# Evaluation of the Response of Maize Inbred Lines to Agroinfection by Three Virulent Maize Streak Virus Isolates

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**Abstract**
Originating in Central America and introduced into Africa by the Portuguese in the 16th century, Maize (*Zea mays* L.) has become the most important staple food crop in Kenya and across Africa. However, biotic and abiotic factors threaten its production. Maize streak disease (MSD), caused by Maize streak virus (MSV), is the most important disease in the maize-growing regions. It can reduce maize yields in an infected plant by up to 100 percent, even in high-potential zones. Very few natural sources of resistance/tolerance amongst maize genotypes have been identified; therefore, there is a need to screen more genotypes for resistance/tolerance. This study aimed to evaluate the resistance/tolerance levels of selected maize genotypes. Maize inbred genotypes were screened under controlled agroinoculation conditions using three cloned MSV genotypes on a split plot arranged in a completely randomized design. Disease severity in growth rooms was scored using a completely objective computer-based image analysis system, and the data were subjected to Analysis of variance (ANOVA), with means separated by Duncan's multiple range test. The study found that the Maseno University-developed maize genotypes AB2, ABLEP, and EX44/42-2 possess MSV tolerance levels similar to previously described resistant genotypes such as Pan77 and are potential sources of new MSV tolerance genes in Kenya. Therefore, these lines are important sources of resistance for breeders in the region.
**Keywords:** Agroinoculation, inbred genotypes, maize streak virus, resistance, tolerance

## Introduction

Originating in Central America and introduced into Africa by the Portuguese in the 16th century, Maize (*Zea mays* L.) has become the most important staple food crop in both Kenya and the rest of Africa (IITA & CIMMYT, 2010). Both large and small-scale farmers grow maize in various ecological zones, and it has replaced traditional food crops, including cereals such as Sorghum and Millet. Maize is so important that, in most of Sub-Saharan Africa, poor maize yields are usually linked to food shortages and famine (Thottappilly et al., 1993). Approximately 15 million ha. of maize (*Zea mays* L.) is planted annually in sub-Saharan Africa (Bosque-Pérez, 2000). Ground maize kernels are the most important staple food source for over 100 million people on the continent, accounting for, on average, more than 50 percent of African calorific intake (Shepherd et al., 2007). In Kenya, it accounts for a larger cultivated crop than any other crop and is produced almost everywhere, including in the arid and semi-arid agro-ecological zones (Jayne et al., 2001).

There is a large yield gap between the potential and present yield for Sub-Saharan African tropical highlands (5 and 0.6 tonnes/ha, respectively), in subtropical and mid-altitude zones (7 and 2.5 tonnes/ha, respectively), and in tropical lowlands (4.5 and 0.7 tonnes/ha, respectively (Pingali, 2001). This large yield gap is attributable mainly to biotic constraints (Wambugu & Wafula, 2000). While the major abiotic constraint is drought, causing annual yield losses of about 15 percent (Kamara et al., 2003; Mkuhlani et al., 2024; Wang et al., 2025), secondary

abiotic constraints are soil, nitrogen, and phosphorus deficiencies (Nziguheba et al., 2002; Salam et al., 2024; Whitbread et al., 2004).

Biotic factors that reduce maize yields in the field in Africa are stem-borers, armyworms, Striga (the parasitic weed), and diseases such as leaf blight, rusts, stalk and ear rots, smuts, and systemic foliar diseases (Alegbejo et al., 2002; Beyene et al., 2017). One such systemic foliar disease is maize streak disease (MSD), caused by the geminivirus, *Maize streak virus* (MSV). Maize streak virus is by far the most important and widespread microbial constraint to maize production in Africa (Bosque-Pérez, 2000; Fajemisin et al., 1976; Martin 2021; Thottappilly et al., 1993)where it causes yield losses in individual plants ranging from negligible to almost 100 percent (Alegbejo et al., 2002; Kyetere et al., 1999).

The overall economic and social costs of MSD remain almost completely unknown (Martin & Shepherd, 2009). Whereas Lagat et al. (2008) and Kyetere et al. (1999)justify the development of MSD control strategies, stating that MSD causes up to 100 percent yield losses in individual severely infected maize plants, MSD has never destroyed anywhere near 100 percent of an entire country's maize yields (Chivasa et al., 2021; Martin & Shepherd, 2009).

However, during the most severe epidemics, the disease will frequently cause 100 percent yield losses in individual fields, depending on the virus virulence, crop susceptibility, and the stage of infection (Afram et al., 2024). At the 2$^{nd}$ leaf stage, when a susceptible crop is infected by a virulent strain, it will not yield any seed, and hence it will suffer 100 percent loss, while at the 10$^{th}$ leaf stage it will produce 25 percent less yield than an uninfected plant (Bock, 1982). Martin and Shepherd (2009) suggest that it is, however, useful to think of the economic cost of MSD in terms of the economic benefits that would be gained by its eradication. These benefits would include lower, more stable maize prices for maize consumers and increased income for small-scale farmers.

It has been estimated that modest use of 'immune' maize genotypes in countries like Kenya, where the yield loss is close to 1 million metric tonnes per year (Wambugu & Wafula, 2000) would result in maize yield increases worth between US$ 4M - US$ 15M per year to mainly subsistence farmers, and maize price decreases of 0.7-2.6 percent, saving consumers between US$ 6M - US$ 17M per year (Andreu et al., 2006).

Management of MSD is difficult partly due to the genetic variability of the virus, the susceptibility of locally adapted maize lines, and the unpredictability of vector migratory and survival patterns (Danson et al., 2006). Sporadic maize streak disease epidemics continue to occur in much of Africa, and the resulting crop losses can be devastating (Oyeniran et al., 2024). Various cultural practices have been suggested to control it. According to Gorter (1953), using barriers of bare ground between early and late-planted maize fields reduces leafhopper movement and the subsequent spread of MSV. Adjusting planting dates to avoid infestation of young plants, avoiding maize plantings downwind from older cereal crops, and the use of crop rotations also minimize invasions by viruliferous leafhoppers (Rose, 1978). Crop rotation and intercropping (Page et al., 1999) are also possible cultural control strategies which might disrupt leafhopper mating behaviour (Khanna et al., 2024).

Martin and Shepherd (2009) questioned the feasibility of cheap and accessible cultural control strategies given the unpredictability of MSD epidemiology. They argue that without accurate weather forecasts and a reasonably predictive MSD epidemiological model, it is pointless to plan planting dates with the hope of avoiding infestation of young MSD-susceptible plants by migrating viruliferous leafhoppers. Cultural strategies that do not require advanced disease-forecasting infrastructure include fertilizers and irrigation. By ensuring strong and healthy plants that are better able to resist pathogens and are potentially less appealing to leafhoppers (which are attracted by yellowing), fertilizers and irrigation may be a viable means of reducing MSD infection rates (Magenya et al., 2010). However, it remains to be demonstrated whether this is the case. Also, since plants are most susceptible to infection and experience their heaviest yield losses when infected young, rapidly maturing maize varieties

should perhaps be favoured in areas where MSD is endemic. Again, however, the value of such a control strategy remains to be scientifically demonstrated.

While insecticides such as Carbofuran granules applied as seed dressing have been reported to reduce MSD incidence (Drinkwater et al., 1979) and have been used to effectively control leafhopper vectors (Barrow, 1992; Rose, 1978), absolute protection against MSD is not, however, achievable unless the insecticide is able to kill all the leafhoppers on contact before they are able to feed on maize (Martin & Shepherd, 2009). Furthermore, the Food and Agricultural Organisation (Allen et al., 1998) and the World Health Organisation (WHO, 1986) have documented a list of concerns associated with the widespread application of pesticides in African maize cropping systems.

Pesticide poisoning affects a million people in Africa annually, with 20,000 cases resulting in death (WHO, 1986). Similarly, in most resource-constrained African farming sectors, pesticide use costs are almost prohibitive. Appropriate insecticide use will continue to play an important role in controlling maize viral diseases and their vectors. Nevertheless, non-chemical alternatives remain the most cost-effective, safe, and environmentally appropriate approaches for tropical, resource-constrained farmers (Pingali, 2001).

Resistance breeding is considered to be the most practical solution for the control of MSV (Danson et al., 2006; Mushayi et al., 2025). Resistance in maize was first reported in the variety Peruvian Yellow in 1931, in South Africa by Fielding (1931). Later, tolerance was found in other maize genotypes and has been utilized to develop a variety of MSV-resistant genotypes such as the hybrid, PxH (Soto et al., 1982). Resistance to MSV has also been identified in East Africa (Storey & Howland, 1967). In West Africa, the International Institute of Tropical Agriculture (IITA) in Nigeria found evidence of resistance in its TZ-Yellow genotype. On the island of Reunion, at the Institut de Recherche Agronomique Tropicale (IRAT), resistance has been detected in the genotype, Revolution (Soto et al., 1982). Finally, in Southern Africa, immunity to MSV has been detected in various inbred lines and in experimental hybrids (Barrow, 1992).

Resistance to MSV in many of the IITA open-pollinated varieties and hybrids has manifested itself as reduced symptom severity combined with low virus incidence in the field (Soto et al., 1982). Moreover, when they do become infected, highly MSV-resistant maize genotypes have been shown to have low virus titres (Asanzi, 1991). Yield comparisons between infected and uninfected plots of resistant and susceptible maize genotypes have unequivocally demonstrated the superiority of resistant genotypes (Bosque-Perez et al., 1998).

Reduction in yield due to MSV is a function of the age of plants at the time of infection, with younger plants sustaining greater reductions than older ones (Bosque-Perez et al., 1998; Guthrie, 1978; Mzira, 1984; van Rensburg, 1981). Moreover, yield loss is a function of the relative level of susceptibility or resistance of the maize variety (Barrow, 1992; Bosque-Perez et al., 1998). Varieties have been found to be differently affected by MSV in relation to the growth stage, with disease incidence and severity being markedly reduced for the most resistant varieties, which yield well even when infected young (Bosque-Perez et al., 1998).

Maize varieties that combine tolerance to MSV with other desirable characters have been developed both at IITA and at the Harare station of the International Maize and Wheat Improvement Centre (CIMMYT). Many high-yielding varieties traditionally grown in various African countries have been upgraded with MSV resistance at IITA in cooperation with CIMMYT and other African MSV resistance breeding programmes (Efron et al., 1989).

Breeding lines of maize with tolerance to MSV have also been independently developed by IRAT in Reunion (Rodier et al., 1995). Several national programmes in Africa are breeding for resistance to MSV, and in Kenya breeding for resistance involves the collection of diverse sources of resistance and assessing the combining abilities of the inbred lines (Gichuru et al., 2011) to develop high-yielding, disease-resistant maize multilines.

Resistance to MSV in IITA maize germplasm is controlled by two or three major genes, with the possible involvement of additional minor genes (Kim et al., 1989). Analysis of the genetic control of resistance in the MSV-tolerant line Tzi 4 was conducted by Kyetere et al. (1999). They reported a single major gene (designated *msv* 1) controlling tolerance to MSV in Tzi 4. The gene was located on the short arm of chromosome 1 and was tightly linked (3 cM distance) to the molecular marker locus npi262.

Martin et al. (1999) formulated a technique to assess the pathogenicity of MSV isolates and the differential response of maize genotypes. Quantification of chlorotic leaf areas through image analysis allowed differentiation of levels of virus resistance not otherwise quantifiable (Martin et al., 1999). Furthermore, Danson et al. (2006) established that a conventional maize breeder requires the use of molecular markers (simple sequence repeats) in order to improve selection intensity and maximise genetic gain. In the study, three genetic markers: p-bnlg 1811, p-umc 1144 and p-umc 1917 were used against three different maize family lines (114, 118 and 141) with the primers targeting three loci of chromosome 1 to reveal the genetic MSV resistance trait. It is, however, widely thought that the best MSV resistance so far achieved is polygenic (Lagat et al., 2008; Ladejobi et al., 2018; Patil et al., 2021), with, for some unknown reason(s), the traits not being transferable with agronomically favoured genes (Martin & Shepherd, 2009).

The development and utilisation of resistant commercially favourable maize genotypes would be the most attractive MSD control option (Martin & Shepherd, 2009). It is however expensive and time-consuming to develop such maize genotypes (Danson et al., 2006). While significant strides have been made in breeding for other desirable traits such as high yield and environmental adaptability, most commercially available maize hybrids are, at best, tolerant to MSV. This has been occasioned by the challenge of removing the undesirable traits associated with complex multigenic MSV resistance (Martin & Shepherd, 2009).

Genetic engineering therefore offers an alternative to conventional MSV resistance breeding strategies (Shepherd et al., 2007). Genetic engineering enables the direct transfer of any genes between any species and also the construction and transfer of genes that do not exist in nature (Gadani et al., 1990). It therefore becomes easier to design virus resistance strategies that, while working against the broadest possible range of virus strains, will also be durable in the wake of persistent virus evolution. A number of strategies have been developed to introduce geminivirus resistance into economically important crops (Shepherd et al., 2007). Pathogen-derived resistance (PDR) has shown particular promise as a technique for producing MSV-resistant maize (Mushayi et al., 2025).

Maize streak disease is the most economically significant viral constraint on maize production in Sub-Saharan Africa, causing lost income and higher maize prices due to poor yields. Unfortunately, researchers have identified very few sources of resistance or tolerance to MSV in cultivated maize. Therefore, more maize genotypes must be screened for MSV tolerance to identify new sources of MSV tolerance adapted to Kenyan environments. This study quantitatively evaluated the responses of selected maize genotypes to three MSV-A variants from Kenya and Mozambique, under controlled agroinoculation conditions.

## Materials and Methods

### Maize Genotypes and Virus Strains

Seeds of the inbred maize lines, including sweet corn varieties: Golden bantam from Millington Seed (USA), Starr 7714 from Starke Ayres (South Africa), PAN77 from Pannar Seed Co. (South Africa), CML389 from the International Maize and Wheat Improvement Center (CIMMYT, Mexico), and AB2, ABLEP, 44/42-2, MSMP1/MSMP2 FW, and EX-CIMMYT, all from Maseno University, were used in agroinoculation experiments. Agroinfectious clones of field-isolated MSV variants currently circulating in Africa: (MSV-A [-KE-MtKA-1997], Genebank accession number: AF 329885; MSV-A [-MZ-Bil6-Bet25-2007], Genebank accession number:

HQ693340; and MSV-A [MZ-Pem2-Moz37-2007], Genebank accession number: HQ693363) (Table 1) were provided by DP Martin at the University of Cape Town. Preliminary studies indicated that these MSV isolates produced symptoms in sweet corn ranging from very severe to moderately severe (D. P. Martin et al., *unpublished data).* A. *tumefaciens* C58C1 (pMP90) was used to deliver agroinfectious constructs during agroinoculation experiments.

**Table 1: MSV-A Genotypes Selected for Evaluation in Maize Genotypes**

| Name of Isolate | Genbank accession number | Restriction enzyme site used for cloning | Host Species | Sample date/ year | Latitude | Longitude | Country |
|---|---|---|---|---|---|---|---|
| MSV-A -KE-MtKA-1997 | AF329885 | BamHI | Maize | 1997 | -0.439743 | 37.127454 | Kenya |
| MSV-A -MZ-Bil6-Bet25-2007 | HQ693340 | KpnI | Maize | 2007 | -25.931800 | 33.407133 | Mozambique |
| MSV-A -MZ-Pem2-Moz37-2007 | HQ693363 | BamHI | Maize | 2007 | -13.072850 | 40.394833 | Mozambique |

**Agroinoculation**

To permit efficient replicative release upon agroinfection, all constructs contained one full MSV genome bounded by two full, large intergenic region sequences (Schnippenkoetter, 1998). Agroinoculation was performed according to Grimsley et al. (1987) with the following modifications:

1) Standardized inoculum was obtained from actively growing *A. tumefaciens* cultures with an optical density at 600 nm of 0.4 OD units and was concentrated 10-fold by low-speed centrifugation (2000 rpm in a microfuge). After two washes with sterile distilled water, tubes were stored at room temperature for no more than 30 minutes before injection.
2) 3 days old seedlings were inoculated.
3) 2-5 µl of inoculum were injected 1 mm beneath the coleoptilar node of seedlings using a 25-µl Hamilton syringe (Hamilton, Bonaduz, Switzerland), with its beveled tip inserted to a standardized depth of 1.5 mm (aided by a specifically designed collar permitting penetration of only 1.5 mm of the tip into the coleoptile.
4) Only seedlings with a coleoptile between 15 and 30 mm in length were selected for injection. Agro-infection experiments involving specific constructs were carried out on groups of twenty plants of each maize genotype.

**The Experimental Design**

The experimental design was a split-plot arrangement in a Completely Randomized Design (CRD), with the inoculum (virus isolates) as the main plot factor and the inbred lines as the subplot factor, replicated three times.

A group of 20 seedlings of each maize variety/line was injected with each MSV isolate, while six seedlings of each genotype were injected with sterile distilled water, which served as uninfected controls. Seedlings were grown under near-uniform conditions in a plant growth room maintained at 27°C, 60 percent relative humidity, with 16 hours of light per day.

**Data Collection**

Disease severity was measured in terms of: (i) percentages of agroinoculated plants that became infected; (ii) percentages of leaf areas (leaves 2 through 6) covered by chlorotic lesions in

infected plants; and (iii) leaf sizes of infected plants relative to uninfected control plants. The proportion of plants showing symptoms was determined at 3-day intervals between 5 and 14 days after injection. For each MSV isolate-maize genotype combination, these measurements were integrated into an infection rate value (IR) calculated as the mean percentage of infected plants observed at the four assessment times.

Percentage of leaf area covered by chlorotic lesions in symptomatic plants was estimated for leaves two through six using a previously described microcomputer-based image analysis technique (Martin & Rybicki, 1998). The image analysis protocol required a digital image capture device (in this case a HP Scanjet 5590P flatbed scanner set at 300 dpi) and analysis software developed in the MCB laboratory (available online free of charge from the University of Cape Town). The percentage of chlorotic areas of leaves two and three was assessed 15 days after agroinoculation; the percentage of chlorotic areas of leaves four, five and six was assessed 22, 29, and 35 days after agroinoculation, respectively. For each MSV isolate-maize genotype combination, the mean percentage of chlorotic area of the six leaves was used as a representative measure of chlorosis.

**Statistical Analysis and Evaluation of Maize Genotypes Resistance to MSV**

The data collected were subjected to analysis of variance (ANOVA) using MSTAT-C program (Freed et al., 1991). Parameters analysed included: leaf area relative to the standard, percentage chlorotic area of the leaf, relative average streak width, and yellowness induced by the MSV variants. The means were separated by Duncan's Multiple Range Test (DMRT)

## Results and Discussion

**Relative Virulence of Different Isolates**

The initial infection symptoms observed were characterised by small circular, chlorotic spots on the younger leaves of maize plants, which became continuous longitudinal chlorotic streaks along the veins of the leaf laminae. Two weeks after inoculation, all successfully infected plants displayed symptoms. Only 41 percent, 60 percent, and 65 percent of plants inoculated with MSV-A_KE_MtKA-1997, MSV-A [MZ-Pem2-Moz37-2007], and MSV-A [MZ-Bil6-Bet25-2007], respectively, became infected. Although MSV-A [MZ-Bil6-Bet25-2007] had a higher infection rate than the other two isolates, MSV-A [MZ-Pem2-Moz37-2007] appeared to be the most virulent of the isolates (Table ***2***2, Figure 1) besides inducing the highest stunting compared to the other two isolates, which were not significantly different.

**Table 2: Means of Various Variables (Leaf Area Relative to Standard, % Chlorotic Area, Relative Average Streak Width and Yellowness) to Compare the Virulence Levels of Different Isolates**

| Isolate | Leaf area relative to standard | % Chlorotic area | Relative average streak width | Yellowness |
|---|---|---|---|---|
| Control | 2.36259a* | 4.6438c* | 0.0268022b* | 0.575319d* |
| MSV-A [KE-MtKA-1997] | 1.24601b | 25.0930b | 0.0381214a | 0.611306b |
| MSV-A [MZ-Bil6-Bet25-2007] | 1.9687b | 35.7833a | 0.0385639a | 0.598686c |
| MSV-A [MZ-Pem2-Moz37-2007] | 1.01150c | 36.7338a | 0.0387624a | 0.619075a |
| **Mean Value** | **1.6472** | **25.563475** | **0.0355625** | **0.601097** |
| **CV (%)** | **25.54** | **8.55** | **26.0** | **5.0004** |

* Means within a column followed by the same letter are not significantly different according to DMRT ($p \leq 0.05$).

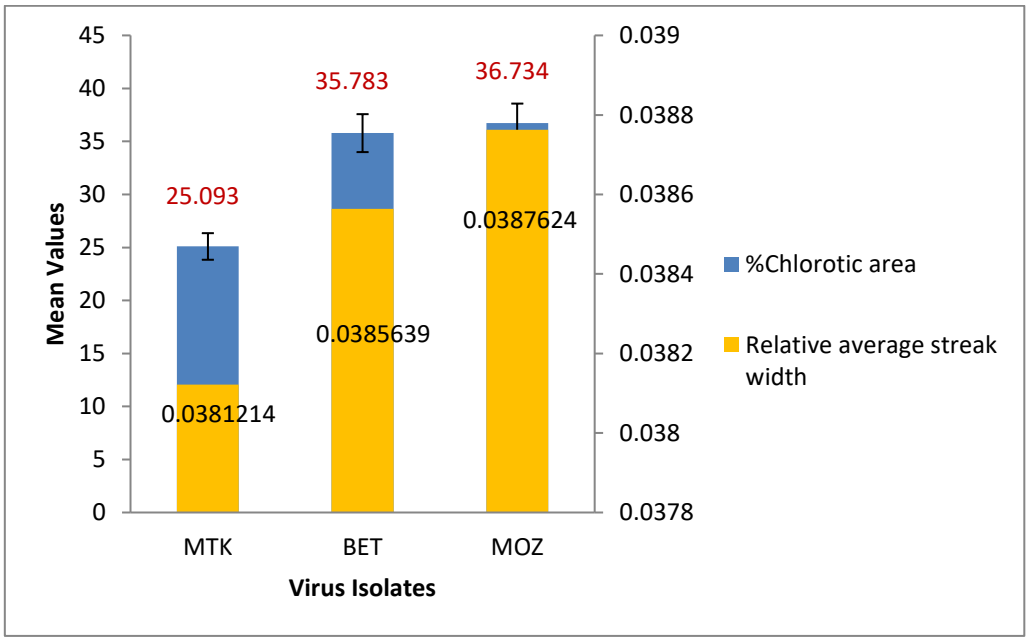



**Fig. 1: Cumulative Mean Values of Relative Average Streak Width and %Chlorotic Area for Different Isolates. The Error Bars Represent 95% Confidence Intervals of the Means**

**The Response of Maize Genotypes to Infection**

Studies on plant responses to pathogens are particularly useful for establishing tolerance to different strains and for breeding plants with elevated tolerance (Rodier et al., 1995). Here, maize genotypes differed significantly across measures of virus virulence (leaf area relative to standard, % chlorotic area, relative average streak width, and yellowness) (Table 3). With respect to % chlorotic area (Figure 2), the sweet corn variety GB had the highest % chlorotic area, followed by the sweet corn variety Starr 7714, MSMP, and CML, which was not significantly different from EXC. AB2, 44/42-2, and PAN then followed in that order. This

pattern is similar to what Martin et al. (1999) observed in sweet corn and PAN varieties' tolerance to MSV isolates: MSV–Mat A, MSV-Kom, and MSV-Set. The same trend was largely mirrored by the relative average streak width, with just a few deviations.

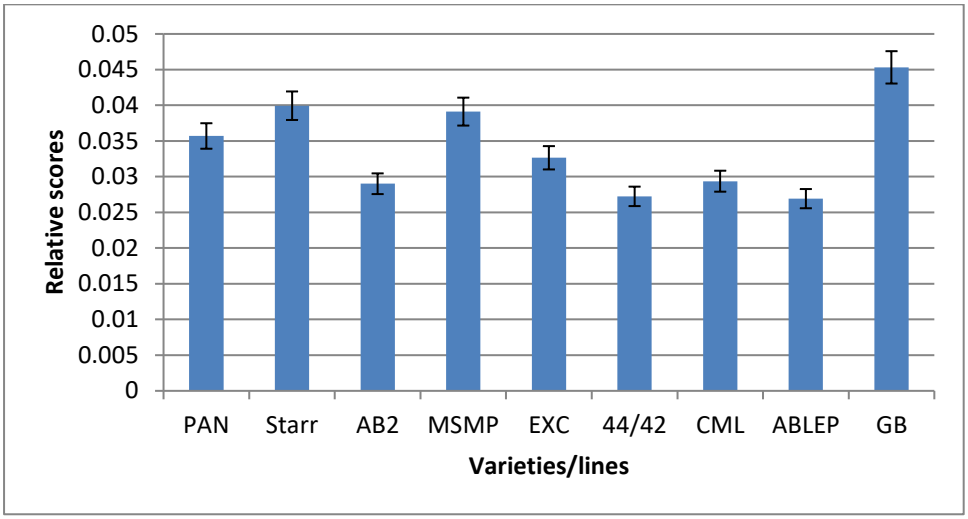



**Fig. 1: Relative Scores on the Streak Width Induced on Varieties as a Result of Infection by the MSV Isolates. The Error Bars Represent 95% Confidence Intervals of the Means**

It is expected that the higher the mean value of percentage chlorotic area, relative average streak width, and yellowness, the more MSV-susceptible a maize genotype is, and vice versa. Conversely, it is expected that the higher the relative leaf area, the more MSV-tolerant a genotype is, and vice versa (Barrow, 1992; Martin & Rybicki, 1998; Rodier et al., 1995).

Based on the percentage of chlorotic area in Table 3, the varieties can be grouped into three categories: a relatively susceptible group consisting of sweet corn variety GB, sweet corn variety Starr 7714, and MSMP (Group I); a moderately tolerant/resistant group consisting of CML, EXC, and AB2 (Group II); and a highly tolerant/resistant group consisting of PAN 77, 44/42-2, and ABLEP (Group III). The results align with the infection frequency, with MSMP and CML exhibiting over 90 percent infection frequency and ABLEP showing less than 40 percent infection frequency.

**Table 3: Comparison of Maize Genotypes by Means of Various Variables (Leaf Area Relative to Standard, % Chlorotic Area, Relative Average Streak Width and Yellowness)**

| Variety/line | % Leaf area reduction | % Chlorotic area | Relative average streak width | Yellowness |
|---|---|---|---|---|
| PAN 77 | 22.12h* | 17.856f* | 0.035705c* | 0.612790c* |
| Starr 7714 | 44.65e | 33.546b | 0.039945b | 0.561991h |
| AB2 | 55.31cd | 21.340e | 0.029015d | 0.596311e |
| MSMP | 61.73b | 31.198c | 0.039119b | 0.611335c |
| EXC | 52.46d | 27.446d | 0.032651c | 0.602856d |
| 44/42-2 | 71.96a | 17.425f | 0.027246d | 0.575282g |
| CML | 42.94f | 25.476d | 0.029369d | 0.618534b |
| ABLEP | 56.92c | 18.606f | 0.026925d | 0.584546f |
| GB | 40.82g | 35.954a | 0.045319a | 0.624597a |
| **Mean** | **49.88** | **25.4274** | **0.0339** | **0.5987** |
| **CV (%)** | **19.66** | **9.4343** | **26.28** | **5.0004** |

* Means within a column followed by the same letter are not significantly different according to DMRT ($p \leq 0.05$).

There was a strong relationship between percentage relative leaf area reduction (in which resistance is best reflected) and susceptibility/tolerance of maize genotypes (Isnard et al., 1998) (Fig. ***2***). Pan 77, regarded as a highly resistant maize genotype, exhibited the least percentage leaf area reduction, while 44/42 expressed the highest stunting. This was inconsistent with the percentage chlorosis induced in the genotypes, since 44/42-2, together with Pan 77, had the least accumulated chlorosis.

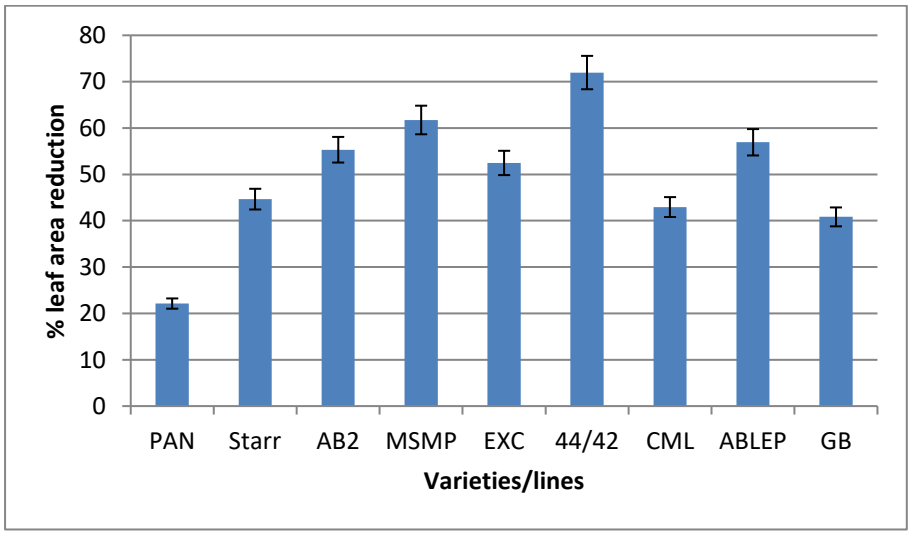



**Fig. 2: Percentage leaf area reduction induced on varieties as a result of infection by the MSV isolates relative to their controls. The error bars represent 95% confidence intervals of the means**

However, the observed means of yellowness (Figure 4) provided little information, as they were not consistent with the other parameters used to measure resistance in the individual maize genotypes. This may be due to genetic differences among varieties.

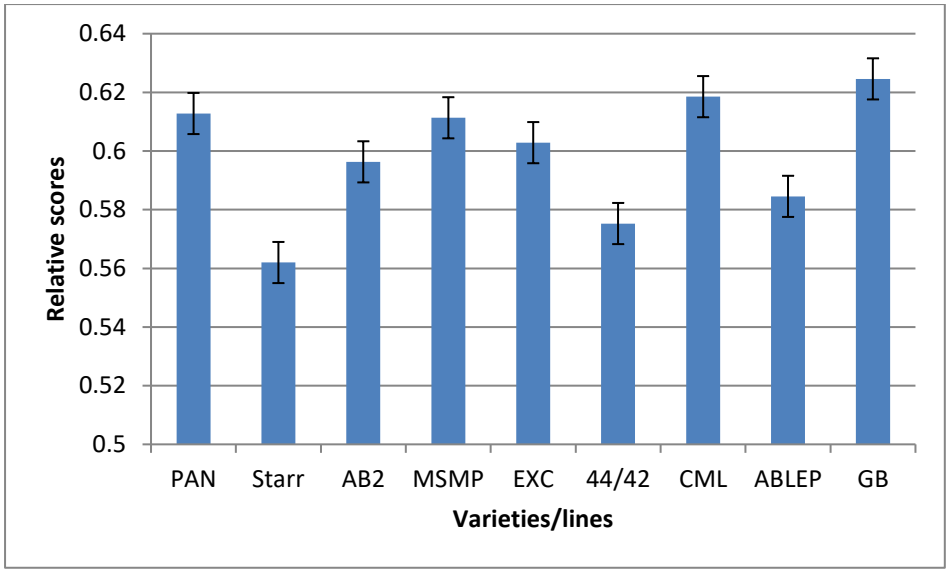



**Fig. 3: Relative scores of yellowness induced in varieties as a result of infection by the MSV isolates. The error bars represent 95% confidence intervals of the means.**

The technique provides sufficient resolution to distinguish maize lines with similar tolerance levels and is rigorous enough to assess highly tolerant lines (Martin & Rybicki, 1998; Martin et al., 1999). The subtle differences observed could be attributed to the highly heterogeneous mix of MSV tolerance genes across the different maize genotypes.

**Progression of Infection Symptoms on Successive Leaves**

Table4 shows the progression of infection symptoms on successive leaf numbers (Martin & Rybicki, 1998; Martin et al., 1999). The percentage of chlorotic leaf area increased from leaf two to four, then leveled off. Leaf four accumulated the highest percentage. However, leaf five and six showed no significant difference.

**Table 4: Comparison of Leaf Number by Means of Various Variables (Leaf Area Relative to Standard, % Chlorotic Area, Relative Average Streak Width and Yellowness)**

| Foliage number | % Chlorotic area | Relative average streak width | Yellowness |
|---|---|---|---|
| 6 | 34.5817b* | 0.041180a* | 0.617665a* |
| 5 | 33.6780b | 0.039623a | 0.619550a |
| 4 | 37.4638a | 0.040311a | 0.607177b |
| 3 | 28.2385c | 0.039909a | 0.587557c |
| 2 | 3.7380d | 0.019494b | 0.577050d |
| **Mean** | **27.54** | **0.0361** | **0.6018** |
| **Cv (%)** | **9.06** | **26.01** | **5.0** |

* Means within a column followed by the same letter are not significantly different according to DMRT ($p \leq 0.05$).

Though leaf five had a lower relative streak width mean compared to leaf four, the trend was consistent, and there was no significant difference between leaves three through to six, similar to what Martin et al. (1999) observed. Mean values of symptom yellowness were also consistent, with leaves six and five having higher means (but not being significantly different from one another), and leaf two having the lowest mean symptom yellowness (Table4).

## Conclusion and Recommendations

The Maseno University-developed maize genotypes AB2, ABLEP, and EX44/42-2 exhibit degrees of MSV tolerance comparable to established resistant varieties like Pan**77**. Consequently, these genotypes represent valuable new sources of MSV tolerance genes in Kenya to mitigate yield losses from maize streak disease. Therefore, regional maize breeders should utilize the lines as donor parents to introgress MSV resistance genes into high-yielding commercial varieties. Additionally, further research should evaluate the agronomic performance and MSV resistance of these promising lines under natural infection conditions across diverse agro-ecological zones.

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