

**PHENOTYPIC AND MOLECULAR EVALUATION OF MAIZE (*Zea mays* L.)
GENOTYPES UNDER FIELD CONDITIONS
IN THE VOLTA REGION OF GHANA**

**Asare-Bediako E^{1*}, Taah KJ¹, van der Puije GC¹, Amenorpe G², Appiah Kubi A¹,
Nee Lamptey J³, Opong A³, Mochiah B³ and I Adama³**



Asare-Bediako Elvis

*Corresponding author email: easare-bediako@ucc.edu.gh

¹Department of Crop Science, School of Agriculture, University of Cape Coast, Ghana

²Biotechnology and Nuclear Agriculture Institute, Ghana Atomic Energy Commission, Accra, Ghana

³CSIR-Crop Research Institute, Kumasi, Ghana



ABSTRACT

Maize streak disease (MSD) is the most devastating and destructive disease of maize (*Zea mays* L.) in sub-Saharan Africa (SSA). Field trials were conducted in the 2014 minor and 2015 major cropping seasons to screen 16 and 17 maize genotypes, respectively, for high yield and resistance to maize streak virus (MSV) infections. The plants were scored for disease severity at 4, 6, 8 and 10 weeks after planting (WAP) based on a 1-5 visual scale (1= No infection and 5= Very severe infection). Polymerase chain reaction (PCR) test was done to detect the presence of MSV in the diseased leaf samples in order to confirm field resistance. Both phenotypic and PCR test revealed that all the maize genotypes tested in the study were infected by MSV. There was a significant varietal effect on the incidence and severity of MSD in both the major and minor seasonal trials. Genotypes 'Abontem', 'Aburohemaa', 'Akposoe', 'Dapango', 'Dorke', 'Etubi', 'Honampa', 'Mamaba', 'Obatanpa', 'Omankwa' and PAN 12 showed mild disease symptoms during both major and minor cropping seasons. On the other hand, genotypes 'Dormabin', 'Dzinu-Eve', 'Enibi', Keta 60 and PAN 53 exhibited moderate to severe symptoms during the two cropping seasons. Incidence and severity of MSD were significantly higher in the minor season than in the major season, indicating a significant seasonal effect of MSV on the maize genotypes. The yield and yield components were observed to vary significantly among the different maize genotypes and between the cropping seasons with mean yields significantly higher in the major season than in the minor season. Genotypes 'Abontem', 'Aburohemaa', 'Akposoe', 'Dorke', 'Etubi', 'Honampa', 'Omankwa', 'Obatanpa' and PAN 12 (All improved varieties), which exhibited partial resistance to MSV infection gave high seed yields during both seasons. The improved maize genotypes that were high yielding and resistant to MSV infection should be evaluated for uniform yield trials on farmers' fields towards their release as varieties to farmers.

Key words: Maize streak virus, Yield losses, Phenotypic, Molecular detection, Polymerase chain reaction



INTRODUCTION

Maize (*Zea mays* L.) is the most widely grown staple food crop in sub-Saharan Africa (SSA), occupying more than 33 million hectares each year [1]. Maize is an excellent source of dietary protein, carbohydrate, saturated fats, polyunsaturated fats, monounsaturated fats, minerals and vitamins [2]. It is rich in dietary fibre and a good source of energy. It is a staple food crop in Ghana and a major constituent in livestock and poultry feed, important for food security and source of cash income and livelihoods especially for the smallholder resource-poor farmers.

Maize streak disease (MSD) caused by maize streak virus (MSV) of the genus *Mastrevirus*, and family *Geminiviridae* is a major threat to cereal crops among smallholder farmers in SSA, including Ghana [3, 4], causing up to US\$480 million losses annually [5]. Infection with MSV causes severe chlorosis on newly emerged leaves of MSV-susceptible maize cultivars, leading to stunted growth, poor ear formation and reduced seed setting. When plants are infected at a young age this results in heavy yield losses or premature death [6]. Maize streak disease is reported to cause yield losses that range from trace to almost 100% depending on the variety [7]. Maize streak virus has a wide host range, infecting over 80 other plant species in the family Poaceae [8] including finger millet, millet, oats, sugarcane and wheat [9]. Maize streak virus is transmitted by as many as six leafhopper species in the genus *Cicadulina*, (Homoptera: Cicadellidae) in a persistent manner [9] but mainly by *C. mbila* and *C. storey* [5].

Maize streak disease epidemic among maize crops in the Volta region of Ghana has been reported [10]. The disease has been reported to affect maize production in the region, thereby affecting livelihoods and food security especially among the smallholder resource-poor farmers in the region. Effective management of MSD is quite pertinent in order to improve yields of maize. There is, however, limited information on the management of MSD in Ghana. Maize streak disease management strategies that have been tried elsewhere include host plant resistance, chemical and cultural controls such as deep residue burial, irrigation and plant density manipulation [3, 5, 9]. Other cultural practices suggested for the control of MSD include the use of 'barriers' of bare ground between early and late planted maize fields to reduce leafhopper movement and subsequent MSV spread [9], the use of crop rotations that will minimize invasion by viruliferous leafhoppers [11] and soil nutrient management [12]. However, cultural practices such as crop rotation are ineffective because of the wide range of alternative hosts for the virus-transmitting leafhopper vector [8], and the limited land size for the smallholder farmers that does not even permit implementation of rotation practice. Further, given the current changing climatic conditions that are associated with unreliable and erratic rainfall, the practice of disease avoidance by altering planting dates to avoid migrating viruliferous leafhoppers from infesting young plants, is not feasible. Pesticides are often applied indiscriminately and inappropriately, resulting in adverse environmental and health effects, and also increase the cost of crop production [5]. Misuse of pesticides by farmers also leads to build up of resistance in the leafhopper vector(s) resulting in ineffective management of both the vector and the MSV. The use of MSV-resistant maize genotypes is seen as the most reliable means of managing MSD [3] and is relevant to the protection of the environment and man [15]. The aim of this



study was to screen maize varieties in order to identify source of resistance to MSV infection.

METHODOLOGY

Study area

The study was conducted at Nkwanta Agricultural Station of the Nkwanta South District of the Volta region of Ghana, an MSV endemic area. This location lies between latitudes 7° 30' and 8° 45' North and longitude 0° 10' and 0° 45' East and falls within the Forest-Guinea savannah transition [13]. The average number of rain days is 86 with extreme annual rainfalls ranging between 922 mm and 1874 mm. The dry season is from November to March during which time the evapotranspiration exceeds water availability at the earth's surface. The mean annual maximum temperatures range between 24°C to 39°C, while the mean annual minimum temperatures are between 11°C to 26°C [13].

Plant material

Sixteen maize genotypes made up of 12 improved varieties (Abontem, Aburohemaa, Akposoe, Dapango, Dorke, Etubi, Honampa, Mamaba, Obatanpa, Omankwa, PAN 12 and PAN 53) and four landraces (Dormabin, Dzinu-Eve, Enibi, Keta 60) were screened at a high disease pressure area at 'Nkwanta' between August and December 2014, in order to identify maize genotypes that are resistant and or tolerant to MSV. The study was repeated in the major season (May-August, 2015) using 17 maize genotypes (with inclusion of PAN 12, an improved variety). The names, characteristics and sources of the maize genotypes used are shown in Table 1.

Experimental design and field layout

The Randomized Complete Block Design (RCBD) with sixteen treatments and four replications was used. A total land area of 2000 m² measuring 80 m x 25 m was ploughed and harrowed to render the soil loose. It was then divided into four blocks and each block was further divided into 16 plots, with each plot measuring 5 m x 4 m. In the major planting season, each block was divided into 17 plots, with each plot measuring 5 m x 4 m. A distance of 1.5 m was left as path between the blocks and 1.0 m between the plots. The 16 (or 17) maize genotypes representing the 16 (or 17) treatments were sown directly at two seeds per hole at a planting distance of 0.8 m x 0.8 m and a planting depth of about 0.5 cm.

Agronomic practices were done when necessary, such as weeding using a hoe and machete. Basal dressing fertilizer (NPK) was applied at a rate of 250 kg ha⁻¹ at three weeks after germination and the field was top-dressed with urea at six weeks after germination. The experiments were carried out under rain-fed conditions.

Agronomic data collection Agronomic data collected include disease incidence and severity, plant height, cob weight, cob length, 100-seed weight and seed yield. In each case, data was taken from 10 plants per plot and the means determined. Disease incidence (DI) per plot was estimated as the percentage of plants on the plot displaying MSD symptoms [4]. The plants were also scored for disease severity at 4, 6, 8 and 10 weeks after planting (WAP) based on a 1–5 scale [4] which is essentially the 1–5 scale [7], with



a modification of 0.5 increments; where 1 - represents no infection; 2 - mild infection; 3 - moderate infection; 4 - severe infection; and 5 - very severe infection.

Grain yield (GY) was adjusted to 15.5% moisture content.

$$GY \text{ (t ha}^{-1}\text{)} = (\text{plot GY}/1000) \times (10000/\text{RW}/\text{H})/1000$$

Where:

RW= row width

H= harvest area

GY= grain yield (adjusted to 15.5% moisture content)

Molecular detection of maize streak virus (MSV) using polymerase chain reaction (PCR) method

Symptomatic leaf samples were collected and used for polymerase chain reaction (PCR) amplification of viral DNA using standard methods [14-16] in order to confirm resistance or otherwise of the various maize genotypes to MSV infection. Total DNA from plants with streak symptoms was extracted by a combination of Dellaporta and CTAB methods [17, 18].

The PCR amplification of viral genome from symptomatic plant samples involved the use of a primer pair 4F (TTC ATC CAA TCT TCA TC) and 4R (GGA AAG TCT ACT TGG GC) [19]. The reaction mixture composition was as follows: Thermo buffer 2.5 μL (1x concentration); MgCl_2 2.0 μL (2.5 mM μL^{-1}); 5% v/v Tween 20 2.5 μL ; Deoxynucleotide triphosphate (dNTP) (2.5 mM μL^{-1} each of dATP, dCTP, dGTP, & dTTP) 1.0 μL ; forward primer (4F) 1.0 μL (2.5 pM μL^{-1}); reverse primer (4R) 1.0 μL (2.5 pM μL^{-1}); *Thermus aquaticus* (Taq) polymerase 0.4 μL (2 units); sample DNA 5.0 μL (1ng μL^{-1}); sterile distilled water (9.6 μL).

Polymerase chain reaction amplification was carried out in a pre-warmed BIO-RAD 1 Cycler™ thermal cycler (Applied Biosystems, New York, USA). The PCR cycles were: an initial denaturation step of 94°C for 1 min followed by 33 cycles of denaturation at 94°C for 1 min, annealing at 50°C for 1 min, and extension at 72°C for 2 min; 1 cycle of 94°C for 1 min, 50°C for 1 min, 72°C for 7 min. The PCR reaction products were kept at 4°C until when electrophoresis was done.

The PCR products were separated by electrophoresis at 100 V for 1.5 hours in 1.5% agarose gel (Sigma Aldrich) stained with Ethidium bromide. A 1 kb DNA ladder (Solis Biodyne, San Diego, CA, USA) was used for DNA fragment size determination and the DNA bands were visualized under UV light using Syngene Gene Flash system (Invitrogen, USA).

Data analyses

Mean incidence data was tested for homogeneity of variance before analyses using Levene's test [20]. The test statistic, W, is equivalent to the F statistic that would be produced by ANOVA, and is defined as follows:



$$W = \frac{(N - k) \sum_{i=1}^k N_i (Z_i - Z_{..})^2}{(k - 1) \sum_{i=1}^k \sum_{j=1}^{N_i} (Z_{ij} - Z_i)^2}$$

Where:

k is the number of different groups to which the sampled cases belong,

N_i is the number of cases in the i th group,

N is the total number of cases in all groups,

Y_{ij} is the value of the measured variable for the j th case from the i th group,

$$Z_{ij} = |Y_{ij} - \bar{Y}_i|$$

The incidence data was then arc-sine transformed [21], in order to homogenise variances. All agronomic data including disease incidence were subjected to analysis of variance (ANOVA) and the means effect separated by least significant difference (LSD) method at 5% level of probability. All statistical analyses were performed using GenStat Release version 12 (VSN International).

RESULTS AND DISCUSSION

Mean incidence of maize streak disease (MSD) during major and minor cropping seasons

The mean incidence of MSD recorded for maize genotypes evaluated at both major and minor cropping seasons are shown in Table 2. In the minor season, ANOVA showed significant difference in the incidence of MSD among the maize genotypes at 4 WAP ($F_{15, 45} = 4.71$; $P < 0.001$), 6 WAP ($F_{15, 45} = 2.93$; $P = 0.003$) and 8 WAP ($F_{15, 45} = 4.22$; $P < 0.001$) but did not show significant difference at 10 WAP ($F_{15, 45} = 1.64$; $P = 0.100$). At 10 WAP, mean MSD incidence ranged between 67.35 % recorded for 'Honampa' and 90 % recorded for 'Dormabin', 'Keta' 60, 'Enibi', 'Etubi' and PAN 53.

In the major season, ANOVA did not indicate significant differences in the incidence of MSD among the maize genotypes at 4 WAP ($F_{16, 48} = 0.93$; $P = 0.545$) and 6 WAP ($F_{16, 48} = 1.56$; $P = 0.119$) but showed significant difference among them at 8 WAP ($F_{16, 48} = 2.67$; $P = 0.004$) and 10 WAP ($F_{16, 48} = 2.19$; $P = 0.019$). At the final observation (10 WAP), mean disease incidence ranged between 30.14 % recorded for PAN 12 and 54.04 % recorded for 'Dzinu-Eve'.

The mean severity scores of MSD recorded for the maize genotypes during the minor and major cropping seasons are shown in Table 3. In the minor season, ANOVA revealed highly significant difference in the severity of MSD among the maize genotypes at 4 WAP ($F_{15, 45} = 3.15$; $P = 0.001$), 6 WAP ($F_{15, 45} = 2.38$; $P = 0.013$), 8 WAP ($F_{15, 45} = 4.73$; $P < 0.001$) and 10 WAP ($F_{15, 45} = 6.13$; $P < 0.001$). At 10 WAP, mean disease severity ranged between 2.363 recorded for 'Dorke' and 3.545 recorded for 'Keta' 60. In the major season, ANOVA of the mean disease severity scores among the maize varieties did not show significant difference at 4 WAP ($F_{16, 48} = 0.91$; $P = 0.562$) and 6 WAP ($F_{16, 48} = 1.72$; $P = 0.075$) but showed significant difference amongst them at 8 WAP ($F_{16, 48} = 4.71$; $P < 0.001$) and 10 WAP ($F_{16, 48} = 5.03$; $P < 0.001$). At 10 WAP, the highest mean



MSD severity score (2.172) was recorded for 'Dzinu-Eve' while the lowest score was recorded for PAN 12 with a mean symptom severity score of 1.242, and an overall mean score of 1.578.

The significant varietal differences in the incidence and severity of MSD could be due to the genetic make-up of the various maize genotypes evaluated. The differences in the resistance levels among lines bred for resistance has been reported by Gichuru [22], to be attributed to different number of genes conditioning resistance or to the influence of genetic background. Significant difference among the maize genotypes could also be as a result of the leaf hopper vector having higher affinity for some of the maize genotypes than others. Differences in the response of genotypes to virus infection have been reported [23] to be common in disease resistance screening and can be attributed to differences in environmental conditions, pathogen variability and virulence. Another possible cause of variation in MSD incidence and severity among the maize genotypes is the evolution of virulent strains of the MSV. This is supported by the finding of Fakorede *et al.* [24] who proposed that one of the major constraints in varietal development is the evolution of a virulent strain to which previously resistant varieties may be susceptible. Genotypes 'Abontem', 'Aburohemaa', 'Akposoe', 'Dapango', 'Dorke', 'Etubi', 'Honampa', 'Mamaba', 'Obatanpa', 'Omankwa' and PAN 12 consistently showed mild symptoms between major and minor cropping seasons (Table 3). This suggests that these maize genotypes possess partial resistance against MSV infection. On the contrary, genotypes 'Dormabin', 'Dzinu-Eve', 'Enibi', 'Keta' 60 and PAN 53 exhibited moderate to severe symptoms between major and minor cropping seasons (Table 3), suggesting they are very susceptible to MSV infection.

In both minor and major cropping season trials, mean MSD incidence and severity scores increased from 4 WAP to 10 WAP (Tables 2 and 3). This suggests that once a maize plant is infected by MSV, it is not able to recover from infection but rather has an increasing effect with time.

Mean sum of squares for season and genotype x season interaction effects on mean MSD incidence and severity scores

Two-way ANOVA on final disease incidence showed significant difference between the cropping seasons ($F_{1, 89} = 258.91$; $P < 0.001$) but did not show significant genotype x season interaction effect ($F_{11, 89} = 0.57$; $P = 0.849$) (Table 4). The final disease incidence recorded in the minor season (80.9 %) was significantly higher ($P < 0.05$) than that of the major season (40.29 %) as shown in Table 2.

The two-way ANOVA on final disease severity showed significant difference between the cropping seasons ($F_{1, 89} = 357.70$; $P < 0.001$) and significant genotype x season interaction effect ($F_{11, 89} = 2.06$; $P = 0.031$). The mean severity score at the final observation (10 WAP) in the minor season (2.918) was significantly higher than that of the major season (1.578) as shown in Table 3.

This finding agrees with those of Asare-Bediako *et al.* [10] who reported higher incidence and severity of MSD in the minor season than in the major season, when they surveyed incidence and severity of MSD in maize farms in the Volta region of Ghana.



Elsewhere in Nigeria, Ladipo and Fakorede [25] and Taiwo *et al.* [26] also reported of higher MSD incidence in the dry season than in the wet season. The significant seasonal effect on the incidence of MSD can be attributed to the preponderance of *Cicadulina* spp. at the beginning of the season. Studies have confirmed that a higher proportion of viruliferous *Cicadulina* spp. is associated with grasses surrounding maize at the beginning of the dry season [27, 28]. Consequently, MSV-A populations can build up in early planted maize and devastate seedlings that germinate in successive plantings [28]. The lower incidence observed during the wet season compared to that of the dry season could also be due to the heavy rainfall which may be responsible for vector mortality, or the increased number of alternative hosts for the vectors during this period [9, 27]. Epidemiology of vector-transmissible viruses may also be attributable to weather conditions. Favourable climatic conditions have been reported to prolong vector migration, enhance vector population and as a result, increase their potential to transmit viruses [29].

Molecular detection of maize streak virus

Figure 1 shows PCR amplification of MSV with the AF /AR primers of the DNA fragment with size 250 bp from all the maize genotypes (lanes 2-18) but no band for negative control (lane 2), indicating that all the maize genotypes tested were susceptible to MSV infection. This result is a confirmation of field observation where all the maize genotypes showed symptoms of MSV infections during both major and minor seasons' trials (Table 2). This suggests that MSV infection of maize could reliably be detected using visual identification as reported by Taiwo *et al.* [24]. Judging from the fact that the majority of the maize genotypes tested were reported to be resistant / tolerant to MSV [30, 31], the result of the current work corroborates that of Karavina [5] who found out that it is difficult to develop maize variety with high degree of MSV resistance through conventional breeding. It is possible that the level of MSV resistance in the maize genotypes tested might have broken down, resulting in all the maize genotypes being infected with MSV.

M 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

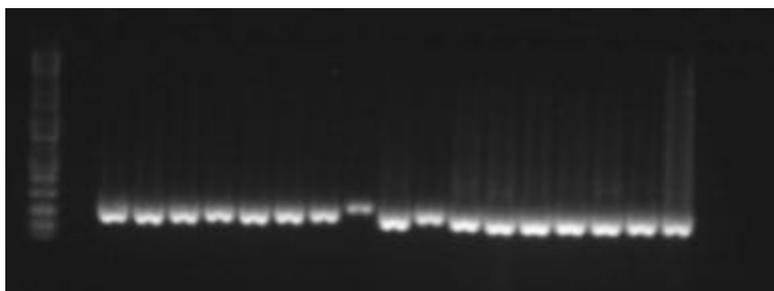


Figure 1: AF/AR primer pairs' amplicon of size 250 bp for MSV in 17 susceptible maize genotypes represented by lanes 2- 18

Lane 1 is the negative control (distilled water), and Lane M denotes 1 kb DNA ladder (Solis Biodyne)

Mean plant height, mean cob weight, mean cob length, mean 100-seed weight and mean seed yield recorded for the maize genotypes

The ANOVA of the mean plant height at tasseling in the major ($F_{16, 48} = 23.07$; $P < 0.001$) and the minor growing seasons ($F_{16, 45} = 4.51$; $P < 0.001$) showed significant differences among the maize genotypes (Table 5). In the major season, the highest mean plant height (251.2 cm) was recorded for 'Dormabin' whilst 'Dapango' had the lowest (260.6 cm). In the minor season, PAN 53 had the highest mean plant height of 223 cm whilst 'Enibi' had the lowest (154 cm) (Table 5).

The mean cob lengths, mean cob weight, mean 100-seed weight (100-SW) and mean grain yield ($t\ ha^{-1}$) recorded in the major and minor cropping seasons showed significant difference among the maize genotypes ($P < 0.001$) (Table 5). In the major cropping season, the highest grain yield was recorded for PAN 12 with a mean yield of $4.58\ kg\ ha^{-1}$, which was not significantly different from 'Obatanpa' ($P > 0.05$) with a mean yield of $4.45\ kg\ ha^{-1}$. These were significantly different from the grain yield obtained from Keta 60 ($2.42\ kg\ ha^{-1}$) which was the lowest. However, in the minor season, 'Obatanpa' had the highest grain yield of $0.536\ kg\ ha^{-1}$ whilst 'Mamaba' had the lowest grain yield of $0.253\ kg\ ha^{-1}$ (Table 5).

These variations in yields and growth have also been reported in different host plant-virus pathosystems, including cassava - African cassava mosaic virus [32], tomato-tomato yellows leaf curl virus [33], and okra-okra mosaic virus interactions [10]. The variations in yields and growth could be due to different host-virus interactions [34] and the age of plants at which they are infected. It is reported that the severity of MSV symptoms depends on the age of the plant at infection; the younger the plant, the higher the severity of symptoms [35]. Genotypes 'Abontem', 'Aburohemaa', 'Akposoe', 'Dorke', 'Etubi', 'Honampa', 'Omankwa', 'Obatanpa' and PAN 12 which exhibited partial resistance to MSV infection also gave high seed yields.

Cropping season and Genotype x season interaction effects on growth and yield of maize genotypes

A two-way ANOVA showed significant seasonal effect on mean plant height ($F_{1, 89} = 7.66$; $P = 0.007$), mean cob length ($F_{1, 89} = 43.27$; $P < 0.001$), mean 100-seed ($F_{1, 89} = 350.87$; $P < 0.001$), mean cob weight ($F_{1, 89} = 236.28$; $P < 0.001$) and mean grain yield ($F_{1, 89} = 2789.88$; $P < 0.001$). In each case, higher values were recorded in the rainy season than in the dry season (Table 5).

The significant seasonal effect on the growth and yields of the maize genotypes could be attributed to higher levels of MSD incidence and severity recorded in the minor season than in the major season (see Tables 2 and 3). This might be attributed to the availability of the vector. This agrees with the report of Bosquez-Perez *et al.* [36] that MSD negatively correlates with plant height and dry weight, grain weight per plot, 1000-grain weight, ear length and diameter. Maize streak disease is reported to have caused yield losses of up to 100% depending on the variety [7, 37]. Another reason for higher growth and yields in major season compared to that of minor season will be differences in the prevailing environmental conditions. Dry season is usually associated with low rainfall and high temperatures, which do not support maize production under natural conditions.



In Ghana, there are reports of lower grain yields of maize in the dry seasons compared to rainy seasons [38, 39].

CONCLUSION

Both PCR test and disease symptoms revealed that none of the seventeen (17) maize genotypes evaluated in the study was immune to MSV infection. Symptom severity was generally higher in the minor season than in the major season, and varied among the maize genotypes. Genotypes 'Abontem', 'Aburohema', 'Akposoe', 'Dapango', 'Dorke', 'Etubi', 'Honampa', 'Mamaba', 'Obatanpa', 'Omankwa' and PAN 12 consistently showed mild symptoms during both trials in the major and minor cropping seasons. These maize genotypes also gave significantly higher seed yields than the rest, suggesting they were resistant / tolerant to MSV infection. These maize genotypes should be evaluated in different agro-ecological zones in the Volta region in order to ascertain the stability of their MSV resistance and high yielding traits under different environments, prior to their recommendation as improved varieties to farmers.

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Table 1: Names, characteristics and sources of maize genotypes used for the study

Maize genotype	Type	Characteristics	Source
Abontem	Improved	QPM*, OPV**, streak tolerant	Crop Research Institute (CRI), Kumasi
Aburohema	Improved	QPM*, OPV**, drought and streak tolerant	CRI, Kumasi
Akposoe	Landrace	Hybrid, streak tolerant	CRI, Kumasi
Dapango	Improved	Unknown	Abor, Volta Region
Dorke	Improved	Hybrid, streak tolerant	CRI, Kumasi
Domabin	Improved	Unknown	Domabin, Volta Region
Dzinu-eve	Landrace	Unknown	Abor, Volta Region
Enii Pibi	Improved	QPM, hybrid, streak tolerant	CRI, Kumasi
Etubi	Improved	Hybrid, streak tolerant	CRI, Kumasi
Honampa	Improved	OPV, streak tolerant	CRI, Kumasi
Jubilee Golden	Improved	Hybrid, streak tolerant	CRI, Kumasi
Keta 60	Landrace	Unknown	Keta, Volta Region
Mamaba	Improved	Hybrid, streak tolerant	CRI, Kumasi
Obatanpa	Improved	OPV, streak tolerant	CRI, Kumasi
Oman kwa	Improved	QPM, OPV, streak tolerant	CRI, Kumasi
PAN 12	Improved	Hybrid, streak tolerant	Agro-input dealer, Accra
PAN 53	Improved	Hybrid, streak tolerant	Agro-input dealer, Accra

QPM*, quality protein maize; OPV**, open pollinated variety ***PAN 12 was planted in the major season trial only

Table 2: Mean incidence of maize streak disease on maize genotypes in both major and minor cropping seasons

Maize genotype	Minor season incidence (%)				Major season incidence (%)			
	4 WAP	6 WAP	8 WAP	10 WAP	4 WAP	6 WAP	8 WAP	10 WAP
Abontem	12.99 ^{cde}	49.20 ^e	74.96 ^{abcde}	73.34 ^{ns}	0.00 ^{ns}	28.01 ^{ns}	29.84 ^{de}	36.84 ^{cd}
Aburohema	14.8 ^{4bcd}	51.43 ^e	62.79 ^{ef}	78.75	0.00	15.89	27.84 ^{de}	33.11 ^d
Akposoe	10.22 ^{cde}	51.13 ^e	74.96 ^{abcde}	78.18	5.35	21.91	29.68 ^{de}	37.88 ^{cd}
Dapango	8.39 ^{de}	61.82 ^{bcde}	72.54 ^{bcdef}	71.23	0.00	0.00	34.91 ^{bcd}	40.52 ^{bcd}
Dorke	28.36 ^{ab}	58.02 ^{cde}	67.71 ^{cdef}	73.31	0.00	26.69	31.16 ^{cde}	40.31 ^{bcd}
Dormabin	23.95 ^{abc}	67.05 ^{abcd}	82.13 ^{abc}	90.00	0.00	25.06	46.32 ^{ab}	49.69 ^{ab}
Dzinu-Eve	15.89 ^{bcd}	71.79 ^{abc}	84.82 ^{ab}	85.39	0.00	22.34	53.06 ^a	54.04 ^a
Enibi	0.00 ^e	59.65 ^{cde}	90.00 ^a	90.00	0.00	21.82	44.45 ^{abc}	47.25 ^{abc}
Etubi	0.00 ^e	56.15 ^{de}	83.98 ^{ab}	90.00	4.19	15.44	34.83 ^{bcd}	39.93 ^{bcd}
Honampa	27.33 ^{ab}	53.23 ^{de}	65.70 ^{def}	67.35	4.61	19.64	33.23 ^{bcde}	38.04 ^{cd}
G. Jubilee	13.00 ^{cde}	58.75 ^{cde}	64.56 ^{ef}	76.48	4.19	25.57	33.71 ^{bcde}	40.33 ^{bcd}
Keta 60	38.12 ^a	78.11 ^a	90.00 ^a	90.00	0.00	10.22	34.24 ^{bcd}	39.27 ^{bcd}
Mamaba	0.00 ^e	61.14 ^{bcde}	80.19 ^{abcd}	84.82	0.00	17.72	40.97 ^{abcd}	46.38 ^{abc}
Obatanpa	10.63 ^{cde}	51.28 ^e	58.82 ^f	74.94	0.00	14.41	28.36 ^{de}	36.50 ^{cd}
Omankwa	21.48 ^{bcd}	57.54 ^{cde}	80.52 ^{abcd}	80.75	7.94	19.55	31.32 ^{cde}	36.37 ^{cd}
PAN 12	-	-	-	-	0.00	23.74	20.08 ^e	30.14 ^d
PAN 53	18.47 ^{bcd}	74.77 ^{ab}	90.00 ^a	90.00	5.18	29.60	32.94 ^{bcde}	38.38 ^{bcd}
Mean	15.2	60.1	75.4	80.9	1.85	19.1	34.5	40.29
l.s.d	14.25	14.61	15.63	17.30	7.98	16.65	13.67	11.52

(P<0.05)

Means in the same column having different letters are significantly different ($P<0.05$)



Table 3: Mean severity scores of maize streak disease in both major and minor cropping seasons

Maize genotype	Mean MSD severity scores in the major season				Mean MSD severity scores in the minor season			
	4 WAP	6 WAP	8 WAP	10 WAP	4 WAP	6 WAP	8 WAP	10 WAP
Abontem	1.000 ^{ns}	1.355 ^{ns}	1.378 ^{defg}	1.467 ^{defgh}	1.125 ^{bcde}	1.902 ^e	2.295 ^f	2.643 ^{cde}
Aburohema	1.000	1.072	1.200 ^{fg}	1.485 ^{defgh}	1.167 ^{bcde}	2.062 ^{de}	2.387 ^{ef}	2.693 ^{cde}
Akposoe	1.010	1.182	1.313 ^{defg}	1.357 ^{efgh}	1.083 ^{cde}	2.012 ^{de}	2.865 ^{bcde}	2.918 ^{bcd}
Dapango	1.000	1.000	1.498 ^{bcdef}	1.600 ^{cdefg}	1.040 ^{de}	2.225 ^{bcde}	2.287 ^f	2.428 ^e
Dorke	1.000	1.350	1.270 ^{efg}	1.460 ^{defgh}	1.272 ^{ab}	2.255 ^{bcde}	2.315 ^f	2.363 ^e
Dormabin	1.000	1.295	1.730 ^{bc}	1.855 ^{bc}	1.167 ^{bcde}	2.620 ^{abc}	3.045 ^{abcd}	3.495 ^a
Dzinu-Eve	1.000	1.247	2.060 ^a	2.172 ^a	1.145 ^{bcde}	2.532 ^{abcd}	2.782 ^{cdef}	3.300 ^{ab}
Enibi	1.000	1.420	1.610 ^{bcd}	1.663 ^{bcde}	1.000 ^e	2.405 ^{abcd}	3.312 ^{ab}	3.405 ^{ab}
Etubi	1.023	1.205	1.395 ^{defg}	1.462 ^{defgh}	1.000 ^e	1.992 ^e	3.212 ^{abc}	2.363 ^e
Honampa	1.125	1.140	1.505 ^{bcde}	1.640 ^{cdef}	1.225 ^{bc}	2.062 ^{de}	2.322 ^f	2.573 ^{de}
Jubilee.Golden	1.043	1.265	1.438 ^{cdefg}	1.475 ^{defgh}	1.087 ^{cde}	1.962 ^e	2.382 ^{ef}	2.673 ^{cde}
Keta 60	1.000	1.120	1.478 ^{cdef}	1.722 ^{bcd}	1.430 ^a	2.867 ^a	3.382 ^a	3.545 ^a
Mamaba	1.000	1.180	1.788 ^{ab}	1.955 ^{ab}	1.000 ^e	2.120 ^{cde}	2.680 ^{def}	2.960 ^{bcd}
Obatanpa	1.000	1.145	1.290 ^{efg}	1.323 ^{gh}	1.087 ^{cde}	2.150 ^{bcde}	2.635 ^{def}	2.760 ^{cde}
Omankwa	1.053	1.210	1.325 ^{defg}	1.347 ^{fgh}	1.195 ^{bcd}	2.177 ^{bcde}	2.455 ^{ef}	2.463 ^e
PAN 12	1.000	1.157	1.163 ^g	1.242 ^h	-	-	-	-
PAN 53	1.048	1.437	1.483 ^{cdef}	1.722 ^{bcd}	1.125 ^{bcde}	2.657 ^{ab}	3.002 ^{abcd}	3.495 ^a
Mean	1.018	1.222	1.466	1.582	1.134	2.252	2.710	2.968
l.s.d (P<0.05)	0.099	0.260	0.300	0.308	0.181	0.526	0.505	0.459

Means in the same column having different letters are significantly different (P<0.05)
MSD symptom severity score (SSS): 1=no infection; 2=mild infection; 3=moderate infection; 4=severe infection; 5= very severe infection



Table 4: Mean sum of squares for mean incidence and severity of maize streak disease (MSD)

Source of variation	Degree of freedom	Final incidence (%)	Final severity score
Genotype	26	800.3**	1.1627**
Season	1	35126.9**	36.4688**
Genotype x Season	11	77.2 ^{ns}	0.2101*
Error	89	135.7	0.1020

*Significant at $P < 0.05$; **Significant at $P < 0.01$

Table 5: Growth and yield performance of maize genotypes at the minor and major cropping seasons

Variety	Major season					Minor season				
	PH (cm)	CL (cm)	CW (g)	100-SW (g)	Yield (kg ha ⁻¹)	PH (cm)	CL(cm)	CW (g)	100-SW (g)	Yield (kg ha ⁻¹)
Abontem	203.5 ^c	15.73 ^{abc}	144.6 ^{cd}	24.15 ^c	3.47 ^{bc}	211.8 ^{ab}	13.85 ^{ab}	79.97 ^{bcd}	17.25 ^{ab}	0.3901 ^{bcd}
Aburohema	199.1 ^{cd}	13.51 ^{def}	136.6 ^{cde}	23.63 ^{cd}	3.46 ^{bc}	172.1 ^{def}	11.25 ^{def}	62.70 ^{cde}	16.75 ^{abc}	0.3058 ^{cde}
Akposoe	185.1 ^{def}	14.21 ^{bcde}	152.9 ^{bc}	21.97 ^{def}	3.74 ^b	169.4 ^{ef}	11.07 ^{ef}	55.21 ^{cde}	16.00 ^{abcd}	0.31693 ^{cd} e
Dapango	168.6 ^g	12.33 ^f	102.6 ^{gh}	20.02 ^f	2.44 ^d	199.9 ^{abc}	13.07 ^{abc}	80.22 ^{bcd}	15.75 ^{bcd}	0.3913 ^{bcd}
Dorke	222.1 ^b	15.97 ^{ab}	146.3 ^{bcd}	26.37 ^b	3.69 ^b	203.0 ^{abc}	13.27 ^{abc}	75.68 ^{bcde}	6.25 ^{abcd}	0.3691 ^{bcd} e
Dormabin	251.2 ^a	17.30 ^a	134.6 ^{def}	29.82 ^a	3.31 ^{bc}	214.1 ^{ab}	14.00 ^{ab}	76.80 ^{bcde}	14.75 ^{bcde}	0.3746 ^{bcd} e
Dzinu-Eve	202.9 ^c	13.71 ^{def}	118.3 ^{fg}	24.09 ^c	3.12 ^c	202.2 ^{abc}	13.22 ^{abc}	57.28 ^{cde}	12.00 ^e	0.2794 ^{cde}
Enibi	171.0 ^{fg}	13.62 ^{def}	101.3 ^h	20.30 ^{ef}	3.26 ^{bc}	154.0 ^f	10.07 ^f	53.28 ^{de}	13.50 ^{cde}	0.2599 ^{de}



Etubi	176.5 ^{fg}	14.87 ^{bcd}	141.5 ^{cde}	20.96 ^{ef}	3.59 ^{bc}	172.0 ^{d^{ef}}	11.24 ^{def}	61.27 ^{cde}	13.00 ^{de}	0.3188 ^{cde}
Honampa	192.2 ^{cd}	14.50 ^{b^{cde}}	135.4 ^{de}	22.82 ^{cde}	3.43 ^{bc}	183.3 ^{cde}	11.98 ^{cde}	80.70 ^{bc}	16.00 ^{abcd}	0.3936 ^{bc}
Jubilee	203.1 ^c	14.28 ^{b^{cde}}	125.9 ^{ef}	24.11 ^c	2.89 ^{0^c}	171.5 ^{def}	11.21 ^{def}	63.06 ^{cde}	13.25 ^{de}	0.2776 ^{cde}
Golden										
Keta 60	206.0 ^c	13.00 ^{ef}	99.1 ^h	24.45 ^c	2.42 ^d	189.2 ^{b^{cde}}	12.37 ^{b^{cde}}	73.65 ^{b^{cde}}	15.75 ^{b^{cd}}	0.2892 ^{cde}
Mamaba	182.7 ^{efg}	14.32 ^{b^{cde}}	151.2 ^{b^{cd}}	21.69 ^{efg}	3.81 ^b	196.9 ^{abcd}	12.88 ^{abcd}	51.80 ^e	15.25 ^{b^{cde}}	0.2527 ^e
Obatanpa	250.9 ^a	15.29 ^{b^{cd}}	162.2 ^{ab}	29.78 ^a	4.45 ^a	188.1 ^{b^{cde}}	12.30 ^{b^{cde}}	92.33 ^{ab}	17.00 ^{ab}	0.5363 ^a
Omankwa	199.6 ^{cd}	14.00 ^{c^{def}}	141.5 ^{cde}	23.69 ^{cd}	3.55 ^{bc}	220.5 ^a	14.42 ^a	91.75 ^{ab}	17.50 ^{ab}	0.4475 ^{ab}
PAN 12	170.0 ^{fg}	17.25 ^a	176.3 ^a	20.18 ^{ef}	4.58 ^a	-	-	-	-	-
PAN 53	233.3 ^b	15.22 ^{b^{cd}}	170.3 ^a	27.69 ^b	3.53 ^{bc}	223.0 ^a	14.58 ^a	109.95 ^a	19.25 ^a	0.4504 ^{ab}
Mean	201.1	14.65	137.7	23.86	3.469	191.9	12.55	72.9	15.58	0.355
l.s.d (P<0.05)	15.32	1.790	16.59	1.819	0.5739	27.34	1.787	27.20	3.433	0.1326

Means in the same column followed by different letters are significantly different (P<0.05)
 PH= Plant height; CL=Cob length; CW= Cob weight; 100-SW= 100-seed weight



Table 6: Mean sum of squares for plant height, cob length, cob weight, 100 seed weight and seed yield

Source of Variation	d.f.	Plant height	Cob length	Cob weight	100-Seed Weight	Seed Yield
Genotype	26	1975.9	7.307**	2352.4**	52.795**	3.16260**
Season	1	2626.9	83.397**	97940.9**	1562.218**	225.1915**
Genotype x Season	11	1314.7	5.279**	869.4*	14.685**	0.4831**
Error	89	343.1	1.927	414.5	4.452	0.08072

*Significant at P<0.05; **Significant at P<0.01



REFERENCES

1. **FAOSTAT**. Statistical databases and data-sets of the food and agriculture organization of the United Nations, Rome, Italy. 2008 <http://faostat.fao.org/default.aspx> Accessed April 2015.
2. **FAO**. The state of food insecurity in the world. Food and Agricultural Organization (FAO) of United Nations. Rome, Italy. 2008; 4-6.
3. **Martin DP and DN Shepherd** The epidemiology, economic impact and control of maize streak disease. *J. Food Secur.* 2009; **1(3)**: 305-315.
4. **Opong A, Offei SK, Ofori K, Adu-Dapaah H, Lamptey JNL, Kurenbach B, Walters M, Shepherd DN, Martin DP and A Varsani** Mapping the distribution of maize streak virus genotypes across the forest and transition zones of Ghana. *Arch. Virol.* 2014; **160 (2)**: 483-492.
5. **Karavina C** Maize streak virus: A review of pathogen occurrence, biology and management options for small holder farmers. *Afr. J. Agric. Res.* 2014; **9**: 2736-2742.
6. **Mawere S, Vincent V, De Meyer J and KV Pixley** Resistance of four inbred maize lines to inoculation with 20 isolates of maize streak virus from Zimbabwe. *Plant Dis.* 2006; **90**: 1485–1489.
7. **Kyeter DT, Ming R, McMullen MD, Pratt RC and J Brewbaker** Genetic analysis of tolerance to maize streak virus in maize. *Genome Res.* 1999; **42**: 20-26.
8. **Shepherd DN, Martin DP, van der Walt E, Dent K and A Varsani** Maize streak virus: An old and complex ‘emerging’ pathogen. *Mol Plant Pathol.* 2010; **11**: 1-2.
9. **Bosque-Pérez NA** Eight decades of maize streak virus research. *Virus Res.* 2000; **71**: 107-121.
10. **Asare-Bediako E, Kvarnheden A, van der Puije GC, Taah KJ, Frimpong KA, Amenorpe G, Appiah-Kubi A, Lamptey JNL, Opong A, Mochiah MB, Adam I and FM Tetteh** Spatio-temporal variations in the incidence and severity of maize streak disease in the Volta Region of Ghana. *J Plant Pathol Microbiol.* 2017; **8(3)**: 401-7.
11. **Rose DJ** Epidemiology of maize streak disease. *Annu. Rev. Entomol.* 1978; **23**: 259-282.
12. **Magenya OEV, Mueke J and C Omwega** Significance and transmission of maize streak virus disease in Africa and options for management: A Review. *Afr. J. Biotechnol.* 2008; **7**: 4897-4910.
13. **Nyarko P** Population and housing census. District analytical report: Nkwanta South district. Ghana Statistical Service, Accra, Ghana, 2014.



14. **Oluwafemi S, Kraberger S, Shepherd DN, Martin DP and A Varsani** A high degree of African streak virus diversity within Nigerian maize fields includes a new mastrevirus from *Axonopus compressus*. *Arch. Virol.* 2014; **159**: 2765-2770.
15. **Owor BE, Martin DP, Shepherd DN, Edema R, Monjane AL, Rybicki, EP, Jennifer A, Thomson JA and A Varsani** Genetic analysis of maize streak virus isolates from Uganda reveals widespread distribution of a recombinant variant. *J Gen Virol.* 2007; **88**: 3154-3165.
16. **Shepherd DN, Martin DP, Lefeuvre P, Monjane AL, Owor BE, Rybicki EP and A Varsani** A protocol for the rapid isolation of full geminivirus genomes from dried plant tissue. *J. Virol. Methods.* 2008; **149**: 97-102.
17. **Dellaporta SL, Wood J and JB Hicks** A plant DNA miniprep: version II. *Plant Mol Biol Rep.* 1983; **1**: 19-21.
18. **Doyle J and JL Doyle** Isolation of plant DNA from fresh tissue. *Focus.* 1990; **12**: 13-15.
19. **Rybicki EP and FL Hughes** Detection and typing of maize streak virus and other distantly related geminiviruses of grasses by polymerase chain reaction of a conserved viral sequence. *J Gen Virol.* 1990; **71**: 2519-2526.
20. **Levene H** "Robust tests for equality of variances". In I. Olkin (ed). *Contributions to Probability and Statistics: Essays in Honor of Harold Hotelling.* Stanford University Press. 1960; 278-292.
21. **Legendre P and L Legendre** *Numerical Ecology: Development in Environmental Modelling. Second edition.* Elsevier Science. 1998; 825.
22. **Gichuru LN** Breeding investigations on utility of maize streak virus resistant germplasm for hybrid development in the tropics (Doctoral dissertation, University of KwaZulu-Natal, Pietermaritzburg, 2013).
23. **Gremillion SK, Culbreath AK, Gorbet DW, Mullinix Jr BG, Pittman RN, Stevenson KL, Todd JW, Escobar RE and MM Condori** Field evaluations of leaf spot resistance and yield in peanut genotypes in the United States and Bolivia. *Plant Dis.* 2011; **95 (3)**: 263-268.
24. **Fakorede MAB, Fajemisin JM, Ladipo JL, Ajala SO and SK Kim** Development and regional deployment of streak virus maize germplasm: an overview. 503-516 in Jacqueline d'A Hughes and Babajide O Odu (eds). *Plant Virology in Sub-Saharan Africa.* Proc. of a conference organized by the International Institute of Tropical Agriculture, Ibadan 4th-8th June, 2001.
25. **Ladipo JL and MAB Fakorede** Influence of seasonal changes on the incidence of maize streak virus disease at the Obafemi Awolowo University teaching and research farm, Nigeria. *Int. J. Plant Prot.* 1992; **14**: 62-6.



26. **Taiwo MA, Hughes JDA and KE Oke** Studies on maize streak virus and maize mottle/chlorotic stunt virus in Lagos, Nigeria. *Plant Dis.* 2006; **90**: no.2, 199-202.
27. **Asanzi CM, Bosque-Perez NA, Buddenhagen IW, Gordon DT and LR Nault** Interactions among maize streak virus disease, leafhopper vector populations and maize cultivars in forest and savanna zones of Nigeria. *Plant Pathol.* 1994; **43 (1)**: 145-157.
28. **Dabrowski ZT, Nwilene F and R Kumar** First regular observations on leafhoppers, *Cicadulina* spp., vectors of maize streak virus (MSV) in south-eastern Nigeria. *Int J Trop Insect Sc.* 1991; **12 (3)**: 249-26.
29. **Orawu M** *Occurrence of cowpea aphid-borne mosaic virus and prospects of improving resistance in local cowpea landraces in Uganda* (Doctoral dissertation, University of KwaZulu-Natal, Pietermaritzburg). 2007.
30. **Twumasi-Afriyie S, Badu-Apraku B, Sallah PY, Haag W and EA Asiedu** Development and release of Obatanpa, an intermediate maturing quality protein maize variety in Ghana. Crop Research Institute, Kumasi, Ghana. 1992.
31. **Twumasi-Afriyie S, Sallah PY, Owusu-Akyaw M, Ahenkora K and RF Soza** Development and utilisation of quality protein maize in Ghana. Crop Research Institute Kumasi, Ghana. 2012.
32. **Asare PA, Galyuon IKA, Asare-Bediako E, Sarfo JK and JP Tetteh** Phenotypic and molecular screening of cassava (*Manihot esculenta* Crantz) genotypes for resistance to cassava mosaic disease. *J. Gen. Mol. Virol.* 2014; **6 (2)**: 6-18.
33. **Osei MK, Akromah R, Lamptey JN and MD Quain** Phenotypic and molecular screening of some tomato germplasm for resistance to tomato yellow leaf curl virus disease in Ghana. *Afr. J. Agric. Res.* 2012; **7**: 4675-84.
34. **Anneke E, Hogerwerf L and J Slingenbergh** Pathogen-host environment interplay and disease emergence. *Emerg. Microbes Infect.* 2013; **2(2)**:e5.
35. **Efron Y, Kim SK, Fajemisin JM, Mareck JH, Tang CY, Dabrowski ZT and IW Buddenhagen** Breeding for resistance to maize streak virus: a multidisciplinary team approach. *Plant Breed.* 1989; **103**: 1-36.
36. **Bosque-Pérez NA, Olojede SO and IW Buddenhagen** Effect of maize streak virus disease on the growth and yield of maize as influenced by varietal resistance levels and plant stage at time of challenge. *Euphytica.* 1998; **101**: 307-317.
37. **Alegbejo MD, Olojede SO, Kashina BD and ME Abo** Maize streak mastrevirus in Africa: distribution, transmission, epidemiology, economic significance and management strategies. *J Sustain Agr.* 2002; **19**: 35-45.



38. **Blankson D** Influence of tillage, N, P, K fertilization and maize varieties on the incidence and severity of maize streak disease. MPhil Thesis, Department of Crop Science, School of Agric, University of Cape Coast, Ghana, 2017.
39. **Appiah-Kubi A** Study of maize streak disease in the Volta Region of Ghana. MPhil. Thesis, Department of Crop Science, School of Agriculture, University of Cape Coast, Ghana, 2017.

