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Resistance to Sweetpotato Chlorotic Stunt Virus and Sweetpotato Feathery Mottle Virus Is Mediated by Two Separate Recessive Genes in Sweetpotato

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Abstract. **When sweetpotato chlorotic stunt crinivirus (SPCSV) and sweetpotato feathery mottle potyvirus (SPFMV) infect sweetpotato [*Ipomoea batatas* (L.) Lam.], they interact synergistically and cause sweetpotato virus disease (SPVD), a major constraint to food productivity in east Africa. The genetic basis of resistance to these diseases was investigated in 15 sweetpotato diallel families (1352 genotypes) in Uganda, and in two families of the same diallel at the International Potato Center (CIP), Lima, Peru. Graft inoculation with SPCSV and SPFMV resulted in severe SPVD symptoms in all the families in Uganda. The distribution of SPVD scores was skewed toward highly susceptible categories (SPVD scores 4 and 5), eliminating almost all the resistant genotypes (scores 1 and 2). Likewise, when two promising diallel families ('Tanzania' x 'Bikilamaliya' and 'Tanzania' x 'Wagabolige') were graft inoculated with SPCSV and SPFMV at CIP, severe SPVD was observed in most of the progenies. Individual inoculation of these two families with SPCSV or SPFMV, and Mendelian segregation analysis for resistant vs. susceptible categories led us to hypothesize that resistance to SPCSV and SPFMV was conditioned by two separate recessive genes inherited in a hexasomic or tetradisomic manner. Subsequent molecular marker studies yielded two genetic markers associated with resistance to SPCSV and SPFMV. The AFLP and RAPD markers linked to SPCSV and SPFMV resistance explained 70% and 72% of the variation in resistance, respectively. We propose naming these genes as s*pcsv1* and s*pfmv1*. Our results also suggest that, in the presence of both of these viruses, additional genes mediate oligogenic or multigenic horizontal (quantitative) effects in the progenies studied for resistance to SPVD.**

Sweetpotato (*Ipomoea batatas* (L.) Lam.), with a mean annual production of 132 million tons between 1991–2000, ranks among the top 10 food crops globally (FAO, 2000). Sweetpotatoes are grown in >100 countries, but developing countries account for over 98% of the total production (Gregory et al., 1990; Horton, 1988). Sweetpotatoes are a good source of carbohydrates, and both sweetpotato storage roots and foliage are nutritious foods (Food and Nutrition Board, 1980; Watt and Merrill, 1975; Woolfe, 1992). Orange fleshed sweetpotatoes are particularly nutritious, ranking highest in nutrient content of all vegetables for vitamins A and C, folate, iron, copper, calcium, and fiber, and they are an excellent source of the carotenoid, β-carotene, a vitamin A precursor (Woolfe, 1992).

Sweetpotato has enormous potential to contribute to the alleviation of widespread food shortages and vitamin A malnutrition common to many developing countries in sub-Saharan Africa (SSA). However, sweetpotato virus disease (SPVD) is a major constraint to increases in sweetpotato productivity in this region. SPVD in SSA is widespread (Hahn et al., 1981; Schaefers and Terry, 1976; Wisler et al., 1998) and causes up to 98% yield loss (Gibson et al., 1998; Karyeija et al., 1998). The disease is caused by the dual infection and synergistic interaction of sweetpotato feathery mottle potyvirus (SPFMV) and sweetpotato chlorotic

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Table 1. Expected phenotypic ratios (resistant: susceptible) in testcrosses involving virus resistance genes according to four cytological hypotheses, assuming sweetpotato genomic constitution is $R_1R_1R_2R_2R_2R_2$ (R = dominant, r = recessive). Tetradisomic and tetrasomic inheritance exhibit similar phenotypic ratios (Jones, 1967). Segregation ratios verified by Kumagai et al. (1990).

			Hypotheses 2 and 3			
			Hypothesis 2			
	Hypothesis 1		Tetradisomic	**Hypothesis 3**	**Hypothesis 4**	
	Autohexaploid		Strict preferential pairing	Tetrasomic	Allohexaploid	
	(hexasomic)		For R_1, R_2; genes on R_1 have	Alleles for resistance	(disomic)	
Gene	nonpreferential		disomic inheritance, genes	located on R_2	Allele for resistance	
dose	pairing, $R_1 = R_2$		on R_2 tetrasomic	genome	located on R_1	
Simplex	Rrrrrr	1:1	R_2rrr r_1r	1:1	Rr rr rr	1:1
			Rrrr Rr	1:1		
Duplex	RRrrrr	4:1	RRrr rr	5:1	Rr Rr rr	3:1
			Rrrr Rr	3:1	RR rr rr	1:0
			Rrrr RR	1:0		
Triplex	RRRrrrr	19:1	RRRr rr	1:0	Rr Rr Rr	7:1
			RRrr Rr	11:1	RR Rr rr	1:0
			Rrrr RR	1:0		
Quadruplex	RRRRrr	1:0	RRRR rr	1:0	RR Rr Rr	1:0

stunt crinivirus (SPCSV) (Cohen et al., 1992; Hoyer et al., 1996; Karyeija et al., 2000; Schaefers and Terry, 1976). SPFMV is transmitted in a nonpersistent, noncirculative manner by aphids (*Myzus persicae* (Sulzer), *Aphis gossypii* Glover and *A. craccivora* Koch) (Stubbs and McLean, 1958), while SPCSV is transmitted semi-persistently by the whitefly [*Bemesia tabaci* (Gennadius)] (Cohen et al., 1992; Schaefers and Terry, 1976).

Although SPVD is devastating, only a couple of studies of its inheritance have been conducted (Hahn et al., 1981; Ngeve and Bouwkamp, 1991). Genetic analysis of inheritance of SPVD and other traits in sweetpotato has been problematic because sweetpotato is a highly heterozygous hexaploid (x = 15, 2n = 90) (Jones, 1967; Magoon et al., 1970; Shiotani, 1988; Ting and Kehr, 1953; Ukoskit and Thompson, 1997) with complex segregation ratios (Jones, 1967; Kumagai et al., 1990; Poole, 1955). In addition to having a very complex genome, sweetpotato has many sporophytic self- and cross-incompatibilities (Jones, 1967; Martin, 1965, 1970; Nakanishi and Kobayashi, 1979; Nishiyama et al., 1975), and many genotypes fail to bloom and set seed (Jones, 1967; Magoon et al., 1970; Poole 1955). In most crop species, backcross, testcross, or F_2 populations are usually used to study the segregation of gametes from a heterozygous individual. However, the regular use of backcross or testcross populations for genetic analysis of sweetpotato is impractical due to the factors mentioned above and the strong inbreeding depression observed in backcrosses (Nissila et al., 1999). As a consequence, F_1 progeny resulting from crosses between clones, which result in high levels of segregation, are used to study the inheritance of traits in sweetpotatoes (Jones, 1977; Jones, et al., 1976a, 1976b; Thompson, et al., 1994).

Much attention has been given to the potential applications of DNA markers to improve plant breeding efficiency. These technologies provide two new opportunities for improving sweetpotato virus resistance. First, they provide a basis for accelerated breeding through early selection of traits, and second, they may enable more efficient selection of parents for subsequent crosses. DNA-based markers such as randomly amplified polymorphic DNA (RAPD), restriction fragment length polymorphisms (RFLP), microsatellites or simple sequence length polymorphisms (SSLP), and amplified fragment length polymorphisms (AFLP) have been associated with simply inherited and complex traits (quantitative trait loci, QTL) in oilseed rape. (*Brassica napus* L.) (Pongam et al., 1998), potato (*Solanum tuberosum* L.) (Barone et al., 1990; Bendahmane et al. 1997; Bradshaw et al., 1998; De Jong et al., 1997; Jacobs et al. 1995) and many other polyploid crops (Jin et al., 1998).

In this study, we report on 1) a series of genetic studies focused on examining the inheritance of resistance to SPCSV, SPFMV and SPVD; 2) the development of a preliminary linkage map of sweetpotato; and 3) the identification of AFLP and RAPD markers linked to genes conferring resistance to SPCSV and SPFMV on this map. AFLPs (Vos et al., 1995), which are typically dominant markers based on the selective PCR amplification of small fragments (80 to 400 bp) of genomic DNA, were selected as the primary marker system because they generate a large number of polymorphic bands (i.e., segregating markers), making it possible to develop linkage maps for plants with relative ease and cost effectiveness.

Materials and Methods

In a previous study, Mwanga et al. (2002) crossed 10 parental (p) sweetpotato clones with varying resistance to SPVD in a modified diallel, disregarding reciprocals, and using the model II, method 4 mating design of Griffing (1956). The work was conducted during 1996–97 at the Namulonge Agricultural and Animal Production Research Institute (NAARI) in Uganda. The mating design yielded a total of 45 families [(p(p – 1)/2]. Details of the plant materials, and screenhouse and field experimental designs were described by Mwanga et al. (2002). The two experiments described herein were conducted to further quantify the genetic basis of resistance to SPVD in these progenies. The first experiment consisted of a test of 15 promising diallel progenies for resistance to SPVD at NAARI. The second consisted of a test for resistance to SPCSV and SPFMV, the component viruses of SPVD, and it was conducted at the International Potato Center (CIP), Lima, Peru.

Inheritance of resistance to SPVD in promising diallel progenies. To generate information on possible modes of inheritance (Kumagai et al., 1990; Jones, 1967) of resistance to SPVD, 15

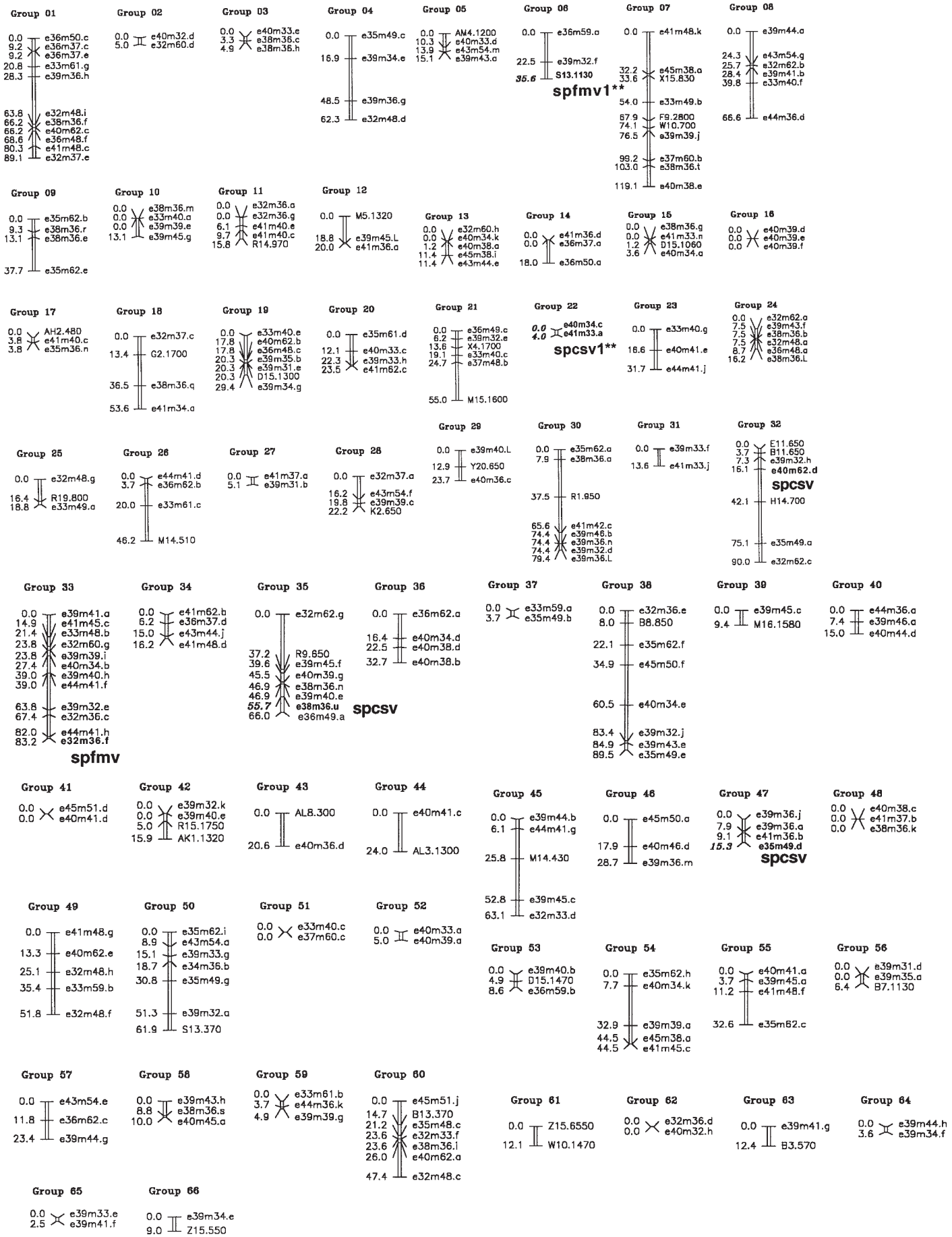
Group 01
0.0 e36m50.c
9.2 e36m37.c
9.2 e36m37.e
20.8 e33m61.g
28.3 e39m36.h
63.8 e32m48.i
66.2 e38m36.f
66.2 e40m62.c
68.6 e36m48.f
80.3 e41m48.c
89.1 e32m37.e
Group 02
0.0 e40m32.d
5.0 e32m60.d
Group 03
0.0 e40m33.e
3.3 e38m36.c
4.9 e38m36.h
Group 04
0.0 e35m49.c
16.9 e39m34.e
48.5 e39m36.g
62.3 e32m48.d
Group 05
0.0 AM4.1200
10.3 e40m33.d
13.9 e43m54.m
15.1 e39m43.a
Group 06
0.0 e36m59.a
22.5 e39m32.f
35.6 S13.1130
spfmv1**
Group 07
0.0 e41m48.k
32.2 e45m38.a
33.6 X15.830
54.0 e33m49.b
67.9 F9.2800
74.1 W10.700
76.5 e39m39.j
99.2 e37m60.b
103.0 e38m36.t
119.1 e40m38.e
Group 08
0.0 e39m44.a
24.3 e43m54.g
25.7 e32m62.b
28.4 e39m41.b
39.8 e33m40.f
66.6 e44m36.d
Group 09
0.0 e35m62.b
9.3 e38m36.r
13.1 e38m36.e
37.7 e35m62.e
Group 10
0.0 e38m36.m
0.0 e33m40.a
0.0 e39m39.e
13.1 e39m45.g
Group 11
0.0 e32m36.a
0.0 e32m36.g
6.1 e41m40.e
9.7 e41m40.c
15.8 R14.970
Group 12
0.0 M5.1320
18.8 e39m45.L
20.0 e41m36.a
Group 13
0.0 e32m60.h
0.0 e40m34.k
1.2 e40m38.a
11.4 e45m38.l
11.4 e43m44.e
Group 14
0.0 e41m36.d
0.0 e36m37.a
18.0 e36m50.a
Group 15
0.0 e38m36.g
0.0 e41m33.n
1.2 D15.1060
3.6 e40m34.a
Group 16
0.0 e40m39.d
0.0 e40m39.e
0.0 e40m39.f
Group 17
0.0 AH2.480
3.8 e41m40.c
3.8 e35m36.n
Group 18
0.0 e32m37.c
13.4 G2.1700
36.5 e38m36.q
53.6 e41m34.a
Group 19
0.0 e33m40.e
17.8 e40m62.b
17.8 e36m48.c
20.3 e39m35.b
20.3 e39m31.e
20.3 D15.1300
29.4 e39m34.g
Group 20
0.0 e35m61.d
12.1 e40m33.c
22.3 e39m33.h
23.5 e41m62.c
Group 21
0.0 e36m49.c
6.2 e39m32.e
13.6 X4.1700
19.1 e33m40.c
24.7 e37m48.b
55.0 M15.1600
Group 22
0.0 e40m34.c
4.0 e41m33.a
spcsv1**
Group 23
0.0 e33m40.g
18.6 e40m41.e
31.7 e44m41.j
Group 24
0.0 e32m62.a
7.5 e39m43.f
7.5 e38m36.b
7.5 e32m48.a
8.7 e36m48.a
16.2 e38m36.L
Group 25
0.0 e32m48.g
16.4 R19.800
18.8 e33m49.a
Group 26
0.0 e44m41.d
3.7 e36m62.b
20.0 e33m61.c
46.2 M14.510
Group 27
0.0 e41m37.a
5.1 e39m31.b
Group 28
0.0 e32m37.a
16.2 e43m54.f
19.8 e39m39.c
22.2 K2.650
Group 29
0.0 e39m40.L
12.9 Y20.650
23.7 e40m36.c
Group 30
0.0 e35m62.a
7.9 e38m36.a
37.5 R1.950
65.6 e41m42.c
74.4 e39m46.b
74.4 e39m36.n
74.4 e39m32.d
79.4 e39m36.L
Group 31
0.0 e39m33.f
13.6 e41m33.j
Group 32
0.0 E11.650
3.7 B11.650
7.3 e39m32.h
16.1 e40m62.d
spcsv
42.1 H14.700
75.1 e35m49.a
90.0 e32m62.c
Group 33
0.0 e39m41.a
14.9 e41m45.c
21.4 e33m48.b
23.8 e32m60.g
23.8 e39m39.i
27.4 e40m34.b
39.0 e39m40.h
39.0 e44m41.f
63.8 e39m32.e
67.4 e32m36.c
82.0 e44m41.h
83.2 e32m36.f
spfmv
Group 34
0.0 e41m62.b
6.2 e36m37.d
15.0 e43m44.j
16.2 e41m48.d
Group 35
0.0 e32m62.g
37.2 R9.650
39.6 e39m45.f
45.5 e40m39.g
46.9 e38m36.n
46.9 e39m40.e
55.7 e38m36.u
66.0 e36m49.a
spcsv
Group 36
0.0 e36m62.a
16.4 e40m34.d
22.5 e40m38.d
32.7 e40m38.b
Group 37
0.0 e33m59.a
3.7 e35m49.b
Group 38
0.0 e32m36.e
8.0 B8.850
22.1 e35m62.f
34.9 e45m50.f
60.5 e40m34.e
83.4 e39m32.j
84.9 e39m43.e
89.5 e35m49.e
Group 39
0.0 e39m45.c
9.4 M16.1580
Group 40
0.0 e44m36.a
7.4 e39m46.a
15.0 e40m44.d
Group 41
0.0 e45m51.d
0.0 e40m41.d
Group 42
0.0 e39m32.k
0.0 e39m40.e
5.0 R15.1750
15.9 AK1.1320
Group 43
0.0 AL8.300
20.6 e40m36.d
Group 44
0.0 e40m41.c
24.0 AL3.1300
Group 45
0.0 e39m44.b
6.1 e44m41.g
25.8 M14.430
52.8 e39m45.c
63.1 e32m33.d
Group 46
0.0 e45m50.a
17.9 e40m46.d
28.7 e39m36.m
Group 47
0.0 e39m36.j
7.9 e39m36.a
9.1 e41m36.b
15.3 e35m49.d
spcsv
Group 48
0.0 e40m38.c
0.0 e41m37.b
0.0 e38m36.k
Group 49
0.0 e41m48.g
13.3 e40m62.e
25.1 e32m48.h
35.4 e33m59.b
51.8 e32m48.f
Group 50
0.0 e35m62.i
8.9 e43m54.a
15.1 e39m33.g
18.7 e34m36.b
30.8 e35m49.g
51.3 e39m32.a
61.9 S13.370
Group 51
0.0 e33m40.c
0.0 e37m60.c
Group 52
0.0 e40m33.a
5.0 e40m39.a
Group 53
0.0 e39m40.b
4.9 D15.1470
8.6 e36m59.b
Group 54
0.0 e35m62.h
7.7 e40m34.k
32.9 e39m39.a
44.5 e45m38.a
44.5 e41m45.c
Group 55
0.0 e40m41.a
3.7 e39m45.a
11.2 e41m48.f
32.6 e35m62.c
Group 56
0.0 e39m31.d
0.0 e39m35.a
6.4 B7.1130
Group 57
0.0 e43m54.e
11.8 e36m62.c
23.4 e39m44.g
Group 58
0.0 e39m43.h
8.8 e38m36.s
10.0 e40m45.a
Group 59
0.0 e33m61.b
3.7 e44m36.k
4.9 e39m39.g
Group 60
0.0 e45m51.j
14.7 B13.370
21.2 e35m48.c
23.6 e32m33.f
23.6 e38m36.l
26.0 e40m62.a
47.4 e32m48.c
Group 61
0.0 Z15.6550
12.1 W10.1470
Group 62
0.0 e32m36.d
0.0 e40m32.h
Group 63
0.0 e39m41.g
12.4 B3.570
Group 64
0.0 e39m44.h
3.6 e39m34.f
Group 65
0.0 e39m33.e
2.5 e39m41.f
Group 66
0.0 e39m34.e
9.0 Z15.550

Fig. 1. Sweetpotato maternal ('Tanzania') linkage map based on AFLP and RAPD markers. Cumulative map distances (on left) are in cM. AFLP markers are denoted in the format exx.mxx.x. All other markers are RAPD markers. Each marker locus represents the individual fragments amplified by the same primer combination. Markers associated with resistance to sweetpotato chlorotic stunt virus (SPCSV) on linkage groups 22, 32, 35, and 47, and sweetpotato feathery mottle virus (SPFMV) on linkage groups 6 and 33 are in bold.

promising F_1 families from the 45 family diallel crosses evaluated during 1998–2000 (Mwanga et al., 2002) were selected for further study. Each progeny had a large number of individuals, up to 130 genotypes per family (1352 genotypes in total). The plants were grown in 2-L perforated plastic buckets in the screenhouse using previously described methods (Mwanga et al., 2002). Three plants per genotype were graft-inoculated with SPVD at 1.5 months after planting. SPVD severity was scored at 1.5, 2.5, and 3.5 months after inoculation using a 1 to 5 scale, where 1 = symptomless and 5 = most severe symptoms (vein clearing, chlorosis, puckering, stunting, tip-dieback) (Mwanga et al., 2002). The experiment was conducted during August to December 1998 and repeated during October 1999 to February 2000.

Inheritance of resistance to SPCSV, SPFMV and SPVD predicted by expected theoretical ratios. To examine resistance to SPCSV and SPFMV individually and in dual graft infection, and formulate hypotheses on the genetic basis of resistance based on expected segregation ratios of resistance to these viruses (Table 1), two crosses between African landrace sweetpotatoes with resistance to SPVD were generated. Family 1 (198 genotypes) consisted of a cross between 'Tanzania' (TAN) x 'Bikilamaliya' (BMA), while family 2 (87 genotypes) consisted of a cross between TAN x 'Wagabolige' (WAG). Seed from each cross were sent to CIP, and grown in screenhouses between May and December 2000. Each of the genotypes in the two families was propagated to furnish nine plants for three 1.1 L plastic pots (3 plants per pot).

All mother plants of the two families were tested for SPFMV, SPCSV, sweetpotato mild mottle virus (SPMMV), sweetpotato latent virus (SwPLV), sweetpotato chlorotic flecks virus (SPCFV), sweetpotato mild speckling virus (SPMSV), C-6 virus, and sweetpotato caulimovirus (SPCaLV) using nitrocellulose membrane enzyme-linked immunosorbent assay (NCM-ELISA) kits developed by CIP and North Carolina State University, Raleigh, N.C. (CIP, 1990). In NCM-ELISA, a sample was considered positive when a bluish-purple color developed on the test spot, and control spots from virus-free sweetpotato retained the color of the membrane without changing after 60 min of reaction with the substrate. Plants testing negative were propagated to furnish cuttings for experimental plants of each genotype.

To generate inoculum of SPFMV and SPCSV for the tests the following protocols were followed. The russet crack strain of SPFMV (RC-SPFMV) was obtained from the Virology Laboratory at CIP and used to infect *Ipomoea setosa* Ker or *I. nil* cv. Scarlett O'Hara seedlings using techniques described by Cadena-Hinojosa and Campbell (1981). Cotyledons of *I. setosa* seedlings were mechanically inoculated with SPFMV at the one- or two-leaf stage. The seedlings were covered with moistened polythene bags for 3 d after inoculation, and then the bags were removed to allow symptoms of SPFMV to develop. About 400 *I. nil* seedlings were mechanically inoculated to supply additional SPFMV-symptomatic scions for the experiments conducted on the two families at CIP using this procedure. Inoculum of SPCSV was also maintained in *I. setosa* (setosa 4) at CIP like SPFMV. However, because SPCSV cannot be transmitted mechanically (Cohen et al., 1992), inoculum was increased by graft inoculating ≈600 *I. setosa* seedlings with SPCSV-infected scions of *I. setosa*. Also, because SPCSV can be symptomless in *I. setosa,* a subsample of the SPCSV-infected plants was tested by ELISA to ensure that SPCSV was present in the grafting materials. To induce SPVD, *I. setosa* seedlings with established SPCSV scions were grafted with SPFMV-symptomatic *I. setosa* or *I. nil* scions about one week after they were inoculated with SPCSV.

The sweetpotato plants in the three pots (3 plants per pot) were treated as follows: 1) each plant in pot number one was graft inoculated with a SPFMV-symptomatic *I. setosa* or *I. nil* scion; 2) each plant in pot number two was grafted with a SPCSV-symptomatic *I. setosa* scion; and 3) each plant in pot number three was grafted with a SPFMV-symptomatic *I. nil* or *I. setosa* scion and a SPCSV-symptomatic *I. setosa* scion to induce SPVD. SPFMV was detected using NCM-ELISA, while SPCSV was detected using TAS-ELISA as previously described (Mwanga et al., 2002). Optical density (OD) values greater than two times the value of control samples were considered positive in for both ELISA tests. Three leaves (one per plant) were sampled per genotype. Shoots from plants testing negative in ELISA assays 3 to 4 weeks after inoculation were grafted on *I. setosa* plants to confirm negative readings (i.e., that the plants tested were not infected by SPCSV and/or SPFMV). The indicator plants were observed for symptom development for 2 months. Presence of virus in *I. setosa* scions of symptomless graft inoculated plants was confirmed and tested by ELISA.

Molecular genetic analysis of resistance to SPCSV, SPFMV and SPVD. The F_1-mapping population used to construct the AFLP map for this work was produced from a two-way pseudo-testcross of TAN x WAG in Uganda. Both parents of this cross are resistant to SPVD, but WAG is more resistant to SPVD than TAN under field SPVD inoculum pressure (Mwanga et al., 2002). True seed from this cross was sent to CIP for AFLP and RAPD genotyping, and SPCSV, SPFMV and SPVD phenotyping. Details of the methods used for DNA extraction, AFLP and RAPD reactions, primer combinations selection, marker nomenclature, marker scoring, estimation of recombination fractions, linkage mapping, and genome coverage and segregation ratios are described by Kriegner et al. (2001). AFLP and RAPD markers present in one parent and absent in the other were tested for 1:1 segregation ratio (presence:absence) in the progeny by a goodness-of-fit χ^2 test (≤ 0.05). This single-dose fragment (SDF) mapping strategy, described by Wu et al. (1992), has been widely used for developing genetic maps of polyploid species, such as sugarcane (*Saccharum spontaneum* L.) (Al Janabi et al., 1993; Da Silva et al. 1995; Ripol et al., 1999), potato (*Solanum tuberosum* L.) (Li et al., 1998), and alfalfa (*Medicago sativus* L.) (Yu and Paulus, 1993). The SDF mapping strategy is based on a simplex segregation pattern. In polyploids and outbreeding tree species, this mapping strategy employs the analysis of progeny of a cross between two unrelated heterozygous parents, with the cross being referred to as a two-way pseudo-testcross (Grattapaglia and Sederoff, 1994). Linkage analysis of SDFs in coupling phase with markers placed on the mapping population results in two separate maps, one for each parent based on the male and female sources of markers.

The parental clones TAN and WAG were screened with 82 EcoRI/MseI primer combinations. The combinations resulting in a high number of polymorphic bands were used to genotype the mapping population. For the female parent (TAN) a total of 460 high quality polymorphic fragments were scored, with 330 markers segregating in a 1:1 ratio. Each of these bands was given a

locus name corresponding to the selective base and a number or letter specific for the individual fragment amplified. For the male parent (WAG) 420 polymorphic marker bands were identified, of which 252 segregated in a 1:1 ratio. The male markers were, however, excluded from the present analysis because the total number of simplex markers was too small to provide adequate coverage of the sweetpotato genome.

Analysis of linkage relationships among markers and map construction was performed using MAPMAKER/EXP/version 3.0 software (Lander et al., 1987). A maximum detectable recombination fraction of r = 0.25 with a minimum of odds (LOD) score of 4.0 were used to group loci. This procedure placed 271 AFLP and RAPD markers derived from segregation in TAN into 66 linkage groups (Fig. 1). A total of 59 markers remained unlinked under the high LOD score. A high LOD score was used to avoid false linkage detection. This linkage map, which is currently incomplete, but suitable for our preliminary gene-tagging efforts spanned 1860 cM with an average of four AFLP markers per linkage group and an average length of 28 cM per group.

The linkage map constructed from the 271 linked markers was combined with the virus phenotypic data and entered into QGENE (Nelson, 1997) to conduct the QTL analyses. Phenotypic data for SPCSV, SPFMV, and SPVD (SPCSV and SPFMV) scores were coded as a one (1) for positive virus infection or zero (0) for negative results based on the results of ELISA tests described above. Using QGENE, the relation between phenotype (virus resistance) and marker genotype was investigated using the single-point ANOVA and multiple-regression analyses with two markers in the model. Interval analysis was not used because it requires a reliable genetic map (Nelson, 1997) and the current sweetpotato genetic linkage map is not complete.

Results

Inheritance of resistance to SPVD in promising diallel progenies. Graft inoculation of the 15 families in Uganda resulted in severe SPVD, with no clones being placed in class 1 (symptomless, highly resistant) (Table 2). Only 4 families, WAG x BMA, WAG x KAN, WAG x SOW, and TAN x BMA, had more than one clone with a SPVD severity rating of 2. Highly susceptible genotypes occurred at high frequencies. For example, 85% of the clones were rated as a 4 or 5 among the families. In contrast, highly resistant genotypes occurred at much lower frequencies (<1%, class 2). In all the families, the observed distribution of resistant to susceptible genotypes was skewed towards the highly susceptible categories (classes 4 and 5).

Inheritance of resistance to SPCSV, SPFMV and SPVD predicted by expected theoretical ratios. The expected segregation ratios and the results of the SPCSV and SPFMV resistance evaluations are presented in Tables 1 and 3, respectively. Based on serological tests, the TAN x BMA progeny was classified as 89 resistant: 109 susceptible to SPCSV, and 39 resistant: 159 susceptible to SPFMV. The TAN x WAG progenies were classified as 42 resistant: 45 susceptible to SPCSV, and 50 resistant: 37 susceptible to SPFMV, respectively. Segregation analysis of resistance to SPCSV showed that the ratios of resistant to susceptible progenies in family TAN x BMA fit a 1:1 Mendelian ratio ($\chi^2 = 2.02$, $p = 0.156$), while the TAN x WAG family observed segregation ratios (OSRs) fit 1:1 ratios, respectively ($\chi^2 = 0.10$, $p = 0.335$). For inheritance of resistance to SPFMV, the OSR in family TAN x WAG fit a 1:1 ratio ($\chi^2 = 1.94$, $p = 0.099$, whereas in family TAN x BMA, the OSRs fit 3:1, 4:1 and 5:1 ratios ($\chi^2 = 2.97$, $P = 0.084$; $\chi^2 = 0.01$, $p = 0.092$; $\chi^2 = 1.31$, $p = 0.253$), respectively.

Graft inoculating the two families (TAN x BMA and TAN x WAG) with SPCSV and SPFMV to induce SPVD at CIP resulted in severe SPVD in all the genotypes except two. Repeated graft inoculations of these two genotypes resulted in severe SPVD in all the genotypes similar to results obtained in Uganda.

Molecular genetic analysis of resistance to SPCSV, SPFMV, and SPVD. Kriegner et al. (2001) provide a more detailed description of the AFLP-based map constructed from these progenies. The QTL analyses for this work were based on a preliminary map spanning 66 linkage groups with an average distance of 28 cM per group (Fig. 1). Table 4 shows the results of single-marker linear regression analysis for association of AFLP markers with virus

Table 2. Frequency distribution of sweetpotato virus disease (SPVD) severity scores among promising families generated in a diallel cross (SPVD rating, 1 = no symptoms; 5 = very severe symptoms). Tests were conducted at Namulonge, Uganda, 1999–2000.

	SPVD rating and number of genotypes per class					
Family[z]	1	2	3	4	5	Total
WAG x TAN	0	0	18	37	40	95
WAG x BMA	0	2	18	28	52	100
WAG x KAN	0	3	10	25	60	98
WAG x BWA	0	0	18	25	56	99
WAG x SOW	0	1	24	42	45	112
TAN x BMA	0	1	8	36	39	84
TAN x TOR	0	0	12	53	64	129
TAN x BWA	0	0	16	54	32	102
BMA x KAZ	0	0	7	32	43	82
BMA x TOR	0	0	10	29	51	90
KAZ x KAN	0	0	5	23	60	88
KAZ x BWA	0	0	21	40	41	102
NKA x TOR	0	0	6	20	32	58
NKA x CTA	0	0	9	13	35	57
KAN x CTA	0	0	9	15	32	56
Total no. of genotypes/class	0	7	191	472	682	1352

[z]Cultivar names: 'Bikila Maliya' (BMA); 'Bwanjule' (BWA); 'Camote Tallo' (CTA); 'Kanziga 1' (KAN); 'Kanziga' 2 (KAZ); 'New Kawogo' (NKA); 'Sowola' (SOW); 'Tanzania' (TAN); 'Tororo 3' (TOR); and 'Wagabolige' (WAG).

resistance in the TAN x WAG population. Based on single point regression analyses, two AFLP markers, *e41m33.a*, and *e38m36.u* located on linkage groups 22 and 35, respectively, were highly significant ($p < 0.0001$). Minor QTL linked to SPCSV resistance that explained greater than 10% of the variation in resistance include markers *e35m49.d* ($p = 0.0009$) and *e40m62.d* ($p = 0.0023$) on linkage groups 47 and 32, respectively. For resistance to SPFMV, RAPD marker, *S13.1130* located on linkage group 6 was highly significant ($p < 0.0001$) explaining 72% of the variation in resistance. In addition, QTL located near markers e32m36.f and e33m59.a on linkage groups 33 and 37, respectively each accounted for 10% and 9% of the variation in resistance to SPFMV (Table 4). Addition of a second locus did not significantly increase the percent variation explained by the multiple regression model for resistance to SPCSV or SPFMV, indicating the importance of *e41m33.a* and *S13.1130*. Two markers, e38m36.u and e38m49.a, located on linkage group 35 were associated with resistance to SPCSV and SPFMV, but the QTL for SPFMV ($p = 0.01$) localized near these loci only explained 8% of the variance (Table 4).

No major QTL were observed for resistance to SPVD when the TAN x WAG sibs were infected by SPCSV and SPFMV to induce SPVD, and, the correlations between AFLP markers and SPVD resistance were relatively low (Table 4).

Discussion

To date, we know relatively little about the genomic organization of sweetpotato. Cytological studies suggest that sweetpotato has three subgenomes and that two of them are very similar (Jones, 1965; Shiotani and Kawase, 1987; Ting and Kehr, 1953). Chromosome pairing in sweetpotato is as bivalents with very few quadrivalents. Assuming no double reduction, predominantly hexasomic ratios would indicate that all three genomes are similar, whereas predominantly disomic ratios would suggest that the three genomes are distinct. Jones (1967) indicated that the prevalence of tetrasomic or tetradisomic ratios would support the hypothesis of two similar and one different genome proposed by Shiotani and Kawase (1978). Jones (1967) proposed four possible models (with dosage effects) of inheritance (hexasomic, tetradisomic, tetrasomic, and disomic) of one or more genes for sweetpotato (Table 1). In the present study, disomic segregation was ruled out because we did not observe any linkage in the repulsion phase when we tested the pairwise linkage among original and inverted markers (LOD = 4 and a maximum recombination fraction of 0.4) (Kriegner et al., 2001). As suggested by Wu et al. (1992), this can be used to exclude the hypothesis that cultivated sweetpotatoes arose as an allopolyploid because in disomic polyploids (allopolyploids) repulsion phase linkages should be easily detected. We also excluded testing for segregation ratios for quadruplex inheritance, because they require very large populations for verification, and our populations were small (198 genotypes for TAN x BMA, 87 genotypes for TAN x WAG).

Graft inoculation of progenies with SPVD resulted in severe SPVD in almost all clones of the 15 promising diallel families in Uganda (1352 genotypes). The distribution of SPVD scores in the populations was skewed towards the highly susceptible category (score 5) and very few highly resistant genotypes were observed (score 1 and 2). Similarly, when TAN x BMA and TAN x WAG were graft inoculated with SPCSV and SPFMV to induce SPVD at CIP, severe SPVD was expressed in most of the 285 genotypes tested. The segregation ratios observed in the TAN x BMA and TAN x WAG families were similar to other traits (e.g., flowering vs nonflowering, red stems vs green stems, storage roots vs no storage roots) studied by Poole (1955), and the presence vs absence of β-amylase activity in storage roots observed by Kumagai et al. (1990).

Chi-square analyses of the observed vs expected phenotypic data for the TAN x BMA and TAN x WAG families for resistance to SPCSV and SPFMV predict that resistance to these viruses is controlled by two separate recessive genes. The expected genetic segregation ratios used for the chi-square analyses were based on the assumption of random chromosome segregation, because the occurrence of primary bivalent pairing with limited tetravalents at meiosis allows the assumption of double reduction (Jones, 1967). Although Jones' (1967) models for major gene inheritance were based on testcrosses, tests conducted by Poole (1955) and Kumagai et al. (1990) included testcrosses and other types of crosses (e.g., open pollination and single crosses) similar to the crosses used in the current study. Therefore, interpreting the

Table 3. Segregation of two sweetpotato families for resistance to sweetpotato chlorotic stunt virus (SPCSV) and sweetpotato feathery mottle virus (SPFMV), International Potato Center (CIP), Lima, Peru, 2000. Negative results indicate that virus was not detected using ELISA and subsequent grafting on *I. setosa* indicator plants.

		Genotypes (no.)					
		Elisa test		Hypothesis I		Hypotheses II and III	
Family	Virus	Negative (resistant)	Positive (susceptible)	Ratio tested	χ^2	Ratio tested	χ^2
TAN x BMA	SPCSV	89	109	1:1	2.02	1:1	2.02
						3:1	34.35**
	SPFMV	39	159	1:1	72.73**	1:1	72.73**
				4:1	0.01	3:1	2.97
						5:1	1.31
						11:1	33.47**
TAN x WAG	SPCSV	42	45	1:1	0.10	1:1	0.10
				4:1	54.72**	3:1	31.44**
						5:1	76.98**
						11:1	214.43**
	SPFMV	50	37	1:1	1.94	1:1	1.94
						3:1	14.26**

*,**Significant deviation from ratio tested at $P = 0.05$ or 0.01, respectively.

results obtained in this study by Jones' (1967) inheritance models (Table 1) is valid, pending validation by testcrosses and further molecular investigations.

According to the four models of inheritance in sweetpotato (hexasomic, tetradisomic, tetrasomic, disomic) proposed by Jones (1967) and elucidated by Kumagai et al. (1990), SPCSV resistance in this study followed a simplex hexasomic and simplex tetradisomic/tetrasomic model of inheritance in the TAN x BMA and TAN x WAG families. The inheritance of SPFMV exhibited duplex hexasomic and tetradisomic/tetrasomic inheritance pattern in family TAN x BMA, while it followed a simplex hexasomic and simplex tetradisomic/tetrasomic pattern in family TAN x WAG (Tables 1 and 3).

Dominance effects for SPCSV and SPFMV resistance genes were ruled out because all resistant genotypes would have at least one dominant allele. That would mean that at least 50% of all progenies derived from the resistant parent would be resistant regardless of the model of inheritance (disomic, tetrasomic, tetradisomic, hexasomic). The frequency of genotypes resistant to SPCSV in TAN x BMA was 45.0% and to SPFMV was 19.7%, while the frequency of genotypes resistant to SPCSV in TAN x WAG was 51.1% and to SPFMV 41.5%.

The exact allelic frequencies or dosage effects could not be estimated in this study. The allelic composition and allelic diversity of a particular genotype may be revealed by molecular techniques and studies on segregation of identified major genes. Although our data suggest that the frequency of resistant SPFMV and SPCSV alleles is high, the resistant phenotypes are not as common due to the polysomic nature of sweetpotato and the traits being recessive.

There are many examples of virus resistance being conditioned by recessive genes in plants. Resistance to bean yellow mosaic virus (BYMV) in the bean cultivar, Great Northern 1140, is conditioned by a single recessive gene (Provvidenti and Schroeder, 1973). In barley, resistance to barley yellow dwarf virus (BYDV) may be conditioned by dominant or recessive genes depending on environmental conditions (Jones and Catherall, 1970). In pepper, *Capsicum chinense* Jacq. (PI 159236), resistance to a pepper strain of potato virus Y (PVY pathotype 1–2) is controlled by a major recessive gene (Boiteux et al., 1996). Fraser (1986) gives more examples of recessive resistance genes for plant viruses. Further, some recessive genes may completely suppress virus multiplication conferring immunity (Provvidenti and Schroeder, 1973), while others may confer resistance in a similar manner to those for resistance to SPCSV and SPFMV by reducing symptom severity and virus multiplication, but maintaining virus levels that can still be detected (Catherall et al., 1970).

It has been previously demonstrated that resistance to SPFMV and SPCSV breaks down when the two viruses coinfect the same plant resulting in severe SPVD symptoms (Karyeija et al., 2000; Mwanga et al., 2002; Schaefers and Terry, 1976). The loss of resistance is expressed as occurrence of SPFMV symptoms and high absorbance values, indicating high SPFMV and/or SPCSV titers in the plant, eliminating the resistant category. Highly susceptible sweetpotato cultivars infected with only SPFMV suffer significant yield losses (Clark and Moyer, 1988). But economic losses due to SPFMV are more commonly associated with external cracking and internal corkiness, which render storage roots unmarketable. In contrast, yield losses due to SPCSV are quite high with up to 87% loss in storage root yield being reported in screenhouse experiments (Gibson et al., 1998).

Based on our preliminary AFLP mapping studies, SPCSV resistance was associated with AFLP markers *e41m33.a* and

Table 4. AFLP and RAPD markers associated with ≥ 10% resistance to sweetpotato chlorotic stunt virus (SPCSV) and sweetpotato feathery mottle virus (SPFMV), and ≥ 5% of the variability in resistance to sweetpotato virus disease (SPVD) in the 'Tanzania' x 'Wagabolige' cross. S13.1130 is a RAPD marker, all others are AFLP markers.

Marker	Linkage group	No. of genotypes (n)	F	R^2	*P*
SPCSV					
e41m33.a	22	85	195.09	0.70	0.0000
e40m34.c	22	86	107.87	0.56	0.0000
e38m36.u	35	87	19.24	0.19	0.0000
e36m49.a	35	86	13.63	0.14	0.0004
e38m36.n	35	80	11.41	0.13	0.0011
e35m49.d	47	86	11.83	0.12	0.0009
e39m45.f	35	86	10.06	0.11	0.0021
e40m62.d	32	86	9.91	0.11	0.0023
e39m36.a	47	87	9.23	0.10	0.0032
SPFMV					
S13.1130	6	84	205.83	0.72	0.0000
e39m32.f	6	80	41.02	0.35	0.0000
e36m59.a	6	85	9.21	0.10	0.0032
e32m36.f	33	87	8.90	0.10	0.0037
SPVD					
e32m48.d	4	86	4.43	0.05	0.0383
e35m49.c	4	86	4.43	0.05	0.0383
e32m48.a	24	86	4.22	0.05	0.0431
e38m36.b	24	87	4.10	0.05	0.0460
e39m43.f	24	87	4.10	0.05	0.0460
e41m37.a	27	87	4.10	0.05	0.0460
e35m49.a	32	84	4.04	0.05	0.0477

e38m36.u on linkage groups 22 and 35, respectively. Marker *e41m33.a* explained 70% of the variation in SPCSV resistance in single-marker regression analysis. Because marker *e41m33.a* explained such a high percentage of the variation in SPCSV resistance we speculate that this marker is linked to a major recessive gene for resistance to SPCSV. We propose the name, s*pcsv1* (sweetpotato chlorotic stunt virus 1), for the gene mediating SPCSV resistance.

SPFMV resistance was associated with several markers, but *S13.1130* was by far the strongest QTL explaining 72% of the resistance to SPFMV. Because marker *S13.1130* explained such a high percentage of resistance to SPFMV, it is also likely associated with another major recessive gene. Thus we propose the name s*pfmv1* (sweetpotato feathery mottle virus 1) for the gene mediating resistance for SPFMV.

The QTL analyses support our hypothesis that resistance to SPCSV and SPFMV in TAN x WAG and TAN x BMA is governed by two separate major genes. The additional QTL associated with SPCSV and SPFMV resistance suggest that there may also be one or more minor genes associated with resistance to these viruses. Further, it is possible that some QTL (e.g., marker e38m36.u on linkage group 35) may be associated with resistance to both viruses. These studies represent the first quantitative and molecular genetic investigations of the inheritance of resistance to SPCSV, SPFMV, and SPVD. Results from this work should provide impetus for more investigations using DNA markers for genetic studies of these important viruses and lead to improvements in breeding for resistance to SPCSV, SPFMV, and SPVD in sweetpotato. However, additional tests will be necessary to 1) confirm that SPCSV and SPFMV are indeed governed by two major genes; 2) determine if the two genes present in a common background will suppress SPVD effectively; 3) establish if there are other genes conditioning resistance to SPVD and determine their nature, and 4) determine the exact allelic composition and allelic diversity of the genes conditioning SPVD resistance using molecular techniques and additional inheritance studies.

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