

JOURNAL OF SCIENCE, TECHNOLOGY, MATHEMATICS AND EDUCATION (JOSTMED)



ISSN: 0748 – 4710

VOLUME 11(3), DECEMBER, 2015

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ISSN 0748-4710

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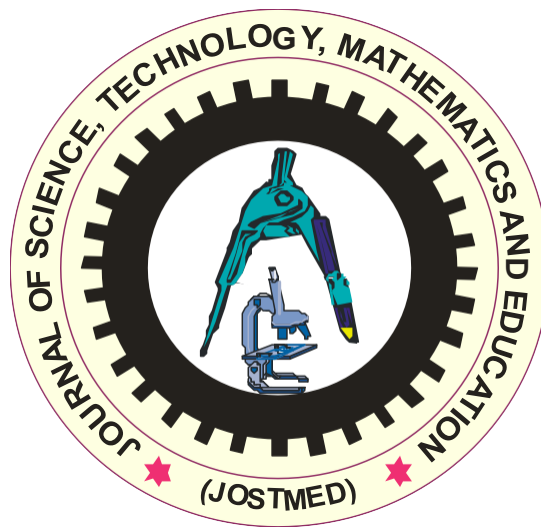
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**ARTICLES AND RESEARCH REPORTS
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REVIEW OF MAIZE STREAK VIRUS DISEASE AND MANAGEMENT STRATEGIES IN SUB-SAHARAN AFRICA

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Abstract

Maize streak disease (MSD) induced by Maize streak virus (MSV) is an economically important viral disease of maize in sub-Saharan Africa. Following its first report in South Africa in 1901, the virus has been confirmed in virtually all African countries. Maize streak virus is a member of the Geminiviruses with characteristic monopartite genome, measuring 18×30 nm in size. Each virion of the MSV contains a single, covalently bound, circular ssDNA molecule of about 2.7 kb. Replication occurs through double stranded DNA (dsDNA) intermediates, using a rolling circle replication mechanism. The virus induces various forms of foliar symptoms such as white, yellow or red streaks in susceptible varieties. Maize streak virus has a wide host range within the grass family (Poaceae) and is transmitted by several leafhoppers (Cicadulina spp.) in a persistent manner. Eleven strains of MSV have been identified, with various degree of pathogenicity, causing maize losses of about US\$480 million annually. Management of MSV could be by application of insecticide, early planting, crop rotation and cultivation of resistant genotypes. Among these control options, adoption of resistant varieties is by far the most effective and sustainable. There is need for multidisciplinary approach and continuous collaboration from maize breeders, entomologists and plant virologists in order to eradicate MSD menace in the region.

Keywords: Maize productivity, Maize streak virus, management, strains and host resistance, transmission and epidemiology.

Introduction

Maize (*Zea mays* L.) is an important cereal crop in sub-Saharan Africa (SSA) where it is cultivated on 25 million hectares (Gianessi, 2015). It is a major food source in many developing countries of Latin America, Africa and Asia. At global level it accounts for 15 % of proteins and 20 % of calorie intake (Sofi et al., 2009). Maize, which may be eaten as a vegetable or processed into various dishes, is regarded as a hunger breaker after a long dry period (Menkir & Kling, 1999). Additionally, maize is extensively traded as feed crop in livestock industries, and about 460 million (65 %) of total world maize production is used for this purpose (Babatunde et al., 2008). Besides food and feed, maize has a wide range of industrial applications including manufacturing of ethanol and pharmaceuticals (Tijani & Osotimehin, 2007). Furthermore, maize may be planted in intercropping or mixed cropping, indicating its compatibility with the common practice among tropical subsistence farmers (Alabi & Esobhawan, 2006). In West and Central Africa (WCA) production potential is greatest in the moist savanna where annual rainfall and solar radiation are favourable, and incidence of pests and diseases are minimal (Badu-Apraku et al., 2008).

Maize productivity is seriously constrained by low soil fertility, lack of improved technology, and subsistence farming (IITA, 2009). The influence of several insect pests, diseases and parasitic weeds pose serious biotic stresses on maize yield (Badu-Apraku et al., 2008). In addition, recurrent drought has been identified as a principal climatic threat to its productivity in sub-Saharan Africa (SSA) (Centro Internacional de Mejoramiento de Maíz y Trigo [CIMMYT], 2010).

Several pathogens such as bacteria, fungi, nematodes and viruses have also been implicated in low yield. However, Maize streak disease (MSD) induced by Maize streak virus (MSV; genus *Mastrevirus*, Family Geminiviridae) is the most important viral disease of maize in SSA (Shepherd et al., 2010). Symptoms of the disease are influenced by host – pathogen interactions, virus strain, maize genotype, plant age at infection, and climatic conditions. They generally include chlorotic streaks along the veins of the leaf laminae, stunting, and plant death (Bosque-Pérez et al., 1998). Disease incidence and yield losses can be as high as 100 %. Annual losses attributed to streak infection about US\$480 million (Karavina, 2014). Complete yield loss and field abandonment is rampant when both drought and streak infection occur simultaneously. Maize streak virus is vectored by eight leafhopper species namely, *Cicadulina arachidis* China, *C. bipunctata* Melichar, *C. ghaurii* Dabrowski, *C. latens* Fennah, *C. mbila* Naudé, *C. parazeae* Ghauri, *C. similis* China, and *C. storeyi* China (= *C. triangula* Ruppel) (Oluwafemi et al., 2007; Fajinmi et al., 2012), in a persistent manner (Nawaz-ul-Rehman & Fauquet, 2009).

Control strategies include crop rotation, timely planting, chemical seed treatment, roguing, insecticidal control of insect vectors and host plant resistance. However, the use of resistant maize genotypes is the most viable option; being cost-effective, safe, sustainable and compatible with integrated pest management (IPM) (Danson et al., 2006). In West Africa, the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria initiated a breeding programme for MSV in 1975. This was aimed at developing maize varieties that combine resistance to MSV with desirable agronomic characters (Efron et al., 1989). This paper reviews MSV with emphasis on its origin and distribution, properties, symptoms induced, strains and diversity, transmission, epidemiology, host range, and management strategies.

Origin and Distribution of Maize Streak Virus

Maize streak virus was first reported in South Africa by Fuller (1901) as 'mealie variegation'. The virus was later renamed Maize streak virus by Storey (1925). MSV is now widely distributed in sub-Saharan Africa (European Plant Protection Organization [EPPO], 2006). In Africa, it is present in Angola, Benin, Botswana, Burkina-Faso, Burundi, Cameroon, Central African Republic, Congo Democratic Republic, Cote d'Ivoire, Egypt, Ethiopia, Gabon, Ghana, Guinea, Kenya, Madagascar, Malawi, Mali, Mauritius, Mozambique, Niger, Nigeria, Rwanda, Réunion, Sao Tome and Principe, Senegal, Sierra Leone, South Africa Sudan, Swaziland, Tanzania, Togo, Uganda, Zambia and Zimbabwe (EPPO, 2006). In Asia, it has been confirmed in India, Indonesia and Yemen (EPPO, 2006). Efron et al. (1989) documented that the virus does not occur in the New World; it is found both in the forest and savanna and from sea level up to elevations of 200 m.

Properties of Maize Streak Virus

Maize streak virus is a member of the genus *Mastrevirus* (family: Geminiviridae) (Willment et al., 2001). Geminiviruses are plant viruses with monopartite or bipartite circular single stranded DNA (ssDNA) genomes. They are 2.5 to 3.0 kb particles of 20 × 30 nm wide (Frischmuth & Stanley, 1993). The Geminiviridae comprises four genera (*Mastrevirus*, *Curtovirus*, *Topocovirus* and *Begomovirus*), based on their genome organizations and biological properties (Fauquet et al., 2003). MSV is a member of the Geminivirus group that consists of viruses having monopartite genomes (Hull et al., 1991). The particle measures 18 × 30 nm in size (Bosque – Pérez, 2000). Each virion of the MSV contains a single, covalently bound, circular, ssDNA molecule of about 2.7 kb which codes for four potential products (Shepherd et al., 2014). Replication occurs through double stranded DNA (dsDNA) intermediates, using a rolling circle replication mechanism (Laufs et al., 1995a, b). Recently, studies have proved that 'recombination – dependent replication' mechanisms are also involved in Geminivirus replication (Jovel et al., 2007). Geminiviruses do not encode their own DNA polymerases but rely on the nuclear DNA replication machinery of the host (Munoz-Martin et al., 2003).

In replicative double stranded DNA (dsDNA) molecules, genetic expression arises from both strands, and diverges from an intergenic region containing the virion – sense origin of replication (Morris – Krsinich et al., 1985). Rolling circle replication begins by binding of the virus replication – associated protein (Rep) to the virion – strand origin of replication, where the protein begins and terminates virion strand DNA synthesis (Willment et al., 2007). Additionally, MSV Rep consists of two complementary sense open reading frames (ORFs), C1 and C2. Rep (Spliced transcript) or Rep A (unspliced transcript) is produced from the C1:C2 containing an intron. Although MSV replication is facilitated by Rep alone (Liu et al., 1998), Rep A plays a variety of important additional functions during its life cycle. Such activities include the modulation of host cell cycle regulation, and probably other development pathways (Shepherd et al., 2005).

Also present in the MSV genome are the movement protein (MP) and the coat protein (CP). The former enhances the movement of the virus from the site of replication (nucleus) to adjacent cells. Conversely, CP is responsible for the encapsidation of the viral nucleic acid, and determines virus – vector interactions (Bridson et al., 1994). Additionally, MSV CP binds non-specifically to both ssDNA and dsDNA (Liu et al., 1999). It is also required for cell – to – cell and systemic spread of the virus in plants (Liu et al., 2001a), and interacts specifically with MP to move virus DNA out of the nucleus (Liu et al., 2001b).

Symptoms of Maize Streak Virus Disease

Maize streak virus disease symptoms are influenced by several factors including the level of resistance of the maize genotype, age at infection, virulence of the strain and environmental factors (Bosque – Pérez et al., 1998). In susceptible maize plants, streak disease normally manifests as minute, pale, circular spots on the lowest exposed portion of the youngest leaves. As leaves expand, conspicuous chlorotic streaks ranging from broken to almost continuous streaks are evident along the veins on the most of the leaf laminae and, since the virus is systemic, symptoms manifest on the inoculated and subsequent leaves (Thottappilly et al., 1993). As infection advances, less streaks are found on secondary and tertiary veins, compared to the primary veins. Lesion colour is either white or yellow, but some MSV strains induce red pigmentation on maize leaves, abnormal shoot and flower bunching in grasses (Shepherd et al., 2010).

The streak pattern arises from the failure of chloroplasts to develop in tissues surrounding the vascular bundles (Bosque – Pérez, 2000), thereby reducing the photosynthetic ability of the plant (Mesfin et al., 1995). Immunohistochemical studies have demonstrated that the virus is restricted only to vascular tissues and does not invade the apical meristems within the shoot apex. In mature leaf tissues which exhibit streak symptoms, MSV is not limited to vascular tissue (Lucy et al., 1996). Infection is more severe in younger than older plants and early infection can result in stunting, small sized, misshapen ears or complete yield loss. Infection of young plants can lead to plant death (Shepherd et al., 2010).

Maize streak virus strains, evolution and diversity

Serological and molecular studies have been conducted to investigate the relationship among MSV isolates. Peterschmitt et al. (1991) characterized MSV isolates from 11 African countries and concluded that they were of the same serotypes. At molecular level, restricted fragment length polymorphisms (RFLPs) and polymerase chain reaction (PCR) have been used to study MSV diversity. For instance, Isnard et al. (1998) used RFLP analysis and sequencing to confirm the occurrence of the mutant spectrum (Quasi-species) nature of three isolates. Additionally, Martin et al. (2001) reported that typing of MSV isolates resulted in classification into strains and subtypes. Willment et al. (2001) applied RFLP analysis involving a set of seven enzymes to type closely related MSV isolates. Also, MSV isolate from Nigeria (Mullineaux et al., 1984), Kenya (Howell, 1985) and South Africa (Lazarowitz, 1988) have been sequenced.

Currently, 11 strains of MSV have been confirmed. These include MSV – A, MSV-B, MSV-C, MSV-D, MSV-E, MSV-F, MSV-G, MSV-H, MSV-I, MSV-J, MSV-K (Varsani et al., 2008). The MSV-A₁ strain arose from isolates MSV – Gat (Kenya), MSV- Mat B (Zimbabwe), MSV-Sag (Kenya), MSV-Mat A (Zimbabwe), and MSV- Ama (Kenya). Presently, there is a high degree of MSV diversity in Nigeria (Oluwafemi et al., 2014). The MSV-A₂ strain originated in Nigeria and was found in the MSV-N isolate. The MSV – A₃ strain was first reported in Kenya and consists of two isolates namely MSV-Ken, and MSV-MtKA. The MSV-Kom, MSV-SA, and MSV-VM, which gave rise to MSV-A₄ strain originated in South Africa (Varsani et al., 2008). Additionally, the MSV-A₅ consists of the MSV-MaKD and MSV-Mat C isolates which originated from South Africa and Zimbabwe, respectively (Willment et al., 2001). The MSV-R₂ and MSV-Rev originating from Réunion gave rise to the MSV-A₆. Conversely, the MSV-Jam (Kenya), MSV-Mom (Kenya), MSV-Tas (South Africa), and MSV-VW (South Africa) were isolates of MSV-B. The MSV-C, MSV-D, and MSV-E were of South Africa origin and were detected in MSV-Set, MSV-Raw, and MSV-Pat, respectively (Varsani et al., 2008).

Out of these strains, only MSV-A strain causes economic damage in maize fields (Martin et al., 2001). Differences also occur within the same strain of the virus. Briddon et al. (1994), Martin et al. (2001) and Willment et al. (2001) reported fairly obvious differences in the genetic make up of MSV-A population in Eastern, Western and Southern Africa. The MSV-A₁ and MSV-A₄ are apparently responsible for over 95 % of MSD cases that have been investigated over the last 20 years. Consequently, these lineages could be implicated for MSD epidemics throughout Southern and East Africa (Martin et al., 2001; Willment et al., 2001; Varsani et al., 2008). Although the negative impact of MSV-A₄ is most felt in Southern Africa, MSV-A₁ is a serious threat to maize productivity in the whole of sub-Saharan Africa (Varsani et al., 2008). The low spread of MSV-A₄ is because it is less severe in maize than MSV-A₁ (Martin et al., 1999). The MSV-B, MSV-C, MSV-D and MSV-E which normally infect wild grasses are also known to produce mild infections in MSV – susceptible maize cultivars (Martin et al., 1999, 2001). These strains probably contributed to the evolution of the economically significant MSV-A strain. For example, the MSV-A₄ is different from other MSV-A lineages because it is the product of a recombination event between MSV-A and MSV-B viruses occurring in southern Africa (Martin et al., 2001).

Recombination is an important phenomenon among the Geminiviruses and this has played a significant role in the evolution of MSV. However, intra-strain or intra-species recombination is probably more prevalent during recent MSV-A evolution than inter-strain or inter-species recombination (Varsani et al., 2008). Additionally, recent investigations indicated that less than 200 nucleotides were exchanged in natural inter-strain recombination events involving MSV-A viruses (Shepherd et al., 2010). Earlier, van der Walt et al. (2009) documented that co-infection of maize plants with reciprocal MSV-A – MSV-B chimaeras resulted in rapid recombination that gave rise to MSV-A like recombinants. Therefore, this is an indication that fitness of contemporary MSV-A genotypes cannot be easily improved through inter-strain recombination (Shepherd et al., 2010). Accumulating evidences have also demonstrated that large inter-strain recombination events and smaller intra-species recombination events have played a significant role in the evolution of various MSV strains other than MSV-A. For instance, MSV-F, MSV-H, MSV-J and MSV-K probably arose via the exchange of large genomic regions of over 1000 nucleotides, amongst two or more different MSV strains (Varsani et al., 2008).

Transmission of Maize Streak Virus by Insect Vectors

Maize streak virus is exclusively transmitted by leafhoppers (*Cicadulina* spp.) (Magenya et al., 2009). Twenty two species of *Cicadulina* are known world wide. Among the eighteen species present in Africa only nine of them are vectors of MSV (Lett et al., 2002). However, only eight species of *Cicadulina* are efficient transmitters of the virus: *Cicadulina arachidis* China, *C. bipunctata* Melichar, *C. ghaurii* Dabrowski, *C. latens* Fennah, *C. mbila* Naudé, *C. parazeae* Ghauri,

C. similis China, and *C. storeyi* China (= *C. triangula* Ruppel) (Oluwafemi et al., 2007; Fajinmi et

al., 2012). Additionally, these species vary in their ability to transmit the virus. According to IITA (1986), *C. mbila* and *C. storeyi* were more active transmitters than a population of *S. ghauri* and *C. arachidis*. Asanzi et al. (1995b) also compared the transmission efficiencies of these insects and found that *C. ghauri* and *C. arachidis* required longer acquisition access time than *C. mbila* and *C. storeyi*.

The virus is vectored in a persistent manner and the latent period in *C. mbila* is 6 – 12 hours. (Storey, 1928). Conversely, a minimum of 14 – 18 hours is required by *C. storeyi* (Okoth et al., 1988). Additionally, Storey (1928) reported that *C. mbila* can acquire the virus from diseased plants within 15 seconds and inoculate it only after 5 minutes. On the other hand, *C. storeyi* can do so in 30 seconds and transmit after a 2-hour inoculation access period (IAP) (Zagre, 1983). Asanzi (1991) reported a minimum acquisition access period of 15 minutes and 1 hour for *C. arachidis* and *C. ghauri*, respectively and a maximum of 1 hour for both species. However, it is generally believed that transmission efficiency increases with increasing duration of acquisition and inoculation access periods. Asanzi et al. (1995b) observed that the differences in the minimum acquisition and inoculation access periods among *C.* species was probably due to several factors, including *Cicadulina* feeding behaviour and the titre of virus within the geographic origin of the leafhopper population. Bock (1974) was of the opinion that it depends on the differences in the permeability of the insect's gut. This report was later confirmed using polymerase chain reaction (PCR) assay in individual insect organs (Lett et al., 2002). The virus was found in the gut, haemolymph and head of a vector species (*C. mbila*), but restricted to the gut of a non-vector species (*C. chinai*) (Lett et al., 2002).

Although MSV accumulated in the alimentary canal (Lett et al., 2002; Ammar et al., 2009), and virus titre decreased considerably over time in both the gut and haemolymph. In contrast, MSV DNA copies remained stable over time in the head (probably in the salivary glands, from which the virus was released into the phloem when the leafhopper feeds on a host plant). Although studies have shown that *C. mbila* is the species most often implicated in MSD epidemics (Magenya et al., 2008). Oluwafemi et al. (2007) found that *C. storeyi* was more efficient transmitter of MSV. Within the same leafhopper species difference exist in the ability to transmit MSV. For example, Alegbejo et al. (2002) reported that females were more efficient in transmitting MSV than the male leafhoppers. Additionally, all five nymphal instars of *C. mbila* are able to acquire and transmit MSV, and this ability is retained during moulting, but transovarial transmission is not feasible (Bock, 1974).

Several studies have been conducted using *C. mbila* in order to understand MSV epidemiology (Liu et al., 2001a). The probing of *C. mbila* into plant tissue facilitates the production of a salivary secretion around the stylets which hardens into a salivary sheath that is required for ingesting plant fluids (Mesfin et al., 1995). The virus ingested into the gut is translocated to the midgut epithelial cells. It is then released into the haemocoel, and later the salivary gland and finally into the salivary ducts. The virus undergoes a latent period between 6 and 12 hours, after which it persists in the insect throughout its entire life (Bosque-Pérez, 2000; Alegbejo et al., 2002). Mesfin et al. (1995) observed that *Cicadulina* spends more time acquiring MSV from tissues other than phloem, possibly the mesophyll, resulting in more virus acquisition. However, virus concentration does not increase in the insect following acquisition (Lett et al., 2001). Mesfin and Bosque-Pérez (1998) documented that *C. storeyi* spend significantly shorter time while feeding on the mesophyll of MSV- infected plants than on healthy ones.

Epidemiology and Host Range of Maize Streak Virus

Maize streak virus is spread by *Cicadulina* species and its incidence is closely associated with vector population dynamics, which in turn, depends on rainfall, temperature and availability of alternative hosts (Atiri et al., 2000). Increased epidemic of MSD is attributable to several factors

including staggered planting which encourages higher incidence of MSV – A in early planted maize and devastation of seedlings that emerge in successive plantings (Dabrowski et al., 1991); the population density of wild grasses which harbour both MSV-A and *Cicadulina* vectors (Autrey & Ricaud, 1983); the availability within leafhopper populations of a high percentage of MSV transmitters; and environmental factors that favour long distance movement of leafhopper (Rose, 1978). Continuous cultivation of maize all the year round also favours leafhoppers population build up and increased MSD epidemiology (Bosque-Pérez, 2000). Additionally, MSV is more severe where ultra-short season hybrids are cultivated. The shorter growing time of hybrids enables farmers to cultivate a second crop which provides continuous hosts for leafhoppers. Thus, temporal overlap of these two crops results in green bridge (Kloppers, 2005). Cultivation of maize in a monoculture aggravates MSV incidence via its leafhopper vectors. Other cases of MSV epidemic include the shift in natural parasites, introduction of new susceptible cultivars and the increased area under maize cultivation (Bosque-Pérez, 2000).

Although maize is a favoured host for *Cicadulina* feeding, several weed species have been implicated in leafhopper abundance and consequently MSD incidence. The leafhoppers preferentially breed on these grasses. Unfortunately, about 70 % of the over 138 grass species on which leafhoppers feed are potential MSV hosts (Konaté & Traoré, 1992). Varsani et al. (2008) observed that the maize adapted MSV-A strain and the closely related grass-adapted MSV-B strain seem to be more pathogenic on the genus *Digitaria*. Earlier, Mesfin et al. (1992) documented that virus isolates are not readily transmissible to susceptible maize field. Therefore, an adequate understanding of the survival of the grasses that harbour vectors and MSV isolates infecting maize during the dry season (November to March), particularly in low – lying hydromorphic soils (Fadamas), provides detailed information on MSV ecology and epidemiology (Bosque-Pérez, 2000). The impact of temperature and wind on the movement and feeding of leafhoppers also affects leafhopper population (Asanzi et al., 1995b), and the spread of MSV. Moreover, studies in West Africa revealed that differences in species distribution and population dynamics of *C. mbila* were related to variation in soil types, altitude and seasons. Also, increase in nutritive status of host plants encourages leafhopper population build up and spread of the virus (Asanzi et al., 1995a).

The virus has a wide host range, attacking cultivated and wild plants. The maize adapted MSV - A strain infects more than 80 plants species in the family Poaceae (Damsteegt, 1983) but it is probable that ancestral MSV – A viruses that first infected maize specifically adapted to infecting species in the genus *Digitaria* (Varsani et al., 2008). Maize streak virus also infects wheat (*Triticum aestivum* L.) barley (*Hordeum vulgare* L.), rye (*Secale cereale* L.), oats (*Avena sativa* L.), sugarcane (*Saccharum officinarum* L.), finger millet (*Eleusine coracana* [L.] Gaertn.), pearl millet (*Pennisetum americanum* [L.] R. Br.), and sorghum (*Sorghum bicolor* [L.] Moench) (Varsani et al., 2008). It has been observed that MSD is not economically important on these crops with the exception of sugarcane streak disease (Van Antwerpen et al., 2008).

Management of Maize Streak Disease

Cultural control

Agronomic practices can be used to check the occurrence and spread of MSV (Bosque-Pérez et al., 1998). These include the use of 'barrier' of bare ground between early and late planted maize fields to reduce leafhopper movement and subsequent spread (Bosque-Pérez, 2000). Control can also be achieved through avoidance of maize plantings downwind from older cereal plants, and the use of crop rotation to reduce invasion by viruliferous leafhoppers (Barrow, 1992). Others include early planting before the outbreak of leafhoppers. Planting date could be adjusted to avoid migrating leafhoppers landing on young plants. However, in areas with erratic and unreliable rainfall, it is not feasible to commence planting of maize, before the onset of rains, or even late planting (Magenya et al., 2008).

Effective soil nutrient management is also recommended to curtail the negative impacts of MSV. Additionally, inhibition of plant viral agents strongly correlates to plants deficiency in nutrients necessary for viral growth and reproduction. This trend seems to apply particularly for both nitrogen and phosphorus elements (Byerlee & Heisey, 1996). The form of fertilizer applied also influences viral agents. Contrast to nitrate fertilizers, alkaline phosphate fertilizers have a beneficial effect against viral diseases, such that, by promoting maturity, they hasten the stage of resistance in the plant (Byerlee & Heisey, 1996). The mechanism is such that while P simultaneously encourages plants growth and virus concentration, K increases enhances plant growth and reduces viral concentration (Byerlee & Heisey, 1996). The level of nitrogen in plant tissues is the phytophagous insects principal attracting components. Thus, a positive correlation has been observed between migrations and reproduction of leafhoppers with the levels of soluble nitrogen content of host plants (Alegbejo & Banwo, 2005).

Biological control

The potential of utilizing natural enemies (predators and parasitoids) and entomopathogenic microbes for leafhoppers control has been accomplished in Asian countries (Mitsuhashi et al., 2002). Thus, a number of parasitoids, predators and entomopathogens of important Cicadellid pests including *Cicadulina* spp that occur in India have been identified (Singh et al., 1993). However, there are no detailed attempts to identify and utilize biological control agents of leafhoppers in Africa (Magenya et al., 2008).

Chemical control

Maize streak virus vectors can be controlled by the application of systemic insecticides to the planting furrows during maize planting or even more effectively, as seed treatments (Shepherd et al., 2010). Additionally, Dahal (1997) documented that leafhopper populations seemed to be controlled by the carbamate class of insecticides. However, control is only partial since the recurring influx of migrant hopper populations re-infects the crop after each application (Magenya et al., 2008). Moreover, the Food and Agriculture Organization (FAO) and the World Health Organization (WHO) have reported various cases of ill-health associated with those applying pesticides in the maize-based systems in Africa (WHO, 1986; FAO, 1998). It has been documented that a million people applying pesticide are poisoned in Africa annually, with 20,000 cases resulting in death (WHO, 1986). Pesticide poisoning is a serious problem among the small – scale farmers, who lack adequate knowledge on pesticides and who fail to wear appropriate protective clothing (Matthews et al., 2003). Additionally, most of the resource-poor farmers cannot afford the pesticide costs (Mathews et al., 2003). Although appropriate insecticide use will continue to play a vital role in maize viral disease / vector control; non – chemical alternatives remain the most cost effective, safest and environmentally suitable approaches for tropical resource- poor farmers (Matthews et al., 2003).

Host Plant Resistance

Adoption of resistant cultivars is the most appropriate and cost – effective approach for controlling the virus (Muiru et al., 2015). Resistance in maize was discovered thirty years after its first report, in the variety 'Peruvian yellow' (Fielding, 1933) in South Africa. Subsequently, tolerance was found in other materials and utilized to develop a hybrid population, P × H (Soto et al., 1982). Resistance to MSV was also found in East Africa (Storey and Howland, 1967). Similarly, the International Institute of Tropical Agriculture (IITA), Nigeria discovered resistance within IITA's maize population Tropical Zea – Yellow (TZ-Y) and the local variety Revolution collected by the Institute Recherche Agronomique Tropicale (IRAT) in Réunion (Soto et al., 1982). Over the years, maize varieties have been developed at IITA that combine high levels of MSV resistance with desirable agronomic characteristics including high yield potential, preferred grain type and early maturity (Kim, 1982; Efron, 1984). These streak- resistant populations are suitable for cultivation in different ecological zones and have good consumer acceptability characteristics (Shoyinka, 1988).

The severe outbreak of MSD in northern Nigeria in 1984 allowed IITA to select in farmers' fields some extremely rare maize populations that stood up to infection with the virus.

Recently, genetic engineering which involves the use of transgenic plants has been demonstrated (Shepherd et al., 2014). Shepherd et al. (2007a, b) developed MSV resistant maize using dominant negative mutants of the virus' replication-associated protein gene (Rep). The MSV resistance inherited by transgenic plants was effective up to the T₃ generation. It was observed that the transgenic plants were able to delay streak symptom development, decrease severity and enhance survival rate following infection by the virus. The investigation was based on the understanding that multifunctional Rep is essential for MSV replication but mostly expressed in low levels. Since Rep acts as an oligomer, it becomes very valuable for pathogen-derived resistance. Therefore, where target plants naturally express mutant forms of the Rep gene, the invading virus will be rendered ineffective.

Conclusion and Recommendations

Maize is one of the cereals that contribute substantially to food security in sub-Saharan Africa but productivity is seriously constrained by maize streak disease. The impacts of the virus are more felt by smallholders maize cultivators who have little or no access to agronomic inputs that could limit infections. Adoption of MSV-resistant/tolerant maize cultivars which is the best management option is recommended for maize growers in the region. Further, there is need for multidisciplinary approach and continuous collaboration from maize breeders, entomologists and plant virologists in order to eradicate MSD menace in the region.

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