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Review

Significance and transmission of maize streak virus disease in Africa and options for management: A review

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The distribution of leafhopper vector populations and the viral diseases they vector are inherently influenced by agro-ecological factors. This review discusses the distribution and classification of MSV strains, their vectors and how agro-ecological factors mediate the prevalence of MSV disease. Important strains of MSV have been characterised on the African continent using molecular techniques, however, evidence on the characterisation of geographically separate populations of *Cicadulina* spp. and the viral strains they vector is inadequate. The potential of applying knowledge on the influence of soil nutrients, altitude and temperature on the biology of maize streak virus (MSV) / vector populations is discussed. This review has considered the potential of using soil nutrients in determining nutritive status of maize host plants and its effects on population dynamics of *Cicadulina mbila* Naudé (Homoptera: Cicadelidae), an important vector of MSV disease and the expression of MSV disease. Future research options that can provide information on the influence of soil fertilization on the behaviour of characterised vector populations, MSV disease transmission, virulence and disease expression are proposed.

Key words: Leafhoppers, *Cicadulina* spp., maize streak virus, molecular characterisation, soils, agro-environmental factors.

INTRODUCTION

Maize production in Africa

Maize (*Zea mays* L.) is a monoecious plant grown from latitude 58 °N to 40 °S, adaptable to a wide range of agro-ecological zones in Africa (Hallauer and Miranda, 1988). Over 100 million people in Africa utilise maize as a staple food crop (Byerlee and Heisey, 1996), including as a constituent in livestock feed (Romney et al., 2003). Its acreage in tropical highlands (1800–2800 meters above sea level (masl)) is 1.7, in the subtropics and mid-altitude zones (1200–1800 masl) 8.1, and in lowland tropics (< 1200 masl) 12.3 million ha (Pingali, 2001).The International Food Policy Research Institute (2000) projected the annual maize demand in sub-Saharan Africa to be 500 million tons by 2020 which is twice that of today.

Yield diminishing factors

The yield potential for Sub-Saharan Africa is 5 tonnes/ha in tropical highlands, 7.0 in subtropical and mid-altitude zones and 4.5 in tropical lowlands, compared to the current yields of 0.6, 2.5 and 0.7 tonnes/ha respectively (Pingali, 2001). This large yield gap is attributable to both abiotic and biotic constraints (Wambugu and Wafula, 2000). The major abiotic constraint is drought that causes an annual yield loss of about 15% (Kamara et al., 2003), while the second most important constraint is nitrogen

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and phosphorus deficiency (Nziguheba et al., 2002; Whitbread et al., 2004). Biotic factors that reduce maize yields in Africa are stemborers, the parasitic weed Striga and maize streak virus (MSV).

The latter reportedly causes yield losses that range from a trace to almost 100% (Kyetere et al., 1999; Alegbejo et al., 2002). The other diseases that affect maize include leaf blight, rusts, stalk and ear rots, and systemic foliar diseases (Alegbejo et al., 2002).

Incidence of maize streak virus

The MSV disease is a significant maize disease in countries in Eastern and Southern Africa (e.g. Kenya, South Africa, Zimbabwe and Zaire), and West Africa (e.g. Nigeria) (Thottappilly et al., 1993; Bosque-Perez et al., 1998; Martin et al., 1999). The disease manifests in a wide range of elevations; from sea level up to elevations of 2000 m (Efron et al., 1989). And its occurrence is severe after periods of irregular rains (Welz et al., 1998; Bosque-Perez et al., 1998). Severe epidemics occurred: 1983-1984 in West Africa, 1988-1989 in Kenya (Rossel and Thottappilly, 1985; Njuguna et al., 1990; Welz et al., 1998). While other epidemics occurred in Nigeria in 1971, 1973, 1976 (Fajemisin and Shoyinka, 1976; Kim et al., 1981; Effron et al., 1989), as well as in countries including, the Democratic Republic of Congo, Zambia, Angola and Mozambique. The MSV epidemics are noted to be frequent in the tropics due to alternate and successive cropping of maize plant hosts and the presence of other hosts such as wild grasses (Mesfin et al., 1995). Often infection of the crop by the MSV disease at seedling stage results in no ear formation, but later infection leads to undersized and poorly filled ears (Kaitisha, 2001). Infection of a maize crop in the first three weeks of planting often results in 100% yield loss (Bosque-Perez and Buddenhagen, 1999), equally, a maize crop that is planted at the end of a rainy season seems to be most severely affected. It is therefore probable that a close relationship exists between leafhopper (*Cicadulina* spp) populations, weather, host plants, and infection rates in maize host (Atiri et al., 2000). This review discusses the interaction of leafhoppers, MSV disease and agro-environmental factors.

Maize streak virus disease

Maize streak virus disease was initially named as ‘mealie variegation,’ but later renamed ‘maize streak virus disease’ in 1925 (Storey, 1925). It is the most economically significant member of genus *Mastrevirus* of the family Geminiviridae (Willment et al., 2001; Bigarré et al., 1999; Bosque-Perez, 2000; Schnippenkoetter et al., 2001). MSV is indigenous to African grasses and is transmitted by leafhoppers of the genus *Cicadulina (*Homoptera: cicadellidae) (Markham et al., 1984; Bosque-Pérez et al., 1998). Other African streak viruses identified include Panicum streak virus (PanSV), sugarcane streak virus (SSV), sugarcane streak Mauritius virus (SSMV) and sugarcane streak Egypt virus (SSEV) (Bigarré et al., 1999; Wilment et al., 2001). Several studies have closely related MSV isolates from Africa and the neighbouring Indian Ocean islands of Madagascar, Mauritius and La Réunion have been Identification (Pernet et al., 1999; Willment et al., 2001).

THE BIOLOGY OF MSV

MSV strains and their host plants

Maize streak virus infects a range of wild and cultivated grass species. The cultivated crops include: maize, rice, wheat, oats, barley, rye, finger millet, sorghum, and sugarcane, while the affected wild grass species belong to the following genera: *Sporobolus, Eleusine*, *Paspalum*, *Brachiara, Imperata*, *Rottboelia, Dactylocterium, Eragrostis, Diplachne, Leptochloa, Setaria, Tragus, Euchlanaena* and *Coix* (Markham et al., 1984; Mesfin and Hollander, 1995; Bigarré et al., 1999; Bosque-Perez, 2000; Willment et al., 2001). Previously, all viruses causing the "streak disease" in maize, grasses and sugarcane in Africa were included as strains of MSV (Pinner et al., 1988). But the application of polyclonal and monoclonal antibodies demonstrated the inherent serological differences between MSV isolates from different host plant species (Pinner and Markham, 1990; Peterschmitt et al., 1991; Mesfin et al., 1992). Subsequent studies using polymerase chain reaction (PCR) amplification, and the sequencing of viral DNA have led to the delineation of a number of distinct MSV genotypes and their geographical distribution in Africa (Rybicki et al., 1998; Martin et al., 2001; Willment et al., 2002). This delineation has separated strains of MSV with differential virulence into maize adapted isolates (MSV), panicum streak virus (PanSV) and sugarcane streak viruses (SSV) (Peterschmitt et al., 1996; Pernet et al., 1999; Bosque-Peréz, 2000) Virus Isolates of MSV from maize hosts share nucleotide sequence identity that is greater than 95%, while isolates from wheat and annual grasses are less related sharing between 89 and 78% nucleotide identity with isolates from maize hosts (Rybicki et al., 1998; Willment, 1999; Martin et al., 2001; Willment et al., 2002).

Martin et al. (2001) determined the geographical and host distributions of MSV isolates using the available full-length MSV sequences to classify MSV isolates into tentative strain and subtype groupings. The above classification utilized eight maize-type MSV isolates which showed 10% nucleotide sequence divergence by grouping them into MSV-MatA, MSV-MatB, MSV-MatC, MSV-Sag, MSV-Ama, MSV-Gat, MSV-MtKA, and MSVMakD. Similarly, cloning and sequencing of grasstype MSV isolates lead to MSV-Mom and MSVJam,

Table 1. Origin of MSV isolates and the grouping of strains from different plant hosts.

S/N	MSV isolate	Country of origin	Plant host	Strain grouping
1	MSV – Gat	Kenya	Maize	MSV – A_1
2	MSV – Mat B	Zimbabwe	Maize	
3	MSV – Sag	Kenya	Maize	
4	MSV – MatA	Zimbabwe	Maize	
5	MSV – Ama	Kenya	Maize	
6	MSV – Ns	Nigeria	Maize	MSV – A_2
7	MSV – ken	Kenya	Maize	MSV – A_3
8	MSV – MtKA	Kenya	Maize	
9	MSV – Kom	South Africa	Maize	MSV – A_4
10	MSV – SA	South Africa	Maize	
11	MSV – VM	South Africa	Maize	
12	MSV – MaKD	South Africa	Maize	MSV – A_5
13	MSV – MatC	Zimbabwe	Maize	
14	MSV – R2	Réunion	Maize	MSV – A_6
15	MSV – Rev	Réunion	Maize	
16	MSV – Jam	Kenya	Grass	MSV - B
17	MSV – Mom	Kenya	Grass	
18	MSV – Tas	South Africa	Maize	
19	MSV – VW	South Africa	Maize	
20	MSV – Set	South Africa	Maize	MSV – C
21	MSV – Raw	South Africa	Grass	MSV – D
22	MSV – Pat	South Africa	Grass	MSV – E

Source: Martin et al. (2001).

MSV-Raw, and MSV-Pat groupings. In the preceding two cases, a pair wise nucleotide sequence identity matrix was constructed using the 12 full-length sequences of the above-mentioned isolates and 10 other full-length MSV sequences available in the MSV GenBank. An assessment of the MSV isolates sharing nucleotide sequence identity greater than 94% contained representatives of five MSV strains named MSV-A, -B, -C, -D and –E (Table 1). The MSV-A isolates were further subdivided into subtype groups on the basis of sharing greater than 98% sequence identity and confirmed by phylogenetic analysis (Martin et al., 2001). The majority of MSV isolates obtained from maize belong to strain A isolates named: MSV-A1, -A2 -A3, -A4, -A5 and -A6 (Table 1). Substantial differences have been noted in the subtype composition of the MSV-A populations infecting maize in different parts of Africa (Rybicki et al., 1998; Martin et al., 2001; Willment et al., 2002). Geographical distributions of MSV isolates indicates that Subtype A1 isolates occur in east and south, while subtypes A2, A3, and A4 occur only in Western, Eastern, and Southern Africa, respectively (Figure 1). Although recombination among mastreviruses was initially thought to occur at a lower frequency, other African streak viruses (Padidam et al., 1999; Martin et al., 2001; Schnippenkoetter et al., 2001). In fact Padidam et al. (1999) suggested that recombination between geminiviruses has been a major contributing factor to the recent emergence of a number of devastating viral crop diseases worldwide.

Virulence of MSV strains

Martin et al. (2001) detected important differences between the virulence of different MSV-A subtypes in maize. Subtypes A1, A2, and A5 isolates produce the severest symptoms, subtypes A3 and A6 isolates produced intermediate symptoms, while subtype A4 isolate produced the mildest symptoms. Severe isolates cause earlier symptoms with wider and more chlorotic streaks than the mild isolates (Bosque-Pérez, 2000; Martin et al., 2001). The MSV-B, -C, -D, and -E isolates on the other hand, were substantially less severe than the MSV-A isolates. The extent of MSV diversity, host specificities, geographical distributions, and virulence in maize is fairly documented (Rybicki et al., 1998; Schnippenkoetter et al., 2001; Martin et al., 2001; Willment et al., 2002). However, little is known about the comparative transmission of the different strains of MSV by the different geographical populations of *C. mbila* vector.

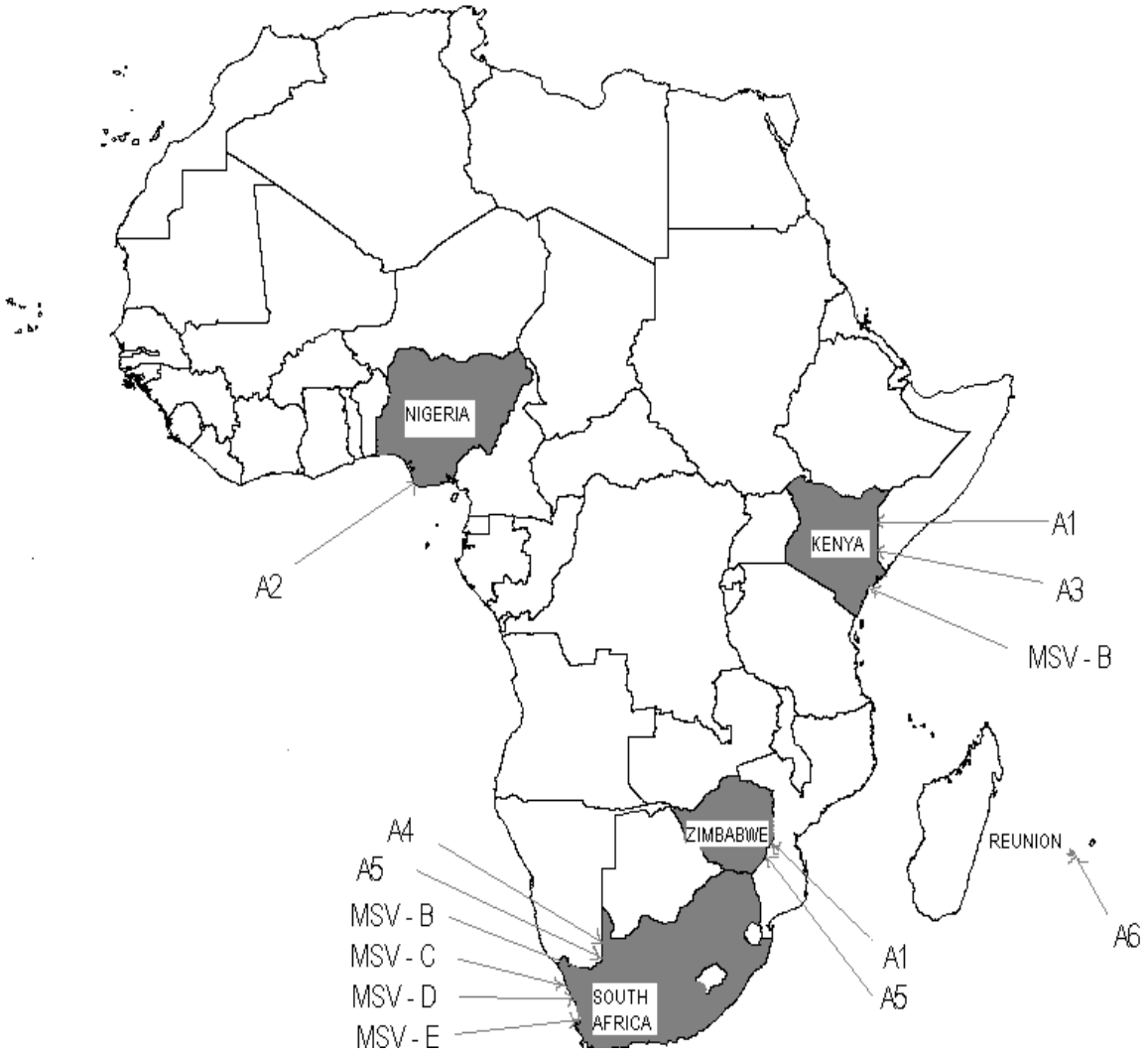

Figure 1. Map of Africa showing the origin of the different MSV isolates. Source: Martin et al. (2001).

MSV symptom expression and damage in maize crop

The maize plant is susceptible to MSV disease from emergence to flowering. The virus has been reported to infect all cell types of the host plant, where streak symptoms manifest only on inoculated leaves and or on leaves produced after infection of the plant (Thottappilly et al., 1993). The specificity of MSV infection in maize tissues shows that the virus occurs only in vascular tissues and does not invade the apical meristems within the shoot apex (Lucy et al., 1996). However, in mature tissues which display streak symptoms, the virus is not restricted to vascular tissues. In fact the MSV coat protein and both positive and negative strands of the MSV genome were detected in mesophyll, bundle sheath cells and vascular-associated parenchyma of the leaf (Lucy et al., 1996). Symptoms of MSV tend to appear quicker in younger maize plants: 3 to 5 days in a one-week-old plant, and 7 to 9 days in a 9-week-old plant (Mesfin et al., 1995). The symptoms begin as small and spherical, chlorotic spots: 0.5 to 2.0 mm in diameter on younger leaves of the maize plant (Barrow, 1992; Guthrie, 1989; Bosque-Pe´rez et al., 1998) which later coalesce into continuous longitudinal chlorotic streaks, mainly along the veins of the leaf laminae (Okoth et al., 1987). In severely affected plants, chlorotic stripes may merge into pale green, or yellow or even white appearance on the leaf surface of the plant. The highly sensitive maize varieties develop chlorosis of the entire leaf lamina, followed by plant death, particularly if infection occurs at an early stage of plant growth (Mesfin et al., 1995; Bosque-Pérez et al., 1998). The streak pattern is a result of the failure of chloroplasts to develop in tissues surrounding the vascular bundles (Barrow, 1992; Bosque-Perez, 2000), and impairing the photosynthetic ability of the plant (Mesfin et al., 1995). In general, the affected maize plants

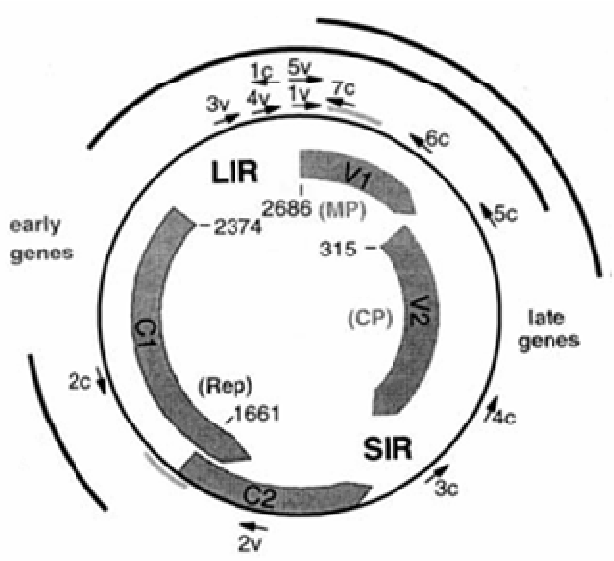

Figure 2. Genome map of MSV indicating the position of the open reading frames (ORFs) and the function of the products encoded by them. ORF V1 encodes the MP, ORF V2, the CP. ORFs C1:C2 encode the replication initiation protein (Rep) fusion protein. The co-ordinates of the initiation codons of the ORFs are shown V, virion-sense; C, complementary-sense; LIR, SIR, large and small intergenic regions, respectively. Source: Wright et al. (1997).

may be shorter, have less vigour and produce smaller grains and ears (Okoth et al., 1987). Maize yield reduction due to MSV in susceptible varieties often exceeds 70% (Bosque-Pérez et al., 1998).

Morphology of the MSV virus

Geminiviruses in the genus Mastrevirus are plant-infecting DNA viruses with a monopartite genome consisting of circular single stranded DNA (ssDNA), encapsidated in a characteristic geminate morphology, that has a quasi-icosahedral particle of 18 x 30 nm in size (Bosque-Perez, 2000; Alegbejo et al., 2002). The MSV genome is composed of a DNA molecule of ca. 2.6 kb which codes for four potential products (Isnard et al., 1998). These viruses rely on the plant host cell machinery for DNA replication and transcription which takes place in non-dividing host cells (Lucy et al., 1996; Munoz-Martin et al., 2003). It is not yet known how mastreviruses achieve replication and expression of their genomes in differentiated cells, but some clues are inferred from the proteins which they encode.

MSV transcription

The MSV viral transcription process in the nucleus of host plant cell is initiated in the intergenic region (IR) at a conserved non-nucleotide sequence and involves a rolling circle mechanism (Horvath et al., 1998; Gutierrez, 2002). The MSV genome has four open reading frames (ORFs): the (C)-sense C1 and C2 and the complementary virion (V)-sense V1 and V2 (Lazarowitz, 1992; Liu et al., 1999; Gutierrez, 2002). The non-encapsulated double-stranded (ds) DNA of the virus provides a bidirectional template for transcription that initiates from the large intergenic region (LIR) of the genome and terminates in the small intergenic region (SIR) (Wright et al., 1997; Gutierrez, 2002) to produce complementary – (C) and virion – (V) sense transcripts (Figure 2). Early genes are C-sense transcripts while late gene products are the movement protein (MP) and coat protein (CP) (Figure 2). The virion strand (V-sense) codes for the coat (CP) and the movement proteins (MP), while the complementary strand (C-sense) produces the two non-structural proteins RepA (replication associated protein) and Rep (replication initiation protein) (Liu et al., 1999; Nikovics et al., 2001;Gutierrez, 2002; Munoz-Martin et al., 2003). The Rep proteins, with a molecular size of ca. 41 kDa is the sole viral protein essential for replication of dsDNA in cell protoplast and are responsible for initiating the rolling circle replication, while RepA is important for the process (Wright et al., 1997; Horvath et al., 1998; Nikovics et al., 2001; Gutierrez, 2002; Muñoz-Martın et al., 2003). However, the mechanisms by which Rep and RepA regulate V-sense gene expression are not established. The MP and CP (Figure 2) are necessary for systemic infection and pathogenicity of the virus in the plant (Wright et al., 1997; Liu et al., 1999; Liu et al., 2001; Nikovics et al., 2001; Gutierrez, 2002). The V1 has been identified as the gene that codes for the movement protein believed to play a role in cell to cell movement of MSV in infected plants (Gutierrez, 2002). While V2 codes for coat protein that is required for systemic spread of the virus and subsequent symptom development in plants (Boulton et al., 1993; Liu et al., 1999). Biological assays have demonstrated that virus replication occurs in the host cell nucleus, whereas free virus is available to the vectors outside the nucleus of mesophyll and parenchyma cells (Markham et al., 1984).

BIOLOGY OF THE MSV VECTOR

Nine of the eighteen species of *Cicadulina* identified in Africa are vectors of MSV (Mesfin et al., 1995; Bosque-Pérez, 2000; Smith et al., 2000; Lett et al., 2002). *C. mbila* Naudé is the most efficient MSV vector in several African countries (Ethiopia, South Africa, Kenya, Nigeria, Tanzania and Mauritius) (Markham et al., 1984; Dabrowski et al., 1991; Downham et al., 1997; Asanzi et al, 1995a; Mesfin and Hollander, 1995). It is also the most abundant across all the agro-ecological zones where maize is grown (Asanzi et al., 1995a). The host-plant range of *C. mbila* includes cultivated crops such as maize, rice, wheat, oats, barley, rye, finger millet, sorghum, and sugarcane, and wild grass species in the following genera: *Sporobolus, Eleusine*, *Paspalum*, *Bra-*

chiara, Imperata, *Rottboelia, Dactylocterium, Eragrostis, Diplachne, Leptochloa, Setaria, Tragus, Euchlanaena* and *Coix* (Markham et al., 1984; Mesfin et al., 1995; Bigarré et al., 1999; Willment et al., 2001; Alegbejo et al., 2002). The developmental time, fecundity and longevity of *C. mbila* is influenced by temperature and the quality of host plant and changes between populations of different geographic origins. Okoth et al. (1987) reported that at 25 ^{o}C eggs of *Cicadulina* spp. hatch after 7-10 days and nymphal development is completed in 14 to 20 days. The mean adult life span at 26^{o}C is in the range of 8-28 days for males and 14-33 days for females.

MSV transmission by *C. mbila*

Maize streak virus is obligatory transmitted by *Cicadulina spp.* (Mesfin and Hollander, 1995). In fact several studies have demonstrated that *C. mbila* is more successful in acquiring MSV from maize than from other hosts, suggesting the occurrence of plant host adaptation (Mesfin and Hollander, 1995; Bosque-Pérez, 2000; Alegbejo et al., 2002). The populations *of C. mbila* are composed of genetically distinguishable active and inactive vector individuals with differences in their ability to transmit the MSV virus (Bosque-Pérez, 2000; Alegbejo et al., 2002). Proportion of MSV transmitters in populations of *C. mbila* was reported to vary between 60 and 100% (Markham et al., 1984). The percentage of active transmitters among females was 2-3 times larger than that among males (Alegbejo et al., 2002). Bock (1974) demonstrated that while all five nymphal instars of *C. mbila*, are able to acquire and transmit the virus, this ability is retainable during moulting, but can not be transferred through the egg.

C. mbila has been reported to spend significantly shorter time while feeding on virus infected plants than on uninfected ones (Mesfin and Bosque-Pérez, 1998). After leafhopper acquisition of MSV, the virus persists in the vector throughout its life span. The characteristic of acquiring the virus by the leafhopper is a genetically inherited trait dependant on the permeability of the insect's gut (Bock, 1974). Differential transmission of the virus by *Cicadulina spp*. indicates that the gut acts as a physical barrier for viral transmission (Bock, 1974; Lett et al., 2002). Furthermore, change in virus titre in the host plant alters the vector's ability to acquire and transmit the virus (Bosque-Perez, 2000). It has been demonstrated that the virus can circulate in its vector without viral replication (Boulton and Markham, 1986). A simple sex linked dominant gene could be responsible for the transmission trait in the vector. Bock (1974) also documented that individual insects of an active race of *C. mbila* do not exhibit equal transmission efficiency.

Acquisition of MSV by the vector

In the recent past, studies on the feeding behaviour of *C. mbila* as a vector of MSV have been conducted in order to understand MSV virus epidemiology (Lett et al., 2001). *Cicadulina* acquires MSV in a short time during initial contact with infected plants (Storey, 1938). The probing of *C. mbila* into plant tissues triggers the production of a salivary secretion around the stylets which harden into a salivary sheath that is essential for ingesting plant fluids (Mesfin et al., 1995). Unlike aphids, *C. mbila* does not puncture cells without their destruction, but instead the insect severely damages the host plant cells, meanwhile ingesting the geminivirus particles accumulated in mesophyll and parenchyma leaf cells (Tjallingii and Gabrys, 1999; Lett et al., 2001).

The acquisition access period (AAP) defined as the time necessary for the leafhopper to reach the mesophyll of the leaf and ingest the virus is about 15-30 s for *C. mbila* (Okoth et al., 1987). The circulative pathway of virus movement in the leafhopper involves ingestion into the gut followed by uptake by midgut epithelial cells (Gray and Banerjee, 1999). The virus is then released into the haemocoel, and later the salivary glands and finally into the salivary ducts. Once in *C. mbila*, the MSV has a latent period of between 6–12 h, after which the virus persists in the leafhopper throughout its life span (Bosque-Perez, 2000; Alegbejo et al., 2002). Leafhoppers reportedly spend more time ingesting from tissues other than the phloem, possibly the mesophyll, resulting in more virus acquisition (Mesfin et al., 1995). The efficiency of virus transmission by *C. mbila* increases with acquisition time; however, the virus titre does not increase in the insect after acquisition which is consistent with circulative non-propagative viral transmission (Okoth et al., 1987; Smith, et al., 2000; Lett et al., 2001).

MSV inoculation by the vector

Inoculation of MSV into maize by *Cicadulina* is associated with injection of saliva into phloem tissues (Kimmins and Bosque-Pérez, 1996; Gray and Banerjee, 1999). *C. mbila* is known to move to different plant tissues while feeding, and this behaviour is dependant on nutritive and health status of host plant (Mesfin et al., 1995; Lett et al., 2001). Salivary sheaths produced during feeding usually either reach the vascular bundle, remain unbranched or terminate in the phloem (Mesfin et al., 1995). Phloem ingestion is not always associated with every probe but is correlated with the production of honeydew. Studies on stylet route using electrical penetration graphs (EPG) did not differentiate ingestion from mesophyll or from vascular bundle sheath cells (Lett et al., 2001).

Lett et al. (2001) demonstrated that short-duration insect stylet probes that never reach the phloem did not enhance virus transmission, which is consistent with the process of inoculation of persistent viruses such as MSV. *C. mbila* requires a minimum of 15 s to acquire the virus but for successful inoculation of MSV into a healthy maize

plant, it takes about 5 min since this can only occur when the vector successfully salivates into the phloem tissue (Gray and Banerjee, 1999; Alegbejo et al., 2002). This information agrees with the observation that MSV did not multiply at the feeding point of *C. mbila* following inoculation on a maize leaf (Peterschmitt et al., 1991), and that the virus moved to other parts of the plant from the inoculated leaf in about two hours. The reported minimum inoculation access period (IAP) for *C. mbila* is 5 min, but usually takes 1-3 h from the initial access (Asanzi et al., 1995a). There is no evidence that the virus multiplies in the vector, therefore the quantity of the virus acquired by the insect is crucial in relation to transmission (Markham et al., 1984). This information implies that successful viral transmission depends on the availability of the virus in plant, and the dose acquired. In general, the longer the viruliferous insects are allowed to feed on healthy plants, the more likely the virus is to be transmitted (Okoth et al., 1987). In addition, virus transmission efficiency increased with the lengthening of AAP and IAP (Asanzi et al., 1995a).

MSV–infection of maize plants changes vector feeding behaviour with implications on increased likelihood of virus acquisition. Leafhoppers feed longer on leaf mesophyll tissues, in infected plants resulting in more MSV acquisition (Mesfin and Bosque-Pérez, 1998). Studies on *C. mbila* feeding behaviour provide information on interactions between the maize host plant and the insect vector. This information is useful in determining virus transmission efficiency (Lett et al., 2001). Differences in the minimum acquisition and inoculation periods among *Cicadulina* species are attributed to several factors, including leafhopper feeding behaviours and the titer of virus in geographically separate maize plant populations (Asanzi et al., 1995a; Alegbejo et al., 2002).

EFFECT OF ENVIRONMENTAL FACTORS AND PLANT HOST ON MSV VECTOR

Biotic (i.e. host plant) and abiotic (temperature, wind) factors have been reported to influence the movement and feeding of leafhoppers (Asanzi et al., 1995a). These environmental conditions together with the nutritive status of host plants, affect leafhopper populations and, possibly, vector population composition, which in turn play an important role in the spread of maize viruses (Dabrowski et al., 1991; Asanzi et al., 1995a). Studies by several workers in Zimbabwe and Nigeria established that population densities of *Cicadulina* species were low at the onset of the rains and then rose gradually as maize host plants became abundant and appropriate nutritive levels (Dabrowski et al., 1991; Asanzi et al.1995b; Bosque-Pérez, 2000). Increase in nutritive status of host plants coincided with the increase of the percentage of infective *Cicadulina* leafhopper individuals (Asanzi et al., 1995a).

In West Africa, differences in species distribution and population dynamics of *C. mbila* were related to variation in soil types, altitude and seasons (Asanzi et al., 1995b). However, in Zimbabwe, MSV epidemics and behaviour of *Cicadulina* leafhopper species are closely associated with the environmental conditions (Rose, 1972). Atiri et al. (2000) postulated that if vector efficiency varies with species and/or populations, then virus transmission rates will depend on the predominant vector species/ populations. In this regard, the epidemiology of MSV would be an expression of the biology and dispersal of *Cicadulina spp* populations (Asanzi et al., 1995a; Mesfin and Hollander, 1995). There is a need to determine the interrelationship between the geographically separated *C. mbila* populations and the predominant MSV strains which they transmit as part of understanding the spread of MSV disease.

CHARACTERIZATION OF THE VECTOR AND VIRUS

The identification of vector species and population composition is important in the assessment of the transmission of MSV viral strains (Abdullahi et al., 2004). This identification is crucial in view of the reported differences in viral transmission abilities within *C. mbila* populations (Alegbejo et al., 2002). Presently reliable molecular biological techniques are available for determining the composition of vector populations and viral strains which they transmit.

Loxdale and Lushai (1998) reviewed the various molecular characterization tools available for use in solving entomological complications of population dynamics. They divided the available electrophoretic marker techniques into protein and DNA markers. Examples of the application of protein markers have included the identification of parasitoids within insect hosts and the use of allozymic variation in distinguishing cultures of parasitoids occurring in specific geographical locations (Pinto et al., 1992; Loxdale and Lushai, 1998). The use of protein markers is cheaper than the use of DNA markers. However, their main disadvantage is the low detectable variability of the enzyme loci and weak banding patterns. On the other hand, the determination of the population genetics of clonal cereal aphids and parental genetic relationships in the solitary bee *Megachile rotundata* used DNA markers such as DNA fingerprinting (Loxdale and Lushai, 1998). The major disadvantage of DNA fingerprinting is the use of radioactive probes, which take time to develop and can be difficult to analyze. Differences between insect populations from different tritrophic systems have been determined using randomly amplified polymorphic DNA (RAPDs) (Daza-Bustamante et al., 2002). Zambrano et al. (2003) used RAPDs in the study of genetic relationships between sugar cane genotypes that were resistant to particular viral diseases. The major drawback of RAPDs is that they use primer

kits that reveal continuous variation between sample populations, making it difficult to distinguish between homozygous and heterozygous alleles in samples (Loxdale and Lushai, 1998).

Amplified fragment length polymorphisms (AFLPs) was initially developed for the fingerprinting of plant genomes but more recently have been used in population genetic studies in a wide variety of insect taxa (Wong et al., 2001). For example, AFLP have been used to analyze the genetic structure of virus-vector populations at a regional level (Dopman et al., 2004). The advantage of AFLP is that it generates large numbers of markers spaning the whole genome even without prior knowledge of primers. Moreover, it offers improved reproducibility compared to randomly amplified polymorphic DNA (RAPD) markers (Zhong et al., 2004). However, its disadvantage is that it generates dominant rather than codominant markers. The later markers are preferable to dominant markers because they allow clear distinction of homozygous and heterozygous genotypes on an electrophoretic gel (Piepho and Koch, 2000).

The use of microsatellites as DNA markers has proved valuable in insect population genetic studies (Loxdale and Lushai, 1998). The advantage of this approach is its ability to detect greater levels of genetic variability. However, the disadvantages include the requirement to screen several loci for adequate population information (a minimum of four polymorphic loci in clonal organisms and ten and above for sexual populations), this has implications such as increased development time and costs. The fifth DNA marker is the use of restriction fragment length polymorphisms (RFLPs) with polymerase chain reaction. Specific references of RFLP + PCR as applied to mitochondrial DNA (mtDNA) include the taxonomic and population genetic studies of biotypes of leafhoppers (Loxdale and Lushai, 1998). Application of RFLP detected strains of Wolbachia coexisting in species of mulberry leafhoppers (Mitsuhashi et al., 2002). The main advantage of this marker is the versatility of its applications, where sequence analysis can differentiate to the level of strain-forms. One disadvantage of RFLP + PCR is the fact that the level of variability between samples may depend on the degree of genetic isolation.

Molecular characterisation of the vector

A survey of the literature indicates minimal work carried out on the molecular characterisation of *Cicadulina spp* populations. Lett et al. (2002) applied polymerase chain reaction (PCR) to understand the mechanism of the gut wall as a barrier to MSV transmission. The authors demonstrated (i) the differential virus persistence and accumulation between vector and non-vector leafhopper species, and (ii) detected and quantified the virus in the insect vectors' body compartments involved in virus circulation and persistence. There is potential in utilizing the available molecular techniques to distinguish the various *Cicadulina* populations in determining vector-virus interaction, as part of understanding MSV transmission.

Molecular characterisation of the virus

Peterschmitt et al. (1991) characterised MSV isolates from 11 African countries and regarded them all as being of the same serotype. The use of RFLP and polymerase chain reaction has been the most applied molecular technique in the characterisation of MSV with good results. Martin et al. (2001) described the use of PCR and RFLP in the typing of 49 MSV isolates. They sequenced and analysed 12 new MSV genomes selected from among the 49 as being representative of major virus groupings. This technique enabled the authors to classify 85 MSV isolates into strain and subtype groupings. Willment et al. (2001) applied RFLP analysis using a set of seven enzymes to accurately type closely related virus isolates. In an earlier study on determining the nature of three MSV isolates, RFLP analysis and sequencing confirmed the occurrence of the mutant spectrum (quasi-species) nature of the three MSV isolates (Isnard et al., 1998). The RFLP approach is useful for investigating virus diversity in field infections, such as the investigation of viral genomes within vector species (Lett et al., 2002). The technique is not too useful for isolates differing in sequence by more than 20% (Hughes et al., 1992); however, as all maize isolates of MSV found to date differ by less than this, it remains useful for the differentiation of closely related viruses.

OPTIONS FOR MSV DISEASE MANAGEMENT

Breeding for MSV resistance

Most recent host plant resistance projects have emphasized the need for an interdisciplinary approach to cultivar development. Literature has few studies on breeding for maize varieties resistant to the vectors of MSV. Kairo et al. (1995) studied the settling, probing and oviposition behaviour of *C. mbila* using four maize genotypes, 100 MSR, HASR, Reunion and H512. They concluded that only H512 was completely susceptible to MSV and, in general, *C. mbila* settled in higher numbers on this genotype. The results suggested the existence of potentially useful resistance mechanisms against the vector. Rensburg (2001) compared streak resistant maize inbred lines derived from various resistance breeding programmes for leafhopper feeding preferences. Pronounced antixenosis to *C. mbila* was observed in inbred lines E739 and CML206, with moderate levels of antixenosis in P606, P590, P612 and CML202, while J2705tv and VH188w showed negligible antixenosis. Since disease severity depends to an extent on the acquired virus dose, there is potential in exploiting the observed antixenosis in national breeding programmes,

to reduce the level of primary infection in plants that have an antiviral resistance component (Rensburg, 2001).

The goal of several programmes in Africa has been the development of maize germplasm resistant to MSV (Barrow, 1993). Resistance in maize germplasm was noted earlier in 1931 in South Africa and later in East Africa, Nigeria (at the International Institute of Tropical Agriculture – IITA) and in Réunion (Soto et al., 1982; Barrow, 1993; Asanzi et al., 1995a; Bosque-Pe´rez et al., 1998, Pernet et al., 1999). Maize varieties with resistance traits to MSV were developed at IITA and at the Harare station of the international Maize and Wheat Improvement centre (CIMMYT) (Efron et al., 1989). In fact, many breeding programmes in Africa use MSV resistant germplasm sources developed at IITA for incorporation into their varieties. MSV resistance in many of the IITA open-pollinated varieties and hybrids manifests itself as reduced symptom severity combined with low virus incidence in the field (Bosque-Pérez et al., 1998). It has been noted that resistance to MSV by the IITA maize germplasm is controlled by two or three major gene pairs, with the possible involvement of minor genes (Kim et al., 1989). Welz et al. (1998) mapped out the quantitative trait loci (QTL) for resistance to MSV; but Kyetere et al. (1999) went further and demonstrated the presence of a single major gene (designated as *msv* 1) that controls MSV tolerance.

In spite of the efforts of breeding of varieties against MSV disease, sporadic outbreaks of MSV have continued to occur in much of Africa, with significant yield losses. In fact it is reported that some maize varieties known to be resistant to MSV in one ecological zone, would show susceptibility to the disease in another, as reported in the island of Réunion (Bosque-Pérez, 2000). There is therefore urgent need to understand the local distribution of MSV strains and how more virulent MSV isolates can easily be disseminated widely across agro-ecological zones and seasons. This information would be invaluable to the numerous on-going maize breeding programmes across Africa.

Chemical control

Investigations on the chemical control of *C. mbila* showed that carbofuran granules applied to the planting furrow at 0.2 g a.i./m was significant in suppressing leafhopper populations (Drinkwater et al., 1979). Carbofuran applied as a seed dressing at 0.80 and 1.04 g a.i. /kg seed suppressed the *Cicadulina* populations (Rensburg and Giliomee, 1989). Moreover, carbofuran pesticide was also reportedly better than conventional spray treatments with endosulfan 31 and 45 days after emergence of maize plants or aldicarb in granules at 0.3 g/m in controlling MSV vectors (Drinkwater et al., 1979). Mzira (1984) reported that application of carbofuran effectively reduced the rate of MSV disease by ten times in the treated plots. This could be due to the systemic carbofuran conferring protection to the most vulnerable young maize. Dahal (1997) in his review, reported that leafhopper populations appeared to be controlled by the carbamate class of insecticides. These studies nevertheless did not mention the incidence of virus diseases and, thus failed to clarify whether the reduction in yield loss was due to reduced direct feeding damage or due to low virus incidence. Nevertheless, though these studies show that partial control of leafhoppers can be achieved by the use of persistent systemic insecticides, the protection of the crop is rendered inappropriate by the recurring influx of migrant hopper populations which re-infect the crop after each application. In addition, the Food and Agricultural Organisation (FAO) and the World Health Organisation (WHO) (WHO, 1986; FAO, 1998) documented various reports of ill health associated with those applying pesticides in the maize based systems in Africa. Pesticide poisoning affects a million people in Africa annually, with 20,000 cases resulting in death (WHO, 1986). Much of the pesticide problem is due to the use of pesticides by many small-scale farmers, who lack adequate knowledge on pesticides and who fail to wear appropriate protective clothing (Christiansson, 1991; Matthews et al., 2003). Similarly, most of the low resource endowed farmers are not able to afford the costs of pesticide applications (Matthews et al., 2003). Appropriate insecticide use will continue to play an important role in maize viral disease/vector control; nonetheless, non-chemical alternatives remain the most cost effective, safer and more environmentally appropriate approaches for tropical, low resource endowed farmers (Pingali, 2001; Matthews et al., 2003).

Biological control

The potential of utilising natural enemies (predators and parasitoids) and entomopathogenic microbes for the control of leafhoppers has been demonstrated in Asian countries (Chandra, 1978; Gupta and Pawar, 1989; Heong et al., 1992; Mitsuhashi et al., 2002). A number of parasitoids, predators and entomopathogens of important cicadellid pests including *Cicadulina spp* that occurs in India have been documented (Gupta and Pawar, 1989; Singh et al., 1993). However, no exhaustive attempts have been made to identify and utilise biological control agents of leafhoppers in Africa. Olmi (1995) reported the rearing of a species of dryinid parasitoid (Anteon traorei) from eggs and larvae of *C. mbila* in Burkina Faso. There is need to explore and identify indigenous natural enemies of *Cicadulina spp.* present in Africa. Likewise, as the understanding of the adaptive causes of plant defenses against insects expands, it will be possible to include the use of transgenes and genetic engineering in targeted maize breeding programmes. This technology will undoubtedly raise new risks and concerns. However, it is vital to measure the risks versus the benefits of GM crops and a comparison made with other alternative pest

management strategies (Dutton et al., 2004; Poppy and Sutherland, 2004).

Cultural control

Several workers (Fajemisin et al., 1984; Bosque-Pérez et al., 1998) have recommended the exploitation of aspects of crop agronomy as a method of reducing crop damage due to MSV disease. Various cultural practices suggested for the control included the use of 'barriers' of bare ground between early and late planted maize fields to reduce leafhopper movement and subsequent MSV spread (Bosque-Pérez, 2000), avoidance of maize plantings downwind from older cereal crops, and the use of crop rotations that will minimize invasion by viruliferous leafhoppers (Rose, 1978; Barrow, 1992). Maize plants infected less than a week after germination produced no yield, at three weeks produced 5% yield and at 8 weeks produced almost full yield. Early planting before the build up of leafhopper populations could be an option of MSV management. Rose (1978) recommended the practice of disease avoidance by adjusting planting dates to avoid migrating leafhoppers landing on young plants. However, given the unreliable and erratic rainfall, it is not feasible to recommend planting of maize, before the onset of rains, or even late planting. In this regard, therefore, there is need to broaden the scope of cultural control options available to the African maize farmer. The use of innovative soil fertility management practices, in the context of sustainable agriculture, promises to be a potentially useful method of widening the scope of MSV disease management available to the tropical, small-scale maize farmer. This review similarly explores the potential of manipulating the interactions of *Cicadulina spp*, MSV disease and agro-environmental factors in relation to MSV disease management.

SOIL NUTRIENT LEVELS AND MAIZE PRODUCTION IN AFRICA

The breakdown of traditional farming practices in Africa, coupled with the commercialization of many farming systems have eliminated the fallow periods, resulting in soil fertility decline (Byerlee and Heisey, 1996; Sanchez, 2002; Omamo et al., 2002). Over decades, small-scale farmers have removed large quantities of nutrients from their soils without replenishment using sufficient quantities of organic or inorganic fertilizer. This has resulted in high average annual depletion rate of 22 kg of nitrogen (N), 2.5 kg of phosphorus (P), and 15 kg of potassium (K) per ha of cultivated land, in 37 African countries (Sanchez, 2002).

The formulation of starter fertilizers for maize farming is calculated to improve plant nutrient uptake and enhancement of early crop growth (Bermudez and Mallarino, 2004). But owing to economic considerations of N use, focus has shifted to the development of production management practices that utilize N fertilizer more effectively. Binder et al. (2000) reported that a maize plant begins to take up N during the middle vegetative growth period, attaining a maximum rate of uptake near silking stage.

In sub-Saharan Africa, P is a limiting nutrient in many soils of the semi-arid tropics and in acid, weathered soils of the subhumid and humid tropics (Buresh et al., 1997; Daroub et al., 2003). Kwabiah et al. (2003) reported P as a major limiting factor in maize production, and that unlike N, P cannot be added to the soil by biological fixation. Phosphorus is a critical nutrient for plant growth, since it is involved in cellular energy transfer, respiration, photosynthesis, early root development and it is a structural component of nucleic acids and of many coenzymes, phosphoproteins and phospholipids (Mollier and Pellerin, 1999; Raghothama, 1998). Potassium on the other hand is involved in opening and closing of stomata in leaves.

Effects of NPK fertilizers on insect pests and viral diseases

The levels of soil nutrients (Orians and Jones, 2001) reportedly influence the variability of spatial and temporal distribution and abundance of leafhoppers and viral pathogens. Plants with sufficient nutrients are stronger, healthier, and better able to compensate for pest damage than those under nutritional deficiencies (Morales et al., 2001; Mesbah et al., 2002). Chaboussou (2004) showed that inhibition of plant viral agents is strongly correlated to plant's deficiency in nutrients necessary for viral growth and reproduction. Moreover, this trend appears to hold particularly true for both nitrogen and phosphorus elements (Byerlee and Heisey, 1996).

Chaboussou (2004) reported that in contrast to nitrate fertilizers, alkaline phosphate fertilizers have a beneficial effect against viral diseases, such that, by promoting maturity, they speed up the stage of resistance in the plant brought about by age. Thus, while P simultaneously stimulates plant growth and virus concentration, K on the other hand increases plant growth, and reduces viral concentration (Chaboussou, 2004). The possible determination of positive interactions between soils and pests would potentially provide necessary guidelines in pest management for maize based agro-ecosystems.

The complex influence of time of fertilizer application on the population dynamics of the planthoppers *Sogatella furcifera* (Horvath) and *Nilaparvata lugens* (Stal) on rice was studied in China (Zhu et al., 2004). The authors observed that spraying organic phosphorous (triazophos) in mid-season (during the 1st generation of the planthoppers) induced a population resurgence of the 2nd generation of *S. furcifera* and *N. lugens*. The main mechanism for the resurgence was the stimulation of re-

production of the 1st-generation adults. Plots that were fertilized early attracted more *N. lugens* immigrants, and enhanced reproduction of *S. furcifera* immigrants. In addition, greenhouse studies (Dhaka, 2002; Prasad et al., 2003) conducted to investigate the influence of N levels (0, 60, 120 and 200 kg/ha) on the population build-up of the brown planthopper (*N. lugens*, BPH) on rice cultivars demonstrated that BPH population build-up was low up to 60 kg N/ha irrespective of the levels of BPH resistance of the rice cultivars.

Nitrogen constituent in plant tissues is the phytophagous insects' principal attracting component (Haltrich et al., 2000; Smith et al., 2002). Haltrich et al. (2000) reported a positive correlation between migrations and reproduction of leafhoppers with the levels of soluble nitrogen content of host plants. Lu et al. (2004) demonstrated that increasing plant N significantly decreased the relative water content in rice plants due to damage by the brown planthopper (*N. lugens* (Stal). This may be considered to be one of the key factors to increased susceptibility of rice plants supplied with high N to BPH damage. Related studies in Nicaragua, on population levels of the maize leafhopper *Dalbulus maidis*, which transmits the maize stunt spiroplasma (mollicute *Spiroplasma kunkelii* (Spiroplasmataceae: Mycoplasmatales)) to maize, demonstrated that manipulating host plant quality by increasing N fertilization resulted in higher leafhopper densities at high N levels (Power, 1987). In addition, exploring the role of vector movement in disease spread, leafhopper movement rates and emigration, the authors established that plant quality and density significantly affected the spread of maize stunt disease through their effects on the abundance and movement behaviour of the leafhopper. Present literature is however deficient in such studies involving MSV leafhopper vectors. In view of the fact that leafhoppers respond to the mineral content of their hosts, it is probable that different rates of fertilizer formulations can possibly mediate the migration patterns *Cicadulina* leafhoppers.

Although the use of organic materials as a source of nutrients in combination with inorganic fertilizers is recommended (Palm et al., 1997), information on nutrient content and quality of organic inputs shows a lot of variation between sites (Murwira et al., 2002). Nevertheless, under low-input conditions the use of organic nutrients supplemented with inorganic nutrient sources is a potential option in maize production (Jama et al., 2000). Similarly, organic inputs enhance nutrient cycling and the transformation of inorganic forms of elements such as N and P into more available organic ones (Mugwira et al., 2002). There is need to determine the effects of time, form and rates of nitrogen application on leafhopper and MSV management

CONCLUSIONS

Maize streak virus disease significantly contributes to maize yield reduction in Sub-Saharan Africa. The disease is ecologically versatile occurring sporadically following climatic instabilities. The causative virus of MSV disease has been identified, described and grouped using full length MSV nucleotide sequences, and the vector species distribution mapped out at continental level. The molecular characterisation of the vector to distinguish the geographically separate populations of *C. mbila* (the most important species) has not been carried out. There is potential of using molecular markers to determine the population dynamics and Cicadulina leafhoppers and MSV strain geographical distribution at country level, as a prelude to understanding the influence of environmental factors on MSV disease epidemiology. The methods used in MSV management such as breeding for MSV resistance, chemical control, biological control, and cultural control have given conflicting results. Soil nutrient management in the context of sustainable agriculture, as given in literature provides a potential method of widening the scope of MSV disease management.

The authors suggest further research on assessing the composition of region specific MSV viral strains, determination of the composition of MSV strains present in geographically separate populations of *C. mbila* and determination of the effects of soil fertilisation (nitrogen, phosphorus and potassium) on vector behaviour MSV disease virulence transmission and expression.

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