

## **Mode of Gene Action to Maize Streak Virus in Mid Altitude Inbred Lines CML202 and Osu23i**

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### **Abstract**

*Maize streak virus disease (MSVD) is the most destructive viral disease of maize in Africa causing significant effects on maize yields. Since breeding for durable resistance is an essential trait to improved maize varieties in sub-Saharan Africa, it is important to understand genetic systems conditioning resistance in diverse sources. The objective of this study was to determine the mode of gene action in two maize inbred lines, susceptible CML202 and immune Osu23i. Two sets of six generations ( $P_1$ ,  $P_2$ ,  $F_1$ ,  $BC_{1:1}$ ,  $BC_{1:2}$  and  $F_2$ ) derived from parental and bi-parental crosses of the MSV susceptible parent EM11-133, and CML202 and immune Osu23i were planted in two trials. MSV mean scores and variance rated on individual plants were fitted onto an additive-dominance model. Results indicated that additive gene effect control resistance to MSV in CML202 and Osu23i with the dominance, additive x additive and additive x dominance genic effects playing an important role in selection. The number of effective factors was estimated to be between 2 and 7 genes. Based on frequency distribution of MSV scores in segregating population ( $BC_{1:1}$ ,  $BC_{1:2}$  and  $F_2$ ), two separate genetic systems appear to be involved in control of MSV. Therefore, maize streak virus is controlled through partial resistance in CML202 while complete resistance is responsible in Osu23i.*

**Keywords:** Gene action, maize, maize streak virus, resistance.

### **1. Introduction**

Maize streak virus disease (MSVD; Genus Mastrevirus, Family Geminiviridae) is an important economic disease, which occurs throughout Africa where it significantly affects maize yields (Oworet *et al.*, 2007b).

The disease is incited by a geminivirus that is transmitted by viruliferous six leafhopper species of the genus *Cicadulina* but mainly by *C. mbila* Naudé and *C. storey* (Asea, 2005). In addition to maize, it can infect over 80 other species in the Family Poaceae (Bosque-Perez, 2000; Willment *et al.*, 2001). The disease manifests in a wide range of elevations; from sea level up to elevations of 2000 m (Efron *et al.*, 1989). Yield losses in maize (*Zea mays* L.) due to maize streak range from a trace to virtually 100% when epidemics occur on susceptible open-pollinated varieties and hybrids (Alegbejo *et al.*, 2002; Barrow, 2000; Danson *et al.*, 2006; Kyetere *et al.*, 1999). The MSVD epidemics are frequent in the tropics due to alternate and successive cropping of maize plant hosts and the presence of other hosts such as wild grasses (Mesfin *et al.*, 1995). Infection of the crop by the maize streak virus (MSV) at seedling stage often results in no ear formation, but later infection leads to undersized and poorly filled ears (Kaitisha, 2001). Severe epidemics of MSVD occurred in Kenya from 1988 to 1989 (Njuguna *et al.*, 1990; Theuri & Njuguna, 1988), making the disease a priority biotic stress in the humid lowlands, mid-altitudes and highlands of Kenya (Tefere *et al.*, 2011).

Management MSVD is difficult due to variability of the virus and the susceptibility of the locally adapted maize lines as well as unpredictable vector migratory and survival pattern (Danson *et al.*, 2006; Rodiere *et al.*, 1995). Although various cultural practices and insecticides are effective in managing the vector for MSV (Rose, 1978), the development and deployment of resistant varieties is the most appropriate and cost effective approach to controlling MSVD (Lagat *et al.*, 2008; Danson *et al.*, 2006; Fraser, 1992; Njuguna, 1996). The development of maize germplasm that is resistant to MSV has therefore been the goal of several breeding programs in Africa (Kuiper-Goodman, 1995). These initiatives have resulted in development and release of resistant populations and inbred lines (Barrow, 2000). Ininda *et al.*, 1999 investigated genetic polymorphisms that existed in 10 inbred sources of resistance, namely CML202, CML197, OSU23i, Tzi3, Tzi35, AO76, VHCV, C390 and EM11-133. Sufficient diversity was shown in different sources having genetic polymorphisms at different loci. Two of these lines, CML202 and Osu23i were chosen to be parental inbred lines in the current study based on their diversity in disease resistance. Fulfilling the growing need for increased and sustainable maize production will depend on preventing yield losses and maximizing yield potential of the crop (DeVries & Toenniessen, 2001). Thus, development of hybrids with higher level of resistance to MSV through introgression of genes from resistant donors requires better understanding of the genetics of MSV-disease resistance since the methods of selection to be used and the expected selection gain will be determined by the mode of gene action. This study therefore investigated the genetic systems conditioning resistance to MSV between two commonly used sources, CML202 and Osu23i.

## 2. Materials and Methods

### 2.1 Description of the germplasm

A MSV susceptible inbred line EM11-133 from Kenya Agricultural Research Institute (KARI) and two MSV resistant inbred lines, one MSV tolerant inbred line CML 202 and an MSV immune inbred line (Osu23i) from International Maize and Wheat Improvement Center, (CIMMYT) were used in this study. The parental inbred line EM11-133 was extracted from Embu 11 (EM11) population through pedigree breeding. EM 11 is a seed parent derived from diverse high altitude Kitale maize programme (H621, Inbred lines F and G) and Katumani dry land maize programme (Kat. VI and Kat. V) germplasm. It is a white endosperm dent breeding population maturing in 150 days at Embu in eastern Kenya (Odongo and Bockholt, 1995). The population is an advanced generation of crosses between (H621 X Kat. IV) (FXG) and (Kat V.) (Eberhart, 1989). Although EM11-133 is of desirable agronomic character, it is susceptible to MSV. CML 202 inbred line has white, semi-dent kernels and was developed by CIMMYT, Harare station. It was derived from the bulk population ZSR 923 'S4 bulk' originating from Cameroon 87' in West Africa. Genetic studies have confirmed that the inbred line has a relatively high level of partial resistance to *Exerohilum turcicum* and MSV (Schechert *et al.*, 1999; Welz *et al.*, 1998). The line is late maturing and generally well adapted to growing conditions in the humid mid-altitude zones of eastern and southern Africa. It is widely used in many tropical breeding programs for production of hybrids and new inbred lines due to its excellent combining ability for disease resistance and yield (Schechert *et al.*, 1999). Osu23i refers to Ohio State University line 23, which is immune to MSV (Gibson *et al.*, 2005). The immune line was obtained from CIMMYT as [MSR X Pool9] CIF2-205-1(OSU23I). Glasshouse and field screening conducted in Kenya showed that the line was immune to MSV (Njuguna, 1999). During the long rains of 2006, initial crosses were made between a MSV susceptible parent EM11-133 (P<sub>1</sub>) as a female and each of the sources MSV resistant parents, CML202 and Osu23i as males to generate EM11-133 X CML202 (F<sub>1</sub>) and EM11-133 X Osu23i (F<sub>1</sub>).

The parents (EM11-133, CML202 and Osu23i) and resultant F<sub>1</sub> progenies were grown in two separate crossing nurseries in 2006 short rains (SR) where two sets of six basic generations were formed.

## 2.2 Field trials

Field experiments were conducted at KARI- Muguga in Kenya, at an altitude of 2095 m above sea level, latitude 36° 34'-36° 39'S and longitude 1° 11'-14'E. The experiment was conducted in Complete Randomized Block Design with three replications. Plots consisted of three rows for parents P<sub>1</sub>, P<sub>2</sub> and F<sub>1</sub>, eight rows for backcrosses BC<sub>1:1</sub> and BC<sub>1:2</sub> and twenty rows for F<sub>2</sub>. Each row consisted of 17 plants. Two seeds were planted per hill in a row of 5 m length and thinned to one seedling per hill 2 weeks after emergence. Planting was done in March 2009 where the row-to-row distance was 75 cm while plant-to-plant distance was 30 cm. Standard agronomic practices were observed. Di-ammonium Phosphate (DAP 18:46:0) was applied at planting at the rate of 125 kg per hectare while top-dressing was done using Calcium Ammonium Nitrate (CAN 26%N) at the same rate, 6 weeks after planting. The crop was protected from stalk borer infestation using Beta-cyfluthrin 0.5 g/kg granules, which is a systemic insecticide and a synthetic parathyroid marketed as Bulldock® 0.05 GR. The crop was kept weed-free through hand and occasional spot weeding. Supplemental irrigation was done when needed. Thirty hybrids were planted including susceptible and resistant checks.

## 2.3 Inoculation with Maize Streak Virus

Maize streak virus was transmitted to test maize plants by leafhoppers (*Cicadulinambila*). The *C. mbila* colony was a direct descent from that used by Storey & Howland (1967) and Bock et al., (1974). Populations of non-viruliferous leafhoppers were maintained on clean pearl millet (*Pennisetumamericanum*) grown in glasshouses, at 25 °C. Two days before inoculation, adult leafhoppers were allowed 48 hours acquisition access period (AAP) by feeding on young maize plants exhibiting severe disease symptoms. The infected plants were collected from MSV hot spots within Kiambu County. After AAP, two to three viruliferous leafhoppers were placed in small plastic vials and attached onto the distal portions of the youngest maize leaves. Plants were inoculated at the two-three leaf stage and allowed two days inoculation access period (IAP). Inoculation was done twice at 14<sup>th</sup> and 40<sup>th</sup> days after emergence in order to obtain severe and uniform expression of the disease on all test plants.

## 2.4 Assessment of maize streak severity

The MSV severity was rated on all plants per plot based on a five point scale (1-5) as described by Kim *et al.*, (1989), where: 1 = no or very few streak symptoms on lower leaves (Highly resistant); 2 = light streak symptoms on most leaves below ears with few symptoms above the ear (light infestation); 3 = moderate or mild streak symptoms on most leaves (tolerance); 4 = abundance symptoms on all leaves (about 60-80% of the leaf area - moderate infestation) and 5 = severe streak on all leaves (over 75-80% of the leaf area) is highly susceptible. Mid points (0.5) on the 1 - 5 scale were also included. The disease resistance ratings were done on 58, 72, 87 and 101 days after first inoculation based on visual evaluation of disease symptoms on individual test plants. Area under disease progress curve (AUDPC), which was derived from the severity data, was calculated using the modified formula by Shaner & Finney (1977):

$$AUDPC = \sum_{i=1}^n \left[ \left( \frac{Y_{i+1} + Y_i}{2} \right) (T_{i+1} - T_i) \right]$$

Where: Y<sub>i</sub> = score of visually infected spikelets on the i<sup>th</sup> day; T<sub>i</sub> = day of the i<sup>th</sup> observation; n = total number of observations.

## 2.5 Statistical and Genetic data analysis

Data on MSV ratings were subjected to analysis of variance (ANOVA) using PROC ANOVA procedure of 8<sup>th</sup> edition of Genstat Discovery statistical software (Version 8.1, Lawes Agricultural Trust, Rothamsted Experimental Station, 2006) and treatment means compared using the Fisher's protected LSD test at 5% significance level. Genetic analysis such as approximation of number of genes (alleles) conferring resistance in MSV sources were determined using three methods: (i) Poehlman Method:

$$\text{number of genes} = \frac{(x\bar{P}_1 - x\bar{P}_2)^2}{8} [(\sigma F_2)^2 - (\sigma F_1)^2]$$

Where  $x\bar{P}_1$  = Parent 1 MSV scores means;  $x\bar{P}_2$  = P2 MSV scores means;  $\sigma F_2$  = F<sub>2</sub> MSV scores variance and  $\sigma F_1$  = F<sub>1</sub> MSV scores variance.

(ii) Mather Method:

$$\text{number of genes} = \frac{(\mu\bar{P}_1 - \mu\bar{P}_2)^2}{2} \{2 \times (\sigma^2 F_2) - (\sigma_{B1}^2 + \sigma_{B2}^2)\}$$

Where  $\mu\bar{P}_1$  = parent 1 MSV scores means;  $\mu\bar{P}_2$  = parent 2 MSV scores means;  $\sigma^2 F_2$  =  $F_2$  MSV scores variance;  $\sigma_{B1}^2$  = backcross 1 MSV scores variance;  $\sigma_{B2}^2$  = backcross 2 variance.

(iii) Lande's Method 11:

$$\text{number of genes} = \frac{(\mu\bar{P}_1 - \mu\bar{P}_2)^2}{8} \{2 \times (\sigma^2 F_2) - (\sigma_{B1}^2 + \sigma_{B2}^2)\}$$

Where  $\mu\bar{P}_1$  = Parent 1 MSV scores Means;  $\mu\bar{P}_2$  = Parent 2 MSV scores means;  $\sigma^2 F_2$  =  $F_2$  MSV scores variance;  $\sigma_{B1}^2$  = backcross 1 MSV scores variance;  $\sigma_{B2}^2$  = backcross 2 variance.

Phenotypic, environmental, genetic and additive variances were calculated using the formulae by Warner (1952) and Wright (1968):

Phenotypic variance  $\sigma_p^2 = \sigma_{F_2}^2$ ; Where  $\sigma_{F_2}^2$  =  $F_2$  MSV Scores variance

Environmental

Variance

$$(\sigma_E^2) = \left\{ \frac{\sigma_{P_1}^2 + \sigma_{P_2}^2 + (2 \times \sigma_{F_1}^2)}{4} \right\}$$

Where  $\sigma_{P_1}^2$ ,  $\sigma_{P_2}^2$ , and  $\sigma_{F_1}^2$  are parent 1, parent 2 and  $F_1$  MSV scores variances, respectively.

Genetic variance  $\sigma_G^2 = \sigma_p^2 - \sigma_E^2$

Where  $\sigma_p^2$  and  $\sigma_E^2$  = Phenotypic and environmental variances, respectively

Additive

variance

$$\sigma_A^2 = \left\{ \frac{(2 \times \sigma_{F_2}^2) - (\sigma_{B1}^2 + \sigma_{B2}^2)}{4} \right\}$$

Where  $\sigma_{F_2}^2$ ,  $\sigma_{B1}^2$ , and  $\sigma_{B2}^2$  =  $F_2$ , backcross 1 and backcross 2 MSV variances, respectively.

Analysis of generation means was conducted using the model by Hayman (1958), for each cross pooled across environments. The linear additive model for the  $k^{th}$  generation is  $gk = m + (\alpha)a + (\beta)d + (\alpha^2)aa + (2\alpha\beta)ad + (\beta^2)dd$ , where  $\alpha$  and  $\beta$  are the coefficients for the genetic effects for the particular generation being estimated (Hayman, 1958).

### 3. Results

#### 3.1 Disease reaction among parents, $F_1$ , $F_2$ and backcrosses of two generations

The streak symptoms were observed as early as 14 days after inoculation with the disease progressing in time course. The susceptible parent, EM11-133 showed conspicuous long chlorotic streaks while the tolerant parent CML202 exhibited few and mild streaks. Highly significant ( $P \leq 0.001$ ) differences were observed among the six generations ( $P_1$ ,  $P_2$ ,  $F_1$ ,  $F_2$ ,  $BC_{1:1}$  and  $BC_{1:2}$ ) derived from EM11-133 ( $P_1$ ) and CML202 ( $P_2$ ) for MSV severity scores (Table 1). The intensity of streak symptoms varied on the individual segregating generations. The MSV scores of the susceptible parent EM11-133 rated an average of 3.09 while the tolerant CML202 parent rated 1.94. The MSV scores average rating of  $F_1$  was 2.3, which was 0.22 units less than the mid-parental value (2.52). The  $F_2$  rated an average of 2.5, while the MSV scores of  $BC_{1:1}$  (2.44) and  $BC_{1:2}$  (2.37) were close that of the mid-parental value (2.52). The MSV reactions of the crosses ( $F_1$ ,  $BC_{1:1}$ ,  $BC_{1:2}$  and  $F_2$ ) therefore deviated little or were similar to mid-parent MSV scores. The MSV susceptible parent EM11-133 had the highest AUDPC percentage (55%) compared to 34% for the MSV tolerant parent CML202. The  $F_1$ ,  $BC_{1:1}$ ,  $BC_{1:2}$ , and  $F_2$  had AUDPC of 40 to 44% which were intermediate between those of the parents. Six generations derived from the susceptible and immune parents showed varied ( $P \leq 0.001$ ) reactions to maize streak virus. The susceptible parent EM11-133 had the highest score (3.0) while Osu23i had the lowest (1.0). Score ratings for  $F_1$ ,  $BC_{1:1}$ ,  $BC_{1:2}$  and  $F_2$  were 1.1, 1.5, 1.1 and 1.3, respectively. Similar to the disease ratings, EM11-133 had the highest AUDPC (53.4) while Osu23i had the lowest (16.7). The  $F_1$  generation and three segregating populations ( $F_2$ ,  $BC_{1:1}$  and  $BC_{1:2}$ ) had AUDPC of 18.1, 18.1, 18.4 and 23.0, respectively (Table 1).

**Table 1: Disease severity scores of F<sub>1</sub>, F<sub>2</sub> and backcross populations derived from EM11-133 and CML202; and EM11-133 and Osu 23i parents**

Parents	Generation	Days post- inoculations					AUDPC
		58	72	87	101	Mean	
EM11-133 and CML202	P1	2.90 <sup>c</sup>	3.18 <sup>d</sup>	3.15 <sup>c</sup>	3.12 <sup>d</sup>	3.09 <sup>c</sup>	54.59
	P2	1.99 <sup>a</sup>	1.92 <sup>a</sup>	1.94 <sup>a</sup>	1.92 <sup>a</sup>	1.94 <sup>a</sup>	34.12
	F1	2.25 <sup>b</sup>	2.23 <sup>b</sup>	2.33 <sup>b</sup>	2.39 <sup>b</sup>	2.30 <sup>b</sup>	39.98
	BC1:1	2.35 <sup>b</sup>	2.40 <sup>c</sup>	2.48 <sup>b</sup>	2.51 <sup>c</sup>	2.44 <sup>b</sup>	42.46
	BC1:2	2.31 <sup>b</sup>	2.36 <sup>c</sup>	2.42 <sup>b</sup>	2.38 <sup>b</sup>	2.37 <sup>b</sup>	41.44
	F2	2.41 <sup>b</sup>	2.48 <sup>c</sup>	2.54 <sup>b</sup>	2.59 <sup>c</sup>	2.51 <sup>b</sup>	43.67
	Mean	2.37	2.43	2.49	2.49	2.44	
EM11-133 and Osu 23i	P1	2.70 <sup>c</sup>	3.04 <sup>c</sup>	3.17 <sup>d</sup>	3.14 <sup>d</sup>	3.01 <sup>c</sup>	53.35
	P2	1.01 <sup>a</sup>	1.01 <sup>a</sup>	1.00 <sup>a</sup>	1.01 <sup>a</sup>	1.00 <sup>a</sup>	16.70
	F1	1.07 <sup>a</sup>	1.08 <sup>a</sup>	1.11 <sup>a</sup>	1.09 <sup>a</sup>	1.09 <sup>a</sup>	18.10
	BC1:1	1.41 <sup>b</sup>	1.37 <sup>b</sup>	1.56 <sup>c</sup>	1.59 <sup>c</sup>	1.48 <sup>b</sup>	18.10
	BC1:2	1.04 <sup>a</sup>	1.06 <sup>a</sup>	1.06 <sup>a</sup>	1.05 <sup>a</sup>	1.05 <sup>a</sup>	18.40
	F2	1.30 <sup>b</sup>	1.31 <sup>b</sup>	1.32 <sup>b</sup>	1.34 <sup>b</sup>	1.32 <sup>b</sup>	22.98
	Mean	1.42	1.48	1.54	1.54	1.49	

Means followed by the same letter within the column for each cross are not significantly different ( $P \leq 0.001$ ).

### 3.2 Mode of gene action conferring MSV resistance in inbred lines CML202 and Osu23i

Scale test using Hayman's (1958) additive-dominance model revealed that the additive gene effects were important in the inheritance of MSV resistance in parent CML202. Estimates of dominance gene effects were of low (negative) magnitude. The dominance, additive x additive and additive x dominance genic effects were the most important in selection for MSV resistance (Table 2). Scale test using Hayman's (1958) additive-dominance model showed that the dominance, and additive x dominance gene effects were important in the inheritance of MSV resistance in immune parent Osu23i. The additive x dominance gene effect was more important than additive x additive genic effects (Table 2).

**Table 2: Additive-dominance model (Hayman, 1958) scale test MSV scores for tolerant (CML202) and immune (Osu23i) lines**

Parent	Main and epistemic genic factors	Genetic effects	Variance	Standard error of means	significance test
CML202	Mean	2.565	0.0002	0.014	181.91
	Additive	0.095	0.0004	0.02	4.75
	Dominance	-0.641	0.002	0.048	-13.38
	Additive x Additive	-0.466	0.006	0.08	-5.825
	Additive x Dominance	-2.44	0.004	0.063	-38.85
	Dominance x Dominance	0.462	0.025	0.159	2.906
Osu23i	Mean	1.33	0.0007	0.026	50.378
	Additive	0.518	0.002	0.042	12.392
	Dominance	-0.981	0.021	0.145	-6.762
	Additive x Additive	-0.052	0.018	0.136	-0.382
	Additive x Dominance	-0.557	0.002	0.049	-11.311
	Dominance x Dominance	1.137	0.053	0.230	4.943

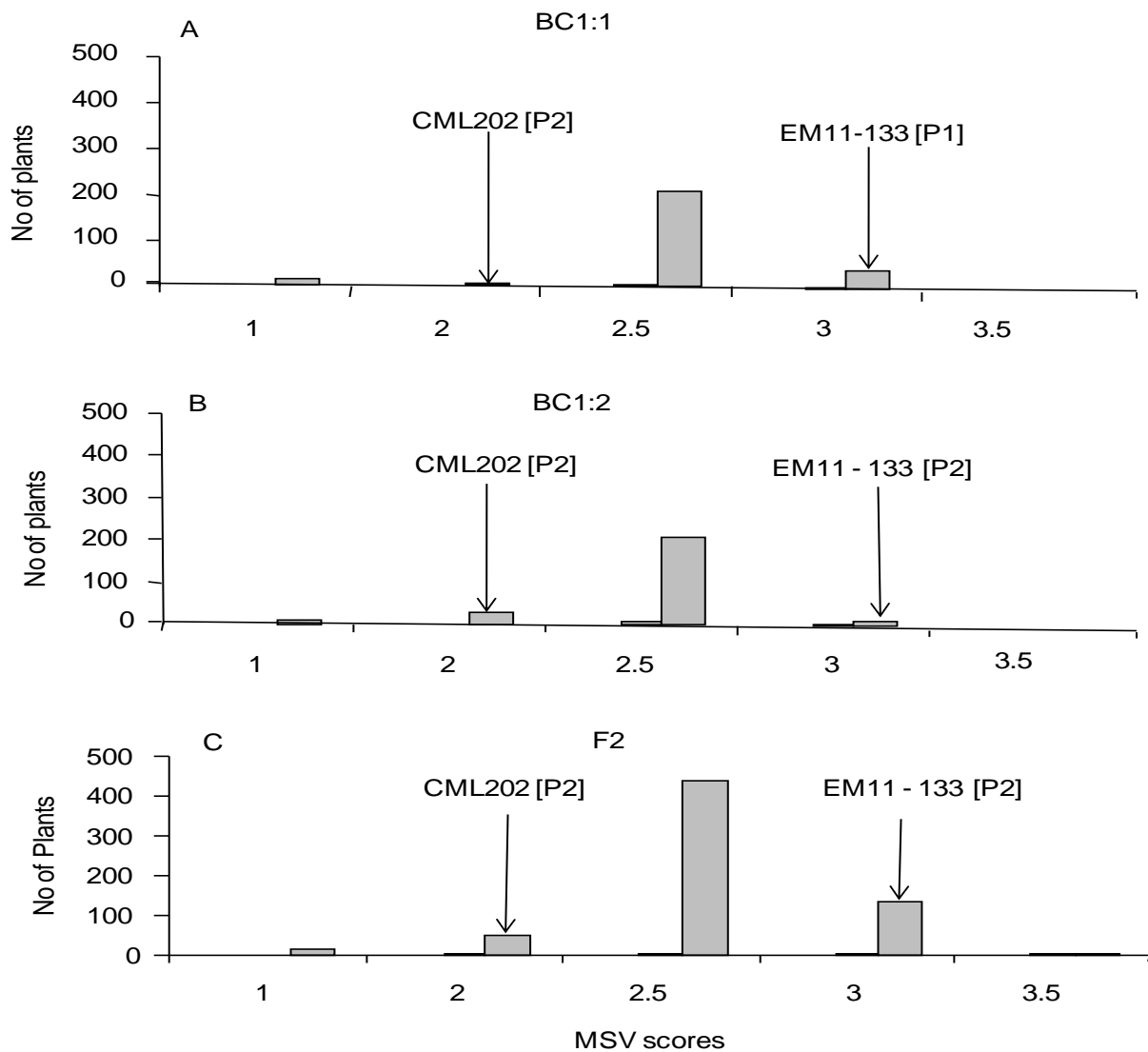
The number of effective factors (genes or "allele") conferring MSV resistance in CML202 ranged from 2 to 7 genes according to the methods by Mather and Lande 11, respectively while the corresponding factors in Osu23i ranged from 2 to 6 based on Poehlman, Mather and Lande 11 methods, respectively (Table 3).

**Table 3: Effective factors and magnitude of variance conferring resistance in CML202**

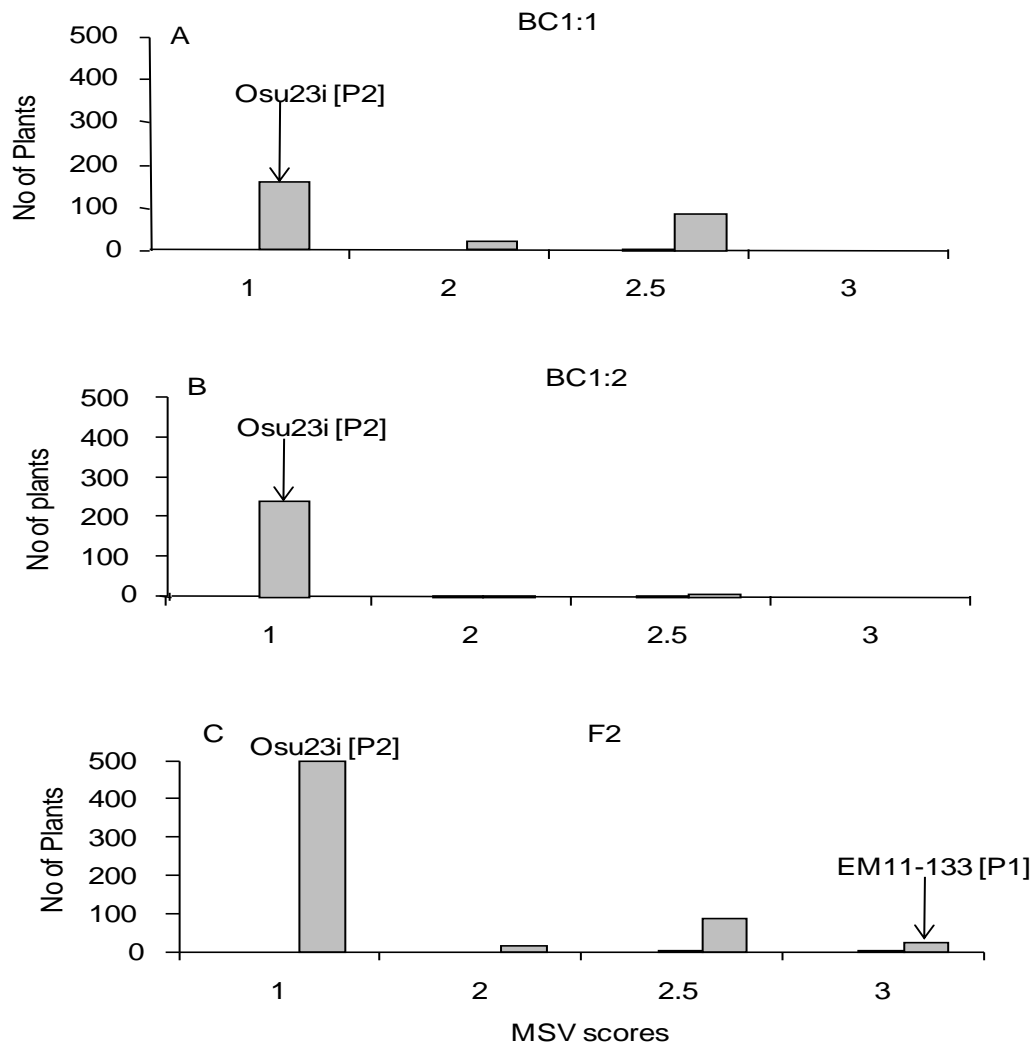
Parent	Method	Number of genes	Variance Type	Magnitude of variance
CML20	Mather	6.947	Phenotypic ( $\sigma^2_p$ )	0.154
	Lande 11	1.736	Environmental ( $\sigma^2_E$ )	0.1385
			Genotypic( $\sigma^2_G$ )	0.0155
			Additive ( $\sigma^2_A$ )	0.026
Osu23i	Poehlman	1.997	Phenotypic ( $\sigma^2_p$ )	0.624
	Mather	6.326	Environmental ( $\sigma^2_E$ )	0.132
	Lande 11	1.582	Genotypic ( $\sigma^2_G$ )	0.492
			Additive ( $\sigma^2_A$ )	0.091

Individual plants of BC<sub>1:1</sub>, BC<sub>1:2</sub> and F<sub>2</sub> segregating populations exhibited a range of symptoms on the scoring scale (Fig. 1). Some plants had scores, which were different from those of either parent. The number of plants falling in different disease categories for each segregating population generally resulted in a uni-modal distribution with high percentage of intermediate symptom ratings (score 2.5), compared to low (2.0) and high (3.0) ratings (Fig. 3A; 3B). Classification of F<sub>2</sub> populations into different categories gave 503 resistant plants rated at less than 2.5; and 138 susceptible plants with a MSV score of more than 3.0 (Fig. 3C). This resulted in a 3.6:1 ratio, which is closer to 3:1 Mendelian segregation ratio.

**Figure 1: Frequency distribution of segregating population derived from MSV susceptible (EM11-133) and immune (Osu23i) parents. (A) BC<sub>1:1</sub>, (B) BC<sub>1:2</sub> and (C) F<sub>2</sub>.**



**Figure 2: Frequency distribution of segregating population derived from MSV susceptible (EM11-133) and immune (Osu23i) parents. (A) BC<sub>1:1</sub>, (B) BC<sub>1:2</sub> and (C) F<sub>2</sub>.**



Analysis of intra-generation distribution of plants exhibiting different symptoms in segregating populations revealed two distributions patterns (Figure 2). For BC<sub>1:1</sub> and F<sub>2</sub> the frequency distribution was bimodal showing quite variable proportions. There was a high percentage of symptom-free plants (score 1.0), intermediate percentage of intermediate symptoms ratings (score 2.5). The BC<sub>1:2</sub> had strictly left skewed unimodal frequency distribution with no completely susceptible lines appearing. Classification of F<sub>2</sub> populations into different categories gave 498 immune plants with a MSV score of 1.0 and 132 plants exhibiting MSV symptoms (Score 2-3). This translates to 3.7: 1 ratio, which is closer to 3:1 Mendelian segregation ratio.

#### 4. Discussion

This study investigated the genetic systems conditioning resistance to MSV between two commonly used sources, the susceptible CML202 and immune Osu23i. The susceptible parent, EM11-133 that served as a check showed conspicuous long chlorotic streaks and had high MSV scores of 3.0 indicating susceptibility. The tolerant parent CML202 rated 2.0 for MSVD and exhibited resistance in form of few and mild streaks indicative of partial resistance (Martin *et al.*, 2001; Rodieret *et al.*, 1995). The immune parent Osu23i was rated 1.0 for MSVD and showed no streaks, except for a single plant – which was perhaps an off type - which showed mild streaks in the field. All other Osu23i were asymptomatic indicative of immunity or complete resistance where virus multiplication was totally prevented (Martin *et al.*, 2001; Rodieret *et al.*, 1995).

However, it should be noted that besides resistance levels, severity of MSV could be influenced by the virus subtype. Martin *et al.*, (2001) reported that subtypes A1, A2, and A5 isolates produce the severest symptoms in maize; subtypes A3 and A6 isolates produced intermediate symptoms, while subtype A4 isolate produced the mildest symptoms. Severe isolates cause earlier symptoms with wider and more chlorotic streaks than the mild isolates (Bosque-Pérez, 2000; Martin *et al.*, 2001). Additionally, some maize varieties known to be resistant to MSV in one ecological zone would show susceptibility to the disease in another, as reported in the island of Réunion (Bosque-Pérez, 2000). There is therefore need to understand the distribution of MSV strains across agro-ecological zones and seasons (Magenya *et al.*, 2008). This information would be invaluable to the on-going maize breeding programmers across Africa.

Unique segregation pattern and expression of resistance to maize streak were observed among the three generations evaluated in current study. The six generations, derived from the susceptible parent EM11-133 and the tolerant parent CML202 showed varied segregation patterns. Disease severity for the susceptible parent EM11-133 was rated 3 compared to the tolerant CML202 which rated 2. The MSV score ratings for F<sub>1</sub>, F<sub>2</sub>, BC<sub>1:1</sub> and BC<sub>1:2</sub> crosses were 2.3, 2.5, 2.4 and 2.7, respectively. These scores were similar to mid-parental MSV scores of 2.5 indicating that co-dominance or partial dominance controlled resistance in the tolerant parent CML202. Storey and Howland (1967) made similar observations that heterozygotes between resistant and susceptible lines reacted to infection in a manner intermediate between the parents. While using Tzi4 as an inbred source of resistance, Kyetere *et al.*, (1999) also found an intermediate reaction for F<sub>1</sub>. A recent study by Gichuru (2008), showed that reactions of the F<sub>1</sub> for disease resistance in crosses generated from five MSV sources either deviated little or there was no deviation from the mid-parental value, showing that MSV resistance in the study material was co-dominant or partially dominant. There are more than one quantitative trait loci, (QTL; Lagat *et al.*, 2008), two or three major gene pairs, with the possible involvement of minor genes (Kim *et al.*, 1989) that control resistance to MSV in the maize germplasm. Danson *et al.*, (2006) identified three loci in one recombinant family, while Lagat *et al.*, (2008) suggested the possibility of MSV resistance being modified by several modifying genes. A major QTL for MSV resistance was found to be on chromosome 1 in CML202 (Welz *et al.*, 1998) a CIMMYT line, D211 (Rodier *et al.*, 1995) a line from Réunion island and Tzi (Kyetere, 1999) a line from International Institute for Tropical Agriculture (IITA). Kyetere *et al.*, (1999) identified MSV 1 as the major resistant gene that controls MSV tolerance in CML 202 and Tzi4.

The area under disease progress curves of the F<sub>1</sub>, F<sub>2</sub>, BC<sub>1:1</sub> and BC<sub>1:2</sub> crosses were intermediate but closer to the AUDPC of the tolerant parent CML202 than the susceptible EM11-133 further showing that there was improvement in resistance among the crosses arising from MSV superior alleles donated by the tolerant parent CML202. This also indicates the importance of dominance over susceptibility for resistance among parental sources. However, the segregation pattern of six generations derived from the susceptible parents EM11-133 and Osu23i exhibited a different pattern compared to that derived from EM11-133 and CML202. The mean MSV scores and AUDPC of the crosses deviated little or were similar to the immune parent Osu23i, hence indicative of complete dominance of the resistant parent Osu23i over the susceptible parent EM11-133. Improvement of MSV resistance observed in the crosses suggests that the immune parent Osu23i donated superior MSV resistance genes, which suppressed severe expression of MSV among the crosses. Rodier *et al.*, (1995) observed similar findings while investigating the mode of gene action in lines extracted from CVR3-C3 (Composite Viroses Resistant 3 –cycle 3) population.

Maize streak virus resistance in the tolerant parental line CML202 was observed to be controlled additively by 2 to 6 genes expressed in a dominant manner while resistance in the immune parental line Osu23i was controlled additively by 2 to 7 genes expressed in a completely dominant manner. These findings concur with the results by Storey and Howland (1967) that a dominant gene controlled MSV resistance in Peruvian yellow X Arkell's Hickory inbred line. The researchers also reported deviations from the theoretical Mendelian segregation similar to those observed in the current study, and attributed it to modifying genes. Therefore, presence and importance of modifying genes cannot be ruled out since they could be contributing to marked variations of symptoms observed among the BC<sub>1:1</sub>, BC<sub>1:2</sub> and F<sub>2</sub> populations. Kim *et al.*, (1989) reported that resistance in IB32 inbred line is controlled quantitatively, mainly additively, with 2 to 3 genes involved. Additive effects are important for resistance (Lagat *et al.*, 2008; Pixley *et al.*, 1997).



## 5. Conclusions

Useful sources of MSV resistance such as CML202 and Osu23i exist which can be utilized by breeders to introgress MSV resistant genes into adaptable high yielding but susceptible hybrids. However, the MSV-immune parent Osu23i resistance should be used over short and medium term in creating inbred lines and formation of hybrids since it offers complete resistance, which can lead to viruses developing resistance. Resistance in CML202 could however be used for long-term breeding but should be backed up by recurrent selection.

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