Resistance of Four Inbred Maize Lines to Inoculation with 20 Isolates of *Maize streak virus* from Zimbabwe

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ABSTRACT

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Maize streak is the most widespread and important disease of maize in sub-Saharan Africa. *Maize streak virus* (MSV)-resistant maize germ plasm is available in several countries, but little is known about the stability of its resistance to MSV strains that may differ from one region to another. We used four maize inbred lines (CML312, CML206, CML216, and MSR) known to differ widely for their resistance to MSV in Harare, and evaluated their resistance to 20 MSV isolates collected from the wild during 2 years at locations across Zimbabwe. Maize streak development and symptom severity were evaluated in replicated greenhouse experiments using artificial inoculation via viruliferous leafhoppers. All 20 MSV isolates induced maize streak symptoms on moderately and highly susceptible genotypes by about 1 week after inoculation (WAI). Differences among isolates for severity of early maize streak symptoms were ephemeral, and resistance was accurately differentiated by all isolates by 3 to 4 WAI. Differences in final maize streak symptom scores induced by the isolates were statistically significant, but differed only by a maximum of 7% in 1999 and 19% in 2000. Although genotype, isolate, and genotype by isolate effects for maize streak symptom scores were significant ($P < 0.01$), relative MSV resistance of the genotypes was consistently ranked by all 20 MSV isolates.

Additional keyword: corn

Maize streak is caused by a single-stranded DNA geminivirus (2,9) that is transmitted by several leafhopper species of the *Cicadulina* genus (29). Maize streak was first recorded in 1901 in South Africa (7) and is widely distributed across sub-Saharan Africa (6). Maize (*Zea mays* L.) crop losses due to maize streak range from trace to nearly 100% depending on susceptibility of the maize genotype and timing and extent of infection.

Maize streak symptoms are characterized by the development of chlorotic spots and streaks in longitudinal lines on leaves. The streaks often fuse laterally, resulting in narrow broken chlorotic stripes, which may extend over the entire length of the affected leaves (32). Chlorosis is caused by failure of chloroplasts to develop in the

tissue surrounding the vascular bundles, which results in reduced photosynthesis and increased respiration (19). Severe chlorosis occurs in very susceptible maize cultivars, leading to stunted growth and premature death, poor ear formation, reduced seed setting, and heavy yield losses.

The occurrence of epidemics of maize streak is closely related to the population dynamics of the vector, which in turn is influenced by rainfall, temperature, and availability of alternate host plants (30). *Maize streak virus* (MSV) may be an increasing threat in Africa, especially as maize area increases and agricultural production intensifies, e.g., irrigated winter wheat crops facilitate the over-wintering of both the virus and the vectors. Autrey and Ricaud (1) also suggested that the spread of MSV between crops could be facilitated by successive cropping and the presence of wild grasses, which act as reservoir for both virus and vectors.

Agronomic practices such as crop rotation, timely planting, seed treatment with systemic insecticides, and removal of infected plants have been recommended for reducing MSV incidence. Some of these practices conflict, however, with resource-poor farmers' efforts to cope with the risks of uncertain rainfall. In Southern Africa, for example, many smallholder farmers use multiple planting dates over extended

periods, a practice that results in greater exposure to maize streak, to ensure that at least part of their crop successfully avoids periods of severe drought stress (28). Furthermore, prices of imported chemicals and equipment for spraying often limit the access by smallholder farmers to these MSV control options.

Use of resistant cultivars is probably the most appropriate, cost-effective, and long-term economically viable approach to reducing yield losses to maize streak epidemics (6,20). Resistance in maize to MSV is controlled by a major gene, with two, three, or "few" modifying genes (6,11,22,23,30). Kyetere et al. (13) were first to map this major gene to chromosome 1, and several authors have subsequently confirmed the importance of this chromosome segment, which has a major effect (typically explaining 50 to 60% of phenotypic variation) on resistance to MSV (12,16,22,23,33). CIMMYT (International Maize and Wheat Improvement Center), IITA (International Institute of Tropical Agriculture), and others have produced numerous MSV-resistant maize cultivars. Breeding efforts have depended on availability of vectors together with alternate plant hosts of MSV, or use of artificial inoculation techniques involving mass rearing of *Cicadulina* leafhoppers together with maintenance of suitable MSV inoculum (3). Successful use of mechanical inoculation (vascular puncture, or VPI) has been reported (15), and marker-assisted selection for MSV resistance has been demonstrated (J. M. Ribaut, K. V. Pixley, and S. Munjoma, *unpublished*), but neither method is yet widely used.

Several reports have indicated differences among maize streak symptom severity caused by different MSV strains or isolates. Pinner et al. (25) transmitted MSV isolates found on different hosts from Nigeria, Kenya, South Africa, Mauritius, Ethiopia, and Reunion to maize cv. Golden Batam, and reported that MSV symptoms varied from severe to mild streaks or lesions, sometimes consisting of only a few sparse or small dots on the leaves. Boulton et al. (4) also concluded that "Nigeria mild" (MSV-Nm) and "Nigeria severe" (MSV-Ns) MSV strains could be differentiated based on five distinguishable characteristics that included severity of chlorosis, streak width, streak length,

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latency, and host range. Rodier et al. (27) and Njuguna (21) reported variations in maize streak development, and in some cases resistant germ plasm became susceptible when exposed to different strains of MSV. The most elegant study was probably that of Martin et al. (18), who used *Agrobacterium tumefaciens*-mediated inoculation with constructs for 11 MSV isolates collected in Kenya, Nigeria, Reunion, South Africa, and Zimbabwe, to quantify differences for relative susceptibility of maize genotypes to different MSV isolates. A. O. Diallo and K. V. Pixley (*unpublished*) acknowledged generally small changes in MSV resistance or susceptibility, but were not aware of instances when MSV-resistant maize lines developed at CIMMYT became susceptible when grown across a wide range of sites in eastern and southern Africa.

Our objective was to investigate the variability of MSV resistance by measuring the variation in maize streak symptom development and severity caused by MSV isolates collected from diverse geographical locations in Zimbabwe for a set of four CIMMYT inbred maize lines ranging from very susceptible to very resistant to MSV.

MATERIALS AND METHODS

Experiments were conducted in 1999 and 2000 using four CIMMYT maize inbred lines: CML312 (S89500F2-2-2-1-1-B*5), very susceptible; CML206 ([EV7992#/EVPOP44-SRBC3]#bF37sr-2-3sr-2-4-3-B*2) and CML216 ([MSR:131]-3-3-3-5-B*2), both moderately resistant; and MSR ([MSRXPOOL9]C1F2-205-1(OSU23i)-1-5-B-B-5-B), very resistant to MSV. MSV-infected plants were collected from 8 and 10 geographical locations in 1999 and 2000, respectively (Table 1). The

locations represented four agricultural productivity regions of Zimbabwe, differing mainly in rainfall (approximately 300 to 900 mm annual average), soil quality, and topography (e.g., 600 to 1,700 m above sea level). We considered the 18 MSV isolates collected from diverse agroecological regions would provide an adequate sample of variability, if any, among MSV strains currently infecting maize in Zimbabwe. The infected plants were transplanted into 13-cm-diameter pots and transported to CIMMYT's Harare research station. These plants were maintained individually in screen cages in the greenhouse with maximum temperature of 31°C, minimum of 21°C, and relative humidity between 50 and 70%. In addition to the "wild" MSV isolates collected as described above, in each year we used the CIMMYT greenhouse MSV strain (a mixture of MSV isolates collected regularly near Harare), which is routinely used for field infestations of CIMMYT's breeding nurseries in Zimbabwe.

The four maize lines were each planted in 44 13-cm-diameter pots at the rate of 10 seeds per pot, in soil mixed with compound fertilizer (7N: 14P₂O₅:7K₂O). The seedlings were grown in insect-free cages in the greenhouse and were thinned to six plants per pot when they reached vegetative stage one (about 10 days postplanting) (26), 1 day before infestation.

Nonviruliferous *Cicadulina mbila* leafhoppers were reared on pearl millet (*Pennisetum americanum*) in CIMMYT greenhouses for use as vectors in our experiments. Approximately 300 leafhoppers were introduced to each cage containing young maize plants infected with a different MSV isolate, and were subsequently exposed to MSV for a 48-h acqui-

sition period. Pots with healthy (grown in protective cages with no insect vectors) seedlings of the four experimental genotypes were then placed in a randomized complete block design with four replications inside the cages containing MSV-acquired (viruliferous) insects. In the same process, the leafhoppers were brushed or shaken from the infected maize plants, and these plants were removed from the cages, leaving only the fresh seedlings of the four experimental genotypes together with viruliferous leafhoppers in each cage. After 7 days access feeding period, leafhoppers were killed using 1-naphthyl methylcarbamate (Carbaryl 85wp, Rhone-Poulec Ag. Co., Research Triangle Park, NC, USA) at the rate of 1.5 g/liter to avoid further infestations. Observations of maize streak symptoms were made up to the 24th day after infestation in 1999 and up to the 42nd day in 2000.

The number of plants showing maize streak symptoms in each pot for each isolate was expressed as a proportion, starting on day 5 after infestation. Severity of maize streak symptoms was assessed using a 1 to 9 breeder's scale (1 = no visible symptoms to 9 = very severe symptoms) (modified from Soto et al. [31]) starting on day 10 after infestation. Maize streak symptom scores were collected on three dates in 1999 and seven dates in 2000. Latency period was defined as the time from infestation until maize streak symptoms were visible on maize plants.

Because the cages with the MSV isolates were arranged in the greenhouse in rows and columns, we performed a spatial analysis (autoregressive correlation structure for columns and rows) (8) to test whether the cages may have been differently affected by gradients of temperature, light, and/or other environmental effects. The analysis indicated no significant spatial effect, so further analyses were done using unadjusted means for maize streak symptom scores. Data were analyzed for each maize streak symptom score date using the mixed model in SAS v9.1 (14), considering replication effects as random. Exact probability of significance for comparisons among genotype, isolate, and genotype × isolate means were calculated for pairwise *t* tests.

RESULTS AND DISCUSSION

All isolates induced maize streak symptoms on one or more of the maize lines within 5 days after infestation (DAI) (Fig. 1). There were indications that some isolates had a shorter latency period than others; for example, isolate 13 induced symptoms on 90 to 100% of plants of CML312, CML216, and CML206 by 7 DAI, whereas seven of the isolates had induced maize streak symptoms on significantly fewer plants by this date (Fig. 1A to C). By 17 DAI, all isolates had visibly infected 100% of plants of CML312 and

Table 1. Location and maize cultivar from which *Maize streak virus* (MSV) isolates were collected for experiments conducted in 1999 and 2000

MSV isolate	Collection location	Host maize cultivar ^a
1999		
1	Chiringi Farm, Guruve	SC401
2	Farmer Matarura, Masvingo	SC701
3	Mapanzure, Masvingo	SC501
4	Rupike Irrigation Scheme, Masvingo	R201
5	Nkayi, Matabeleland	SC501
6	Nyandlhovo, Matabeleland	Unknown
7	Samanga Village, Mutasa	R201
8	Nemohuere Irrigation Scheme, Mutare South	SC401
9	CIMMYT greenhouse, Harare	Unknown
2000		
10	Home Eden, Mazoe	PHB3442
11	Mushandike, Masvingo	SC709
12	TRB Cottages, Harare	SC401
13	Mapanzure, Masvingo	SC501
14	Rozva, Bikita	CG4141
15	Don King, Mazoe	PAN338
16	Osborne Dam, Mutare	SC501
17	Dewure, Birchnough Bridge	Unknown
18	Nyanyadzi, Mutare South	Unknown
19	CIMMYT greenhouse, Harare	SC709
20	Nkayi Folk Dev. Coop., Matabeleland North	SC401

^a According to each farmer who allowed an MSV-infected plant to be collected from his or her field.

90 to 100% of plants of CML216, while only one isolate had visibly infected fewer than 90% of plants of CML206. Failure of isolate 15 to induce symptoms on many (22%) plants of CML206 was perplexing and suggests a specific genotype $\times$ isolate interaction effect (Fig. 1C). All 32 other susceptible- or moderately resistant genotype $\times$ isolate interactions resulted in 100% disease incidence, so it seems very unlikely that the 22% of disease-free plants for CML206 by isolate 15 was due to escapes. While we have no alternative explanation for this negative interaction effect on disease incidence, we note that isolate 15 induced the least severe disease symptoms on CML312, CML206, and CML216 (see below) and hence may have been a weak isolate. By contrast to the other maize lines, MSR plants remained largely free of maize streak symptoms; by 42 DAI, six of the 11 MSV isolates had induced symptoms on fewer than 10% of plants,

three isolates on between 10 and 50%, and only two isolates induced symptoms on more than 50% of MSR plants (Fig. 1D). First maize streak symptoms were recorded on maize line MSR from 10 DAI (isolates six and eight) to 42 DAI (isolate one), with the largest number of isolates inducing first maize streak symptoms 21 DAI. These results suggest differences in latency period for the MSV isolates, particularly when infecting the most resistant maize line.

Maize genotype and MSV isolate effects were significant ($P < 0.01$) for maize streak symptom score on all dates, whereas genotype $\times$ isolate interaction effects were significant for all dates except 10 DAI in 1999 (Table 2). Because the four maize lines were selected for this study to represent a range from very susceptible to highly resistant to MSV, the significant genotypic differences were expected. The significant differences among MSV iso-

lates are not necessarily of serious practical concern, unless one or more very severe isolates are found that overcome resistance of the most resistant lines, thereby signaling vulnerability to possible future maize streak epidemics. Most importantly, the significant genotype $\times$ isolate interaction effects merit closer examination to determine if they involve resistance breaking or reversals in MSV resistance of maize genotypes.

At 10 DAI, in both years, greater MSV resistance of genotype MSR relative to the other genotypes was apparent, whereas differences for maize streak symptoms among the remaining three genotypes were generally not significant in 1999 and were significant only for some genotypes and isolates in 2000 (Tables 3 and 4). By 3 weeks after infestation, most pairwise differences for maize streak symptom scores were statistically significant, and all MSV isolates ranked the genotypes simi-

Fig. 1. Percentage of plants ($n = 24$) of four inbred maize lines with visible maize streak symptoms on nine dates following artificial inoculation with 11 *Maize streak virus* (MSV) isolates collected from diverse geographical regions of Zimbabwe in 2000. Standard errors of means are of the same magnitude for all means within a same date; error bars indicate a mean plus and minus its standard error.

Table 2. Summary of F values from analyses of variance for maize streak symptom scores (1 = no symptoms to 9 = very severe symptoms) for four maize inbred genotypes artificially inoculated with 9 *Maize streak virus* (MSV) isolates in 1999 and 11 MSV isolates in 2000, and evaluated on two dates in 1999 and three dates in 2000

		F values					
		1999		2000			
	df	10 d ^a	24 d	df	10 d	21 d	42 d
Isolates, I	8	3.75** ^b	6.75**	10	8.12**	5.37**	9.84**
Rep within I	27	0.54	0.77	33	1.15	1.39	1.06
Genotypes, G	3	68.04**	2,850.94**	3	130.62**	490.15**	1,342.97**
G $\times$ I	24	0.86	2.76**	30	3.00**	3.61**	5.60**

^a Days after artificial infestation with viruliferous leafhoppers.

^b *, ** Statistically significant at $P < 0.05$ and $P < 0.01$, respectively.

larly, with CML312 most susceptible, and an increasing level of resistance for CML206, CML216, and MSR, respectively. By 42 DAI (2000 only, Table 4), all except two pairwise differences were significant, and genotypes were ranked the same by all MSV isolates, in exactly the same order as ranked by all MSV isolates at 21 DAI. Thus, it was clear that maize streak symptoms were not sufficiently expressed at 10 DAI to permit reliably ranking MSV resistance among all of the maize genotypes. More importantly, and without exception, all 20 MSV isolates (9 in 1999 and 11 in 2000) ranked the MSV resistance of the four maize genotypes consistently by 3 weeks after controlled infestation with viruliferous leafhoppers.

Our results seem to disagree with some of the findings reported by Martin et al. (18), who used *Agrobacterium tumefaciens*-mediated transfer ("agroinoculation") of 11 MSV isolates to study maize streak severity on 19 maize genotypes. They found three categories—mild, moderate, and severe—of MSV isolate severity, but all of the mild isolates originated from alternate hosts (not maize) for MSV. Although substantial differences were recorded for maize streak symptom severity (54 to 75% chlorotic leaf area) induced on

three maize genotypes (very susceptible, moderately susceptible, and moderately resistant) by seven moderate or severe MSV isolates, all of these isolates ranked the maize genotypes in exactly the same order from least to most resistant. Whereas a mild MSV isolate only induced maize streak symptoms on very susceptible maize genotypes, one severe and one moderate MSV isolate similarly classified 19 maize genotypes into most resistant to most susceptible thirds, but were not entirely consistent in ranking genotypes within each of those three categories. Thus, our results agree with those of Martin et al. (18) in that different MSV isolates ranked maize streak resistance of maize genotypes similarly.

Agroinoculation may produce different results than *Cicadulina mbila*-vectored inoculation for at least two important reasons: (i) any host-plant genetic or physical barriers to insect feeding, including possible insect nonpreference for a host genotype, are obviated by agroinoculation; and (ii) the constitutive nature of MSV replication by the agroinfectious MSV constructs (17) would obviate any viral replication rate-reducing resistance mechanisms of the host genotype. Peterschmitt et al. (24) reported partial resistance to *C. mbila*-

transmitted MSV involving host resistance to virus multiplication with important reductions in severity of primary and secondary infection, leading to 10- to 90-fold differences in viral particle concentrations in leaves of susceptible versus resistant genotypes. The fact that Martin et al. (18) measured larger differences for maize streak symptoms caused by different MSV isolates than reported herein may have been due to differences in inoculation methods, of which *Cicadulina*-vectored inoculation resembles nature most closely, or may have been due to differences in severity between the MSV isolates used in the two studies.

Although we discontinued these experiments prior to flowering stage, it is worth mentioning that we have commonly observed in artificially inoculated field nurseries that some maize genotypes develop initially severe maize streak symptoms on their lower leaves, and may "out-grow" symptoms, showing much more moderate symptoms on their upper leaves; this implies that there is more than one mechanism of MSV resistance (24,27), and that useful "field-level" resistance may be variously achieved. It is also important to consider that field performance or field-level resistance almost always exceeds

Table 3. Mean and probability of significant differences (for pairwise *t* tests of adjacent means within each row: ns = not significant; * = *P* < 0.05; ** = *P* < 0.01) for maize streak symptom scores (1 = no symptoms to 9 = very severe symptoms) for four maize inbred genotypes (CML312, CML216, CML206, MSR) artificially inoculated with nine *Maize streak virus* (MSV) isolates and evaluated on two dates in 1999

MSV isolate	10 days after infestation				24 days after infestation			
	CML312	CML206	CML216	MSR	CML312	CML206	CML216	MSR
1	4.90 ^a **	2.85 ns	3.44 ^a **	1.00	7.94 **	7.27 **	6.58 **	1.12
2	3.74 ^a ns	3.62 ns	2.50 ^a **	1.00	7.70 ^b ns	7.42 ns	7.04 ^b **	1.15
3	4.24 ns	3.63 ns	3.09 **	1.00	8.07 **	7.28 ns	7.15 **	1.00
4	4.13 ns	3.66 ns	3.47 **	1.00	8.22 **	7.22 **	6.17 **	1.11
5	2.66 ns	2.46 ns	2.54 **	1.00	7.76 **	6.33 *	5.83 **	1.15
6	3.58 ns	2.57 ns	2.61 **	1.00	7.56 **	6.63 ns	6.30 **	1.16
7	4.53 ^a ns	3.59 ns	3.07 ^a **	1.00	7.76 **	6.89 **	5.95 **	1.18
8	3.15 *	1.79 ns	2.18 *	1.00	7.76 **	6.89 **	5.95 **	1.18
9	3.45 ns	2.38 ns	2.31 *	1.00	7.33 ns	7.09 **	6.02 **	1.15

^a Nonadjacent means within row (isolate) and infestation date, and followed by the same symbol were significantly different at *P* < 0.05.

^b Nonadjacent means within row (isolate) and infestation date, and followed by the same symbol were significantly different at *P* < 0.01.

Table 4. Mean and probability of significant differences (for pairwise *t* tests of adjacent means within each row: ns = not significant; * = *P* < 0.05; ** = *P* < 0.01) for maize streak symptom scores (1 = no symptoms to 9 = very severe symptoms) for four maize inbred genotypes (CML312, CML216, CML206, MSR) artificially inoculated with 11 *Maize streak virus* (MSV) isolates and evaluated on three dates in 2000

MSV isolate	10 days after infestation				21 days after infestation				42 days after infestation			
	CML312	CML206	CML216	MSR	CML312	CML206	CML216	MSR	CML312	CML206	CML216	MSR
10	3.50 **	2.50 ns	2.50 **	1.00	7.50 *	6.50 **	4.50 **	1.00	9.00 **	6.25 **	3.75 **	1.50
11	3.50 **	2.25 ns	2.25 **	1.00	7.75 **	6.25 **	4.25 **	2.00	9.00 **	6.00 **	3.75 **	2.50
12	3.00 **	2.00 ns	2.00 **	1.00	7.00 **	4.50 ns	3.75 **	1.50	9.00 **	5.50 **	3.25 **	1.50
13	4.50 ^a **	3.25 ns	3.50 ^a **	1.00	8.00 **	6.25 **	4.00 **	1.50	9.00 **	7.00 **	3.50 **	1.50
14	2.50 ns	2.25 ns	2.00 **	1.00	7.25 **	5.50 **	4.00 **	2.00	9.00 **	6.75 **	3.75 **	2.75
15	2.75 ^b **	1.75 ns	2.00 ^b ns	1.50	6.25 **	3.50 ^b ns	3.00 ns	2.50 ^b	8.50 **	3.50 **	2.25 ns	2.25
16	2.25 *	3.00 *	2.25 **	1.00	7.50 **	6.00 **	4.25 **	1.25	9.00 **	6.75 **	4.00 **	1.50
17	3.25 ^b **	2.00 ^c ns	2.50 ^b **	1.25 ^c	7.00 **	5.25 **	3.75 **	1.25	9.00 **	7.75 **	4.00 **	1.25
18	2.75 *	2.00 **	3.00 **	1.00	8.50 **	4.50 ns	3.75 **	1.75	9.00 ns	8.25 **	4.00 **	2.00
19	2.00 ns	2.00 ns	2.25 **	1.00	5.25 ns	5.25 **	3.75 **	1.75	9.00 **	6.25 **	3.50 **	2.25
20	3.25 ns	3.00 ns	2.75 **	1.00	6.50 *	5.50 **	4.00 **	1.50	8.75 **	6.75 **	4.25 **	1.50

^a Nonadjacent means within row (isolate) and infestation date, and followed by the same symbol were significantly different at *P* < 0.01.

^b Nonadjacent means within row (isolate) and infestation date, and followed by the same symbol were significantly different at *P* < 0.05.

^c Nonadjacent means within row (isolate) and infestation date, and followed by the same symbol were significantly different at *P* < 0.05.

performance in artificially inoculated nurseries or experiments because natural inoculation is unlikely to occur uniformly and at early-seedling stage, as is imposed during artificial inoculation. Thus, we expect field performance (MSV resistance) of the experimental maize inbreds to be better than observed in these experiments even under high disease pressure conditions.

There were statistically significant differences among latency periods for infection of maize genotypes by different MSV isolates, and among final maize streak symptom scores induced by some of the isolates on these four maize genotypes. The genotypes were consistently ranked, however, as resistant or moderately or highly susceptible by all 20 isolates collected across Zimbabwe over 2 years. These results indicated that, as long as effective inoculation techniques were used, any of the 20 MSV isolates would similarly and effectively differentiate among resistance levels of the four inbred maize genotypes evaluated in our study. Our conclusion is consistent with reports of 98 to 100% amino acid sequence similarity among 15 MSV isolates collected from a wide geographical range across Africa and the Indian Ocean islands (5). Our conclusion is also consistent with the hypothesis of Isnard et al. (10), that "from an agronomic viewpoint, this apparent homogeneity of MSV [referring to Briddon et al., 5] suggests that resistant cultivars of maize should be effective in most African countries."

Despite the fact that we obtained consistent results with all 20 MSV isolates we evaluated, the literature suggests that breeding programs should avoid using very mild MSV isolates, such as those collected from alternate host plants such as *Triticum aestivum* and *Setaria* sp. (18). Use of very susceptible check cultivars in maize streak nurseries will provide a useful guide as to virulence of the MSV isolate and the effectiveness of the infestation. On the basis of these experiments with 20 MSV isolates, any very susceptible genotypes with no maize streak symptoms 1 to 2 weeks after inoculation have very likely "escaped" inoculation, or if only mild symptoms are present 3 to 4 weeks after inoculation, have likely been infected by a mild MSV isolate. Although we have no basis to conclude that our results are applicable to all MSV isolates globally, our findings, that maize streak symptom severity was similarly and consistently ranked by all 20 randomly collected Zimbabwean MSV isolates, suggest that MSV resistance, at least for the four inbred lines studied, is widely stable.

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