst the most important virus diseases in maize worldwide and cause serious yield losses in susceptible cultivars (Fuchs and Grüntzig 1995). While SCMV is more prevalent than MDMV in Germany (Fuchs and Grüntzig 1995) and China (Wang et al. 2003), MDMV is a widespread viral disease in the southern US Corn Belt (Louie et al. 1991). Symptoms include stunting, chlorosis, reduction in plant weight, and, therefore, a reduction in yield (Fuchs and Grüntzig 1995). In addition, maize is a host of several other members of the Potyviridae family, including wheat streak mosaic virus (WSMV), Johnson grass mosaic virus (JGMV), sorghum mosaic virus (SrMV) and zea mosaic virus (ZeMV) (Kuntze et al. 1997, Seifers et al. 2000).

Chemical control of SCMV and MDMV is not effective because of the non-persistent mode of transmission of these viruses by aphids. Hence, the best method to control SCMV and MDMV infection is cultivation of resistant maize varieties. Resistance to SCMV, MDMV and WSMV within the USA germplasm was investigated in a number of studies (Redinbaugh et al. 2005). Inbred Pa405 has shown complete resistance to MDMV and SCMV under both field and greenhouse conditions (Louie et al. 1991). Rosenkrantz and Scott (1987) reported five genes in Pa405 causing resistance to MDMV. Restriction fragment length polymorphism analysis localized a major dominant gene, *Mdm1*, near the centromere of chromosome 6 giving resistance to MDMV in Pa405 (McMullen and Louie 1989). Moreover, Pa405 confers resistance to WSMV due to presence of three major resistance genes *Wsm1*, *Wsm2* and *Wsm3*, located in chromosome bin regions (Neuffer et al. 1997) 6.00/6.01, 3.04/3.05 and 10.04/10.05 respectively (McMullen et al. 1994).

Kuntze et al. (1997) screened 122 early maturing European maize inbreds for resistance to potyviruses under both greenhouse and field conditions. Three lines (D21, D32 and FAP1360A) displayed complete resistance to SCMV, MDMV, JGMV and SrMV (Kuntze et al. 1997). Field trials and segregation analysis uncovered one to three genes involved in resistance to SCMV in different crosses (Melchinger et al. 1998). Two major dominant genes, *Scmv1* on the short arm of chromosome 6 and *Scmv2* near the centromere of chromosome 3, were mapped by Melchinger et al. (1998). Both major resistance genes are essential for expression of complete resistance to SCMV. Whereas *Scmv1* suppresses the expression of SCMV symptoms during all stages of infection, *Scmv2* is expressed in later stages of plant development (Xia et al. 1999). More recently, the *Scmv1* region was shown most likely to contain two closely linked resistance genes (Dußle et al. 2003, Yuan et al. 2003). Two major genes were detected in Chinese germplasm in the *Scmv1* and *Scmv2* region as well (Wang et al. 2003, Zhang et al. 2003). In conclusion, the (partially) dominant resistant genes *Mdm1*, *Scmv1* and *Wsm1* identified in different crosses are located in the same region of chromosome 6 (6.00/6.01), whereas *Scmv2* and *Wsm2* are located in the same region of chromosome 3 (3.04/3.05) (Redinbaugh et al. 2005). However, it is still unknown, whether the same pleiotropic genes in each of the two regions confer resistance to several potyviruses or whether both regions harbour resistance gene clusters, the latter being supported by clusters of resistance gene analogues in both regions (Collins et al. 1998, Quint et al. 2003).

The objectives of this study were to validate at the isogenic level, whether introgression of both the *Scmv1* and the *Scmv2* region from the resistant donor FAP1360A is sufficient for complete SCMV resistance of the otherwise susceptible inbred

F7, as expected from previous mapping studies (Xu et al. 1999, Dußle et al. 2000, Yuan et al. 2003), and if the converted inbred F7 confers multiple resistance to different potyviruses. In the case of multiple potyvirus resistance, marker-assisted selection for both genome regions would provide a promising option to convert susceptible maize inbreds to lines with multiple potyvirus resistance. High plains virus (HPV) was included in this study, although not a potyvirus, since it is often found in association with WSMV (Redinbaugh et al. 2005). Moreover, quantitative trait loci conferring resistance to systemic spread of HPV were previously located in the *Scmv1* and the *Scmv2* region (Redinbaugh et al. 2005).

Materials and Methods

Plant materials: The early maturing European maize (*Zea mays* L.) inbreds, FAP1360A, completely resistant to SCMV, and F7, highly susceptible to SCMV (Kuntze et al. 1997), were crossed to produce F_1 offspring, and backcrossed seven times to F7 with two generations per year from 1995 to 1998. Selection for backcross (BC) plants without SCMV symptoms was only possible during the summer seasons, but not in the winter nursery (Dußle et al. 2003). Thus, BC generations 1, 3, 5 and 7 have been tested for SCMV resistance, whereas BC families in generations 2, 4 and 6 have been backcrossed to F7 without selection for SCMV resistance. Seed of the homozygous line F7R (a F7 near-isogenic line with FAP1360A introgression at the two target regions) was produced by three subsequent selfing steps starting from one SCMV-resistant BC7 plant carrying the donor regions from FAP1360A surrounding *Scmv1* and *Scmv2*, as confirmed by microsatellite [simple sequence repeat (SSR)] markers.

Microsatellite analyses: More than 150 publicly available SSRs (http://www.maizegdb.org/) distributed over all 10 maize chromosomes were employed to characterize F7R in comparison with F7 and FAP1360A. All polymorphic SSRs (112) are displayed in Table 1. Primers and polymerase chain reaction protocols were used as published at http://www.maizegdb.org/.

SCMV assays: FAP1360A, F7 and F7R were assayed for SCMV infection in three independent greenhouse experiments at TU Munich (two experiments) and the Research Centre Flakkebjerg. Infection experiments were carried out using a randomized complete block design with two replications per experiment. A minimum of 10 plants per genotype and replication were sown in 1.5-l pots in the greenhouse. Young leaves with typical mosaic symptoms of the SCMV-infected susceptible parent F7 were homogenized using five volumes of a 0.01 M phosphate buffer at pH 7.0. Carborundum was added to the sap. Plants were artificially inoculated with SCMV (isolate Seehausen) by rub inoculation at the three-leaf stage (Kuntze et al. 1997). The first scoring of mosaic symptoms was conducted 1 week after initial inoculation. At the same time, all plants without symptoms were inoculated a second time. Virus symptoms were recorded at weekly intervals on seven dates.

MDMV assays: Seedlings were mechanically inoculated at the three-leaf stage by rub inoculation with MDMV isolate MD (Salomon et al. 2005). Two independent replicated greenhouse experiments were conducted at the Volcani Center in Bet Dagan. Extracts were prepared from leaves of MDMV-infected sweetcorn cv. Spirit in 50 mM borate buffer, pH 8.0, containing 1 mM EDTA and carborundum beads. Under greenhouse conditions, the first symptoms appeared 4 days after inoculation. Plants were inspected visually for symptoms at 14 (experiment 2) or 16 (experiment 1) days post-inoculation (DPI).

ZeMV assays: Zea mosaic virus isolate Z (Salomon et al. 2005) was extracted from an infected sweetcorn cv. Spirit and utilized to inoculate and score maize seedlings as described for MDMV. Two independent replicated greenhouse experiments were conducted at the Volcani Center in Bet Dagan.

WSMV and HPV assays: Resistance to WSMV and HPV were determined by inoculating the viruses individually (experiments 1 and 2 for WSMV and HPV respectively). Simultaneous inoculation with WSMV (Wooster isolate; McMullen and Louie 1991) and HPV (Kansas isolate; Seifers et al. 2002) was used to determine whether WSMV infection influenced maize susceptibility to HPV (experiment 3 for WSMV (+HPV) and for HPV (+WSMV), see Table 2). Vascular puncture inoculation (VPI, Louie 1995) was used to introduce HPV, WSMV and a mixture of HPV and WSMV into kernels of F7, F7R, FAP1360A and a susceptible sweetcorn line (Spirit). An inoculum was prepared and VPI was carried out on maize kernels presoaked in water for 2.5 h as described by Louie (1995), except that the HPV inoculum extraction buffer contained 50 mM $NaHPO_4$, 10 mM Na_2SO_3 and 5 mM EDTA, pH 7.8. Plants were scored for symptoms of HPV or WSMV infection on both the first emerging leaves (leaves 1–3), and on upper systemically infected leaves (systemic leaves).

High plains virus or WSMV presence was confirmed in extracts of six to 10 leaf samples from a subset of the plants using $F(ab')_2$ enzyme-linked immunosorbent assay (ELISA) (Louie et al. 2000). Both the lower (leaves 1–3) and upper (≥leaf 4) leaves were tested. The WSMV and HPV antibodies against the purified virus were raised in rabbits using a standard protocol (Harlow and Lane 1988), and showed specificity to WSMV and HPV respectively. The HPV antibodies reacted with a single 32-kDa protein that was present only in infected leaf tissue. Six to 10 samples from two independent experiments were tested. All WSMV and HPV experiments were conducted in replicated greenhouse trials at the Ohio State University in Wooster.

Results

SSR analysis of isogenic lines

The SSR alleles in chromosome bins 6.00/6.01 and 3.04/3.05, including *Scmv1* and *Scmv2*, were fixed in F7R from the donor line FAP1360A (Table 1). According to the public IBM2 map (http://www.maizemap.org/), the *Scmv1* region introduced from FAP1360A spans at least 21 cM (distance between bnlg161 and phi077) and the *Scmv2* region spans at least 8 cM (distance between umc2002 and bnlg1035). No additional FAP1360A alleles were discovered outside these two regions. Inbred line F7 was heterozygous for five markers on chromosomes 4, 5 (2), 7 and 8 (Table 1). In all five cases, one of both alleles was fixed in F7R.

SCMV, MDMV and ZeMV

In three independent experiments, no SCMV-infected plant was found for either FAP1360A or F7R, whereas 90 of 92 plants tested for F7 showed SCMV symptoms after final scoring (Table 2). In two independent experiments, no MDMV-infected plant was found for F7R, and only one for FAP1360A, whereas 12 of 20 plants tested for F7 showed MDMV symptoms after final scoring (Table 2). In two independent experiments, no ZeMV-infected plant was found for either FAP1360A or F7R of 101 plants tested for each, whereas 20 of 101 plants tested for F7 showed ZeMV symptoms at 16 DPI (Table 2).

WSMV and HPV

F7 was clearly susceptible to WSMV, and symptoms were observed on both lower and systemic leaves (Table 2). In contrast, limited symptoms (local lesions) of WSMV infection

Table 1: Polymorphic microsatellite (simple sequence repeat) markers between inbred lines F7 and FAP1360A (donor) used to identify donor segments transferred by repeated backcrossing to F7 (recurrent parent) into the isogenic sugarcane mosaic virus-resistant line F7R

	Bin												
Chromosome	00	01	02	03	04	05	06	07	08	09	10	11	12
1		bnlg149 bnlg1014 phi056	bnlg1429	bnlg176	bnlg2086 umc1849 umc1917			bnlg615	phi037 dupssr12	bnlg1502 bnlg1720		phi064 phi120 umc1681	umc1797
2			bnlg1017		phi083 bnlg1018 umc1026	dupssr21	bnlg1138 bnlg1831	bnlg1329	bnlg1662 bnlg1940	bnlg1520			
3		umc1746	bnlg1325		**umc2002**	**bnlg1456** **phi053** **umc1102** **bnlg1035** bnlg420 phi073	bnlg1160			bnlg1182 bnlg1257	umc1639		
4			umc1294	nc004 phi021	phi096	phi079	bnlg252 *bnlg1137*	bnlg1189	dupssr28	umc1101 umc1740	umc1109	phi006	
5		phi024 umc1478		bnlg1046	bnlg1208 bnlg1287 umc1221	bnlg278 *bnlg1237*	phi085	bnlg118	*umc1225*				
6	**bnlg161** **bnlg1043**	**bnlg426** **umc1753** **bnlg1371** **phi077**			nc009 umc1105	phi025 phi078 bnlg1732	umc1462	umc1063 bnlg1740	phi089				
7	bnlg2132	umc1066	phi034 *bnlg1094* bnlg1808	bnlg1070	bnlg2259	phi051	phi116						
8			phi119	bnlg669 bnlg1834		bnlg1176	bnlg1782 bnlg1031	*bnlg1065*	phi080	bnlg1131			
9			dupssr6	phi061			bnlg1191	bnlg619 bnlg1375 umc1675					
10			phi059	phi050 bnlg1079		bnlg1074 umc1506	bnlg1028	bnlg1360 bnlg1839					

Marker alleles in F7R originating from FAP1360A are in bold. The assignment of markers to chromosomal bin regions follows the concept of Neuffer et al. (1997) implemented in the US maize mapping project (http://www.maizemap.org/). Markers given in italics were scored with two marker alleles in F7, with only one of both bands fixed in F7R.

Table 2: Response of the isogenic line pair F7 and F7R as well as the donor line FAP1360A in greenhouse experiments to infection with sugarcane mosaic virus (SCMV), maize dwarf mosaic virus (MDMV), zea mosaic virus (ZeMV), wheat streak mosaic virus (WSMV) and high plains virus (HPV)

		No. infected vs. tested plants				
Virus[1]	Experiment	F7	F7R	FAP1360A	Inoculation	Scoring
SCMV	1	36/38	0/41	0/40	Mechanical	Systemic
	2	14/14	0/14	0/14	Mechanical	Systemic
	3	40/40	0/50	0/50	Mechanical	Systemic
MDMV	1	8/10	0/10	1/6	Mechanical	Systemic
	2	4/10	0/8	0/9	Mechanical	Systemic
ZeMV	1	11/51	0/51	0/51	Mechanical	Systemic
	2	9/50	0/50	0/50	Mechanical	Systemic
WSMV	1	29/29	18/30	20/33	VPI	Local
	1	29/29	0/30	0/33	VPI	Systemic
	2	56/56	67/67	65/65	VPI	Local
	2	40/56	0/67	0/65	VPI	Systemic
(+HPV)[2]	3	25/27	17/29	17/30	VPI	Local
(+HPV)[3]	3	25/27	0/29	0/30	VPI	Systemic
HPV	1	2/25	0/23	17/37	VPI	Local
	1	0/25	0/23	0/37	VPI	Systemic
	2	7/50	15/48	25/67	VPI	Local
	2	0/50	0/48	0/67	VPI	Systemic
(+WSMV)[2]	3	25/27	17/29	17/30	VPI	Local
(+WSMV)[3]	3	0/27	1/7	1/9	VPI	Systemic

Independent replicated experiments are indicated. Inoculation was either carried out by mechanical rub or vascular puncture inoculation (VPI). Scoring was performed for systemic or local (only WSMV and HPV) infection.
[1](+HPV), mixed inoculation of HPV with WSMV and scoring for WSMV; (+WSMV), mixed inoculation of WSMV with HPV and scoring for HPV.
[2]Symptoms for local infection with HPV and WSMV were rated visually (leaves 1–3).
[3]Symptoms for systemic infection (≥leaf 4) with HPV or WSMV were monitored both visually and by enzyme-linked immunosorbent assay, the latter included in this table.

were found only on the lower leaves of FAP1360A and F7R (Table 2). In VPI, these lower leaves are considered as 'inoculated leaf', in that these leaves are present in embryonic form at the time the kernels are inoculated (Louie and Abt 2004). No WSMV symptoms were present on the upper leaves of FAP1360A or F7R, which is consistent with a high degree of resistance to WSMV in these lines. ELISA analysis indicated that WSMV was present in systemic leaves of F7, but was present only in the lower leaves of FAP1360A and F7R.

The positive control inbred line, Spirit, was susceptible to HPV, with more than 90% of plants showing symptoms on both lower and systemic leaves. In contrast, F7, FAP1360A and F7R had similar responses to HPV inoculation, with symptoms being present only on some lower leaves, but not on systemic leaves (Table 2). However, ELISA analysis of systemic leaves indicated that HPV was only present in Spirit.

To determine whether co-inoculation with WSMV and HPV had any effect on HPV resistance, kernels of all four lines (including Spirit) were inoculated with a mixture of HPV and WSMV. Spirit inoculated with both viruses showed symptoms characteristic of both viruses on all leaves, and the presence of both viruses in systemic leaves was confirmed by ELISA. Neither FAP1360A nor F7R had symptoms of either virus on systemic leaves (Table 2), when inoculated together with the other virus. While both viruses could be detected in lower leaves by ELISA, WSMV was not detected in systemic leaves. However, the ELISA assays for one of nine and one of seven samples of FAP1360A and F7R, respectively, showed a high level of HPV antigen in systemic leaves. For F7, symptoms characteristic of WSMV infection were seen on both the lower and systemic leaves, and the presence of WSMV was confirmed in these samples by ELISA. However, no symptoms characteristic of HPV infection were seen on systemic leaves, and none of these samples contained HPV as determined by ELISA. Thus co-inoculation of F7 with WSMV and HPV did not support systemic movement of HPV in this line.

Discussion

The *Scmv1* and *Scmv2* genome regions received from the SCMV resistant donor FAP1360A confer complete SCMV resistance in the genomic background of the susceptible inbred F7. Although the presence of additional segments from FAP1360A in F7R cannot be excluded, no SSR markers outside the *Scmv1* and *Scmv2* regions indicated the presence of donor sequences. These findings are consistent with studies from Xu et al. (1999), Dußle et al. (2000) and Yuan et al. (2003), showing that both regions are sufficient for complete SCMV resistance in mapping populations derived from the cross F7 × FAP1360A. In contrast, in a $F_{2:3}$ mapping population derived from D32 × D145, three additional minor quantitative trait loci outside *Scmv1* and *Scmv2* were required for complete SCMV resistance (Xia et al. 1999). Thus, either the *Scmv1* and *Scmv2* regions from FAP1360A confer a higher level of resistance than the respective regions from D32 or F7 contributes alleles increasing SCMV resistance at other loci, absent in D145. As FAP1360A and D32 share identical marker haplotypes in the *Scmv1* region and similarities in the *Scmv2* region (Xu et al. 2000), additional but not yet identified resistance genes in F7 are the most likely explanation for these findings.

D21, D32 and FAP1360A were the only lines of an assortment of 122 European inbreds showing complete resistance against the potyviruses SCMV, MDMV, JGMV and SrMV (Kuntze et al. 1997). It was not known whether this was due to specific resistance genes for each of the viruses or the presence of the same genomic regions and/or genes conferring

resistance to several viruses in these three lines. Resistance of F7R in contrast to F7 against SCMV, MDMV, ZeMV and WSMV is in agreement with two genome regions (*Scmv1* and *Scmv2* regions) sufficient for complete resistance, at least in the F7 background. Thus, either a few pleiotropic genes or clusters of resistance genes in these two genome regions are the major determinants of potyvirus resistance in maize. Recombinant studies and ultimately gene isolation will be required to address the question of pleiotropy vs. clustering of potyvirus resistance genes in maize.

F7R is the first potyvirus-resistant European Flint line, since all completely or partially SCMV-resistant European inbreds reported so far are Dent lines (Kuntze et al. 1997). Thus, potyvirus resistance can be transferred from the Dent to the Flint heterotic pool in European maize by simple marker-assisted selection for two genome regions based on SSRs. This finding is relevant, as (i) Flint × Dent hybrids are predominant in Central Europe; and (ii) the modes of gene action of resistance alleles at the *Scmv1* and *Scmv2* loci were shown to be partially dominant, at least for some of the scoring dates (Xia et al. 1999, Yuan et al. 2003). For high levels of resistance, resistance alleles should be fixed at both loci in the hybrid, and thus be present in both parental inbreds. It remains to be shown, whether introduction of the *Scmv1* and *Scmv2* loci from FAP1360A will confer potyvirus resistance in any Flint line or whether additional resistance genes fixed in F7 will be required.

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References

Collins, N. C., C. A. Webb, S. Seah, J. G. Ellis, S. H. Hulbert, and A. Pryor, 1998: The isolation and mapping of disease resistance gene analogues in maize. Mol. Plant Microbe Interact. **11,** 968—978.

Dußle, C. M., A. E. Melchinger, L. Kuntze, A. Stork, and T. Lübberstedt, 2000: Molecular mapping and gene action of *Scm1* and *Scm2*, two major QTL contributing to SCMV resistance in maize. Plant Breeding **119,** 299—303.

Dußle, C. M., M. Quint, A. E. Melchinger, M. L. Xu, and T. Lübberstedt, 2003: Saturation of two chromosome regions conferring resistance to SCMV by targeted BSA. Theor. Appl. Genet. **106,** 485—493.

Fuchs, E., and M. Grüntzig, 1995: Influence of sugarcane mosaic virus (SCMV) and maize dwarf mosaic virus (MDMV) on the growth and yield of two maize varieties. J. Plant Dis. Protect **102,** 44—50.

Harlow, E., and D. Lane, 1988: Laboratory Manual of Antibodies. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY.

Kuntze, L., E. Fuchs, M. Grüntzig, B. Schulz, D. Klein, and A. E. Melchinger, 1997: Resistance of early-maturing European maize germplasm to sugarcane mosaic virus (SCMV) and maize dwarf mosaic virus (MDMV). Plant Breeding **116,** 499—501.

Louie, R., 1995: Vascular puncture of maize kernels for the mechanical transmission of maize white line mosaic virus and other viruses of maize. Phytopathology **85,** 139—143.

Louie, R., and J. J., Abt, 2004: Mechanical transmission of maize rough dwarf virus. Maydica **49,** 231—240.

Louie, R., W. R. Findley, J. K. Knoke, and M. D. McMullen, 1991: Genetic basis of resistance in maize to five maize dwarf mosaic virus strains. Crop Sci. **31,** 14—18.

Louie, R., M. G. Redinbaugh, D. T. Gordon, J. J. Abt, and R. J., Anderson, 2000: Maize necrotic streak virus, a new maize virus with similarity to species of the family Tombusviridae. Plant Dis. **84,** 1133—1139.

McMullen, M. D., and R. Louie, 1989: The linkage of molecular markers to a gene controlling the symptom response in maize dwarf mosaic virus strains. Mol. Plant Microbe Interact. **6,** 309—314.

McMullen, M. D., and R. Louie, 1991: Identification of a gene for resistance to wheat streak mosaic virus in maize. Phytopathology **81,** 624—627.

McMullen, M. D., M. W. Jones, K. D. Simcox, and R. Louie, 1994: Three genetic loci control resistance to wheat streak mosaic virus in the maize inbred Pa405. Mol. Plant Microbe Interact. **7,** 708—712.

Melchinger, A. E., L. Kuntze, R. K. Gumber, T. Lübberstedt, and E. Fuchs, 1998: Genetic basis of resistance to sugarcane mosaic virus in European maize germplasm. Theor. Appl. Genet. **96,** 1151—1161.

Neuffer, M. G., E. H. Coe, and S. R. Wessler, 1997: Mutants of Maize. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY.

Quint, M., C. M. Dußle, A. E. Melchinger, and T. Lübberstedt, 2003: Identification of genetically linked RGAs by BAC screening in maize and implications for gene cloning, mapping and MAS. Theor. Appl. Genet. **106,** 1171—1177.

Redinbaugh, M., M. Jones, and R. Gingery, 2005: The genetics of virus resistance in maize (*Zea mays* L.). Maydica **4,** 183—190.

Rosenkrantz, E., and G. E. Scott, 1987: Type of gene action in the resistance to maize chlorotic dwarf virus in corn. Phytopathology **77,** 1293—1296.

Salomon, R., N. Voronov, and D. L. Seifers, 2005: A heterogeneous population of maize dwarf mosaic virus causes epidemic in sweet corn. Parasitica **61,** 29—32.

Seifers, D. L., R. Salomon, V. Marie-Jeanne, B. Alliot, P. Signoret, S. Haber, A. Loboda, W. Ens, Y.-M. She, and K. G. Standing, 2000: Characterization of a novel potyvirus isolated from maize in Israel. Phytopathology **90,** 505—513.

Seifers, D. L., T. L. Harvey, R. Louie, D. T. Gordon, and T. J. Martin, 2002: Differential transmission of isolates of the High Plains virus by different sources of wheat curl mites. Plant Dis. **86,** 138—142.

Wang, F. G., X. D. Liu, Z. H. Wang, S. H. Zhang, X. H. Li, L. X. Yuan, X. Q. Han, and M. S. Li, 2003: Preliminary studies on QTL mapping of resistance to Sugarcane Mosaic Virus in maize. Acta Agron. Sin. **29,** 69—74.

Xia, X. C., A. E. Melchinger, L. Kuntze, and T. Lübberstedt, 1999: QTL mapping of resistance to sugarcane mosaic virus in maize. Phytopathology **89,** 660—667.

Xu, M. L., A. E. Melchinger, X. C. Xia, and T. Lübberstedt, 1999: High-resolution mapping of loci conferring resistance to sugarcane mosaic virus in maize using RFLP, SSR and AFLP markers. Mol. Gen. Genet. **261,** 574—581.

Xu, M. L., A. E. Melchinger, and T. Lübberstedt, 2000: Origin of Scm1 and Scm2 – two loci conferring resistance to sugarcane mosaic virus (SCMV) in maize. Theor. Appl. Genet. **100,** 934—941.

Yuan, L., C. M. Dußle, A. E. Melchinger, H. F. Utz, and T. Lübberstedt, 2003: Clustering of QTL conferring SCMV resistance in maize. Maydica **48,** 55—62.

Zhang, S. H., X. H. Li, Z. H. Wang, M. L. George, D. Jeffers, F. G. Wang, X. D. Liu, M. S. Li, and L. X. Yuan, 2003: QTL mapping for resistance to SCMV in Chinese maize germplasm. Maydica **48,** 307—312.