Maize Streak Virus (*Geminiviridae*)☆

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Abbreviations

C-ori complementary strand (i.e., the half of the DNA duplex that is not packaged into virus particles) origin of replication.
CP coat protein. The only protein component of the virus particle also believed to be involved in nuclear trafficking and cell-to-cell movement of viral DNA.
LIR long or large intergenic region containing the origin of virion strand replication and gene promoters.
MP movement protein. A small (c. 10 kDa) protein believed to be involved in intercellular virus movement via plasmodesmata.
Rep replication-associated protein involved in the initiation of virion strand replication.
RepA a truncated version of Rep with a unique C-terminal domain believed to be involved in regulation of host and/or virus gene expression.
SIR short or small intergenic region containing gene polyadenylation signals and the origin of complementary strand replication.
V-ori virion strand (i.e., the half of the DNA duplex that is packaged into virus particles) origin of replication.

Glossary

Agro-infection Technique used for the infection of host plants with cloned virus genomes involving transfer of virus DNA into the nuclei of host cells by the bacterium *Agrobacterium tumefaciens*.
Agro-inoculation See agro-infection.
Bicistronic Contains two protein-coding regions within a single mRNA transcript.
Capsomer A subunit of the mature virus particle containing an ordered series of polymerized coat protein molecules.
***Cicadulina* spp.** A group of leafhopper species involved in transmission of MSV.
Oviposition Egg-laying. To oviposit means to lay eggs.
Plastochron The time interval between successive leaf primordia, or the attainment of a certain stage of leaf development.
Viruliferous A state in which an MSV vector species is carrying and is capable of transmitting the virus.

Introduction

Maize streak virus (MSV) is the causal agent of maize streak disease (MSD), one of the most serious viral diseases of maize in Africa. It is a major contributor to the continent's food security problems and is endemic throughout Africa south of the Sahara. It is also found on the Indian Ocean islands of Madagascar, Mauritius, and La Réunion. There is no obvious barrier to spread of the virus outside of this region; hence it should be considered as a serious potential problem for other as yet unaffected maize-growing areas.

History and Taxonomy

"The disorder of the mealie plant, locally described as "Mealie Blight", "Mealie Yellows", or "Striped Leaf Disease", belongs to a group of plant troubles arising from obscure causes …" was how MSD was first described by Claude Fuller in 1901 in Natal, South Africa. Fuller mistakenly attributed the disease to a soil disorder, but in retrospect it is quite clear that the "mealie (a local word for maize or corn) variegation" he described and drew in minute detail can be attributed to MSV.

The first milestone in MSD research was reached in 1924, when Storey determined that a virus transmitted by leafhopper species of the genus *Cicadulina* (Fig. 1) was the causal agent of MSD. Storey named the virus "maize streak virus", and was also the first to determine both the genetic basis of MSV transmission by *Cicadulina mbila*, and the heritability of MSD resistance in maize.

When MSV particles were first purified in 1974 they were found to have a novel twinned quasi-icosahedral (geminate) shape (Fig. 2), from which the name "geminivirus" was derived. This was followed by the unexpected discovery in 1977 that geminivirus particles contain circular single-stranded DNA (ssDNA); a genome type never before observed in plant viruses. These novel characteristics led to the proposal of a new virus group – the geminiviruses – comprising MSV and other viruses with geminate particles and ssDNA genomes. *Maize streak virus* is now recognized as the type species of the genus *Mastrevirus*, in the family *Geminiviridae*.

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Fig. 1 The leafhopper vector of MSV, *Cicadulina mbila* Naudé. Photograph courtesy of Dr. Benjamin Odhiambo, Kenyan Agricultural Research Institute (KARI).

Fig. 2 MSD symptoms on a maize leaf: note characteristic veinal streaks. Photograph courtesy of Dr. Frederik Kloppers, PANNAR (Pty) Ltd., Greytown, KwaZulu-Natal, South Africa.

Host Range and Symptoms

While most notorious for the yield losses it causes when infecting maize, MSV also infects over 80 other grass species including the economically important crops wheat, barley, and rye. In susceptible maize and grass genotypes, the virus first causes symptoms between 3 and 7 days after inoculation. These first appear as almost circular pale spots of 0.5–2 mm diameter in the lowest exposed portions of the youngest leaves. Later, fully emerged symptomatic leaves show veinal streaks from a few millimeters long to the entire length of the leaf and between 0.5 and 3 mm wide. These streaks often fuse laterally and symptomatic leaves may become >95% chlorotic (**Fig. 2**).

Plants are worst affected when infected within a few days of coleoptile emergence; symptoms only develop above the site of inoculation on newly emerging leaves. Susceptible varieties may display severe stunting as well as very severe streaking. Afflicted plants will frequently produce deformed cobs or may fail to produce cobs altogether and per-field yield losses can reach 100% when susceptible maize genotypes are infected early.

Of the eleven major MSV strains so far identified (designated MSV-A through MSV-K; (**Fig. 3**)), only MSV-A produces economically significant infections in maize. The "grass-adapted" MSV strains (MSV-B, -C, -D, -E, -F, -G, -H, -I, -J and -K) differ from MSV-A types by 5%–25% in nucleotide sequence, and produce substantially milder symptoms in maize than do MSV-A viruses. In many cases these grass-adapted strains are incapable of producing symptomatic infections in MSV-resistant maize genotypes.

Diversity and Evolution

MSV is closely related to the other distinct 'African streak' mastreviruses, Panicum streak virus, Sugarcane streak virus, Sugarcane streak Egypt virus, and Sugarcane streak Reunion virus, with which it shares ~65% genome sequence identity. It is, however, most similar to, although not necessarily more closely related to, an isolate of *Digitaria streak virus* from the Pacific island of Vanuatu, with which it shares ~67% genome sequence identity (**Fig. 3**).

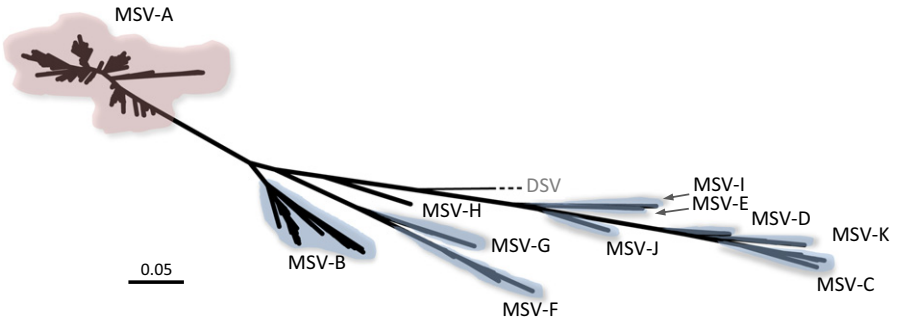

Fig. 3 Phylogenetic relationships between the full genome sequences of different MSV strains. The tree is constructed using the maximum-likelihood method (HKY model transition and transversion weight determined from the data and 100 bootstrap replicates) and numbers associated with branches indicate degrees of bootstrap support for those branches. Branches with less than 70% support have been collapsed and the genome sequence of a Digitaria streak virus (DSV) from Vanuatu is included as an outgroup. Only viruses in the MSV-A group have been isolated from maize. All viruses in the other groups have been isolated from wheat, barley, or wild grass species.

The full genome nucleotide sequences of MSV-A isolates display relatively low degrees of diversity, with any two MSV-A isolates obtained from anywhere in Africa invariably having genome sequences that are more than 97% identical. MSV isolates from La Réunion share ~95% identity with mainland isolates.

Although Maize was first introduced at multiple points into Africa and its neighboring islands in the 16th century, it is apparent that the most recent common ancestor of all presently sampled MSV-A genomes only came into existence in the mid 1800s somewhere in South-Eastern Africa. It is also likely that this ancestral virus was the recombinant offspring of grass adapted-adapted parental viruses: one parent an MSV-B variant, and the other parent belonging to a presently unsampled MSV strain that is most closely related to MSV-F and MSV-G.

Transmission

In nature, MSV and other African streak viruses are neither seed nor contact transmissible and rely instead on transmission by cicadellid leafhoppers in the genus *Cicadulina* (including, among others, *C. mbila*, *C. storeyi*, *C. bipunctella zeae*, *C. latens*, and *C. parazeae*). Of these, *C. mbila* is considered the most important MSV vector as it is the most widely distributed. Also, a greater proportion of *C. mbila* individuals are capable of transmitting the virus than is found in other *Cicadulina* populations. The virus may be acquired by leafhoppers at any developmental stage in less than 1 h of feeding with a minimum acquisition time of 15 s. A latent period within the vector, during which the virus cannot be transmitted, lasts between 12 and 30 h at 30°C. Once this latent period is over (signaled by the appearance of virus within the leafhopper's body fluids) the virus can be transmitted within 5 min of feeding. Once MSV has been acquired by a leafhopper, the insect will transmit the virus for the rest of its life.

Particle Structure

MSV particles (Fig. 4) consist of two incomplete icosohedra with a $T = 1$ surface lattice, comprising 22 pentameric capsomers each containing five coat protein (CP) molecules. Particle dimensions are 38 nm × 22 nm with 110 CP molecules in each virion packaging a ~2690 nt covalently closed mostly single-stranded circular DNA genome. The packaged DNA has annealed to it a complementary ~80 nt sequence believed to act as a primer for complementary strand synthesis following infection and uncoating.

Genome Organization

As with all other mastreviruses discovered to date, the MSV genome encodes four proteins: a movement protein (MP), a coat protein (CP), a replication-associated protein (Rep), and a regulatory protein (RepA). Whereas CP and MP are expressed off alternatively spliced virion sense transcripts, Rep and RepA are expressed of alternatively spliced complementary sense transcripts. MSV genomes also contain two intergenic regions: these are a short or small one (SIR), and a long or large one (LIR), which are the complementary sense strand and virion sense strand origins of replication, respectively (Fig. 5).

The Long Intergenic Region

Besides containing divergent RNA polymerase II-type promoters and other transcriptional regulatory features necessary for the expression of the complementary and virion sense genes, the LIR also contains sequence elements that are essential for replication. The

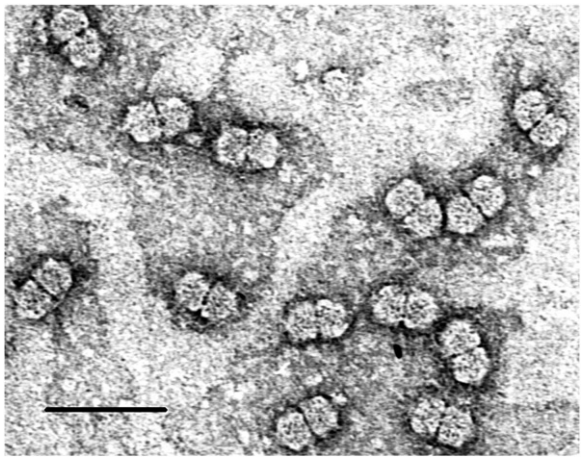

Fig. 4 Electron micrograph of MSV purified from infected maize, showing particles of 18 nm × 30 nm stained with uranyl acetate. Scale = 50 nm. Photograph courtesy of Kassie Kasdorf; copyright EP Rybicki.

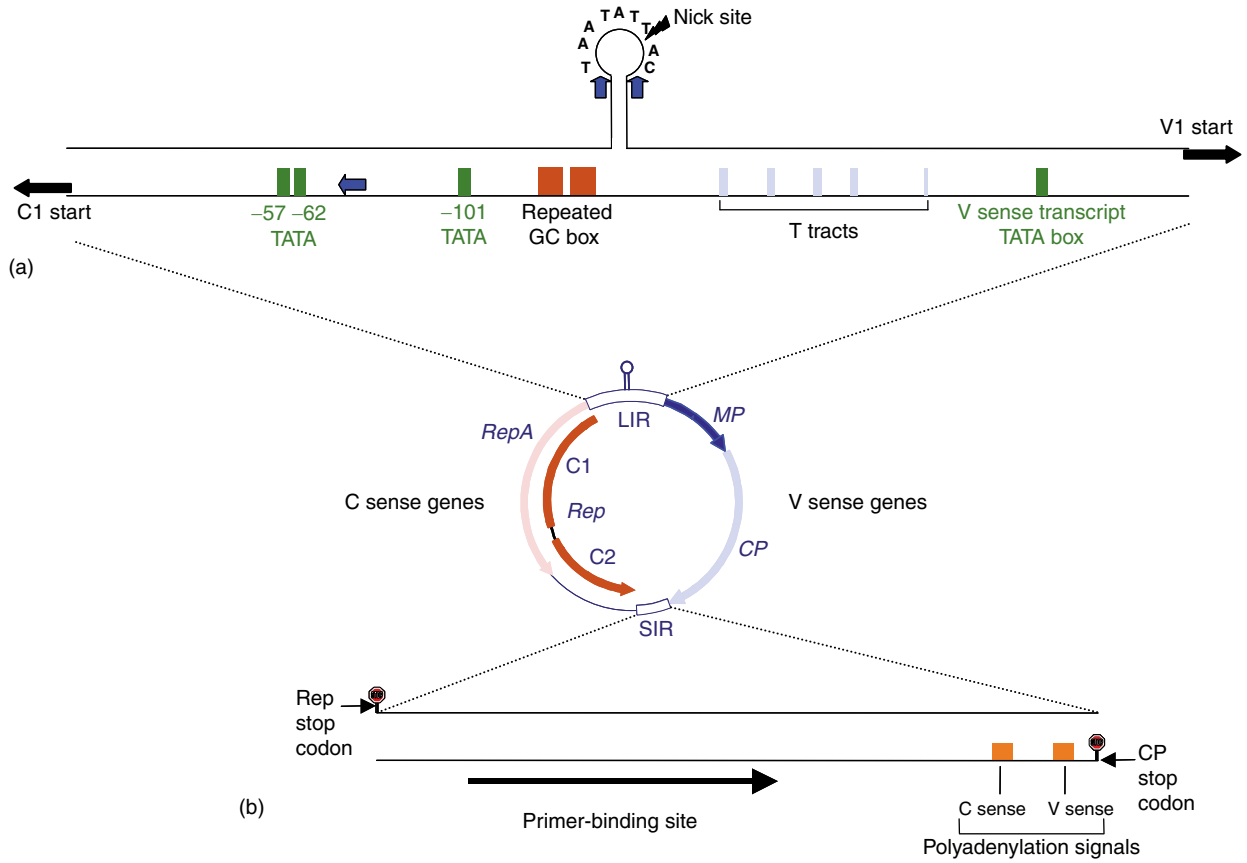

Fig. 5 A schematic representation of the MSV LIR (a) and SIR (b), shown in context with the MSV genome. In (a) the main features of the MSV LIR are shown. These include a stem-loop structure with the loop's nonanucleotide sequence conserved among all geminiviruses and other rolling-circle systems. The site at which Rep introduces an endonucleolytic nick to initiate virion strand replication is shown. Iterated sequences (iterons) are shown in the V sense, with blue arrows indicating their location in the LIR. Iterons are potentially specific Rep-recognition sequences via which Rep may bind to the LIR. 5′ of the stem–loop is a repeated GC-box, which binds host transcription factors. A series of T tracts 3′ of the stem–loop may be involved in DNA bending of this region of the LIR. TATA boxes 5′ and 3′ of the stem–loop are potential C sense and V sense transcription initiation sites, respectively. In (b), the main features of the SIR include polyadenylation signals for V and C sense transcripts, and a primer-binding site on the plus strand. An ~80 bp DNA primer-like molecule, encapsidated with the viral genome and annealed to this site, is thought to be involved in initiating complementary strand replication. Both the MSV LIR and SIR are essential for viral replication.

most striking of these is an inverted repeat sequence that is capable of forming a stable hairpin loop structure. All geminiviruses sequenced to date have the highly conserved nonanucleotide sequence TAATATT↓AC within the loop sequence of similar hairpin structures: this sequence contains the virion sense strand origin of replication (V-*ori*;↓).

A sequence 6–12 nt long occurring in all known mastreviruses between the TATA box that directs rep/repA transcription and the repA initiation codon is directly repeated in the stem near the V-*ori* hairpin, and is probably involved in Rep and/or RepA binding during replication.

The hairpin and two GC boxes on the 5′ side of the stem also forms part of an upstream activator sequence (UAS) required for efficient CP expression. The GC boxes bind nuclear factors to the UAS and are known as the rightward promoter element (rpe1).

The Short Intergenic Region

The MSV SIR occurs between the termination codons of the *CP* and *rep* genes (Fig. 5) and contains the polyadenylation and termination signals of the virion and complementary sense transcripts. The SIR also contains the origin of complementary strand synthesis (C-*ori*). A small 80-nt-long primer-like molecule is bound to the SIR of encapsidated MSV DNA and, at the onset of an infection, probably enables synthesis of double-stranded DNA (dsDNA) replicative forms (RFs) of the genome from newly uncoated virion strand DNA.

The Complementary Sense Genes (*Rep* and *RepA*)

Rep is the only MSV gene product that is absolutely required for virus replication. In mastreviruses Rep is encoded within two open reading frames (ORFs) referred to as C1 and C2 (Fig. 5). Beginning at the same transcription initiation site, two C sense transcripts (1.5 and 1.2 kbp in size) are produced during MSV infections. Splicing of the larger transcript removes an intron, which permits expression of full-length Rep from the two ORFs.

It is very probable, although as yet unproven *in vivo*, that RepA is translated from both the unspliced 1.5 kbp transcript and the 1.2 kbp transcript. If expressed in infected cells, MSV RepA would have the same N-terminal 214-amino-acid sequence as Rep, but would have a different C-terminus. RepA is likely a multifunctional protein that modifies the nuclear environment to favor viral replication.

The N-terminal portions of Rep and RepA contain three conserved amino acid sequence motifs commonly found in replication-associated proteins of many extremely diverse rolling-circle replicons. Other significant landmarks include a plant retinoblastoma-related protein (pRBR) interaction motif (via which RepA binds to host pRBR molecules to manipulate the cell cycle), and oligomerization domains (via which Rep and RepA bind to other Rep/RepA molecules to form homo- and heterooligomers). It is also likely that the approximately 100 N-terminal amino acids of both Rep and RepA are involved in binding these proteins to the viral LIR during replication.

The C-terminal portion of Rep contains a dNTP-binding domain with motifs similar to those found in proteins with kinase and helicase activities. The dNTP-binding domain also sits within a region with similarity to the DNA-binding domains of the myb-related class of plant transcription factors: this domain may be functional in the induction of virus and/or host gene transcription.

The C-terminal portion of RepA, which is different from that of Rep, contains another potential transactivation domain also possibly involved in the regulation of virus and/or host gene expression. A second domain within the C-terminal portion of RepA possibly interacts with host proteins involved in developmental regulation.

The Virion Sense Genes (MP and CP)

Transcription of the MSV virion (V) sense genes is directed by two TATA boxes within the LIR 26 and 214 nucleotides 5′ of the MP start codon. Each TATA box directs the production of different-sized transcripts, both of which terminate at the same place. Splicing of an intron within the MP portion of V sense transcripts appears to be an important determinant of relative MP and CP expression levels. Whereas CP is expressed from both long and short, spliced and unspliced V sense transcripts, MP is most likely only expressed from unspliced long transcripts.

The MP is post-translationally modified and contains a hydrophobic domain that may either facilitate its interaction with host cell membranes or be involved in homo- or hetero-oligomerization with the CP.

The N-terminal ~100 amino acids of the CP contain both nuclear localization signals and a sequence-nonspecific dsDNA- and ssDNA-binding domain. The CP and MP interact with one another and it is possible that this interaction is involved in trafficking of naked and/or packaged virus DNA from nuclei through nuclear pores, to the cell periphery, through plasmodesmata, and into the nuclei of neighboring cells.

Molecular Biology of MSV

Replication

As with other geminiviruses, MSV replicates by both a rolling-circle mechanism (rolling-circle replication, or RCR; (Fig. 6)) and a recombination-dependent mechanism (recombination dependent replication or RDR). As with other rolling-circle replicons, MSV

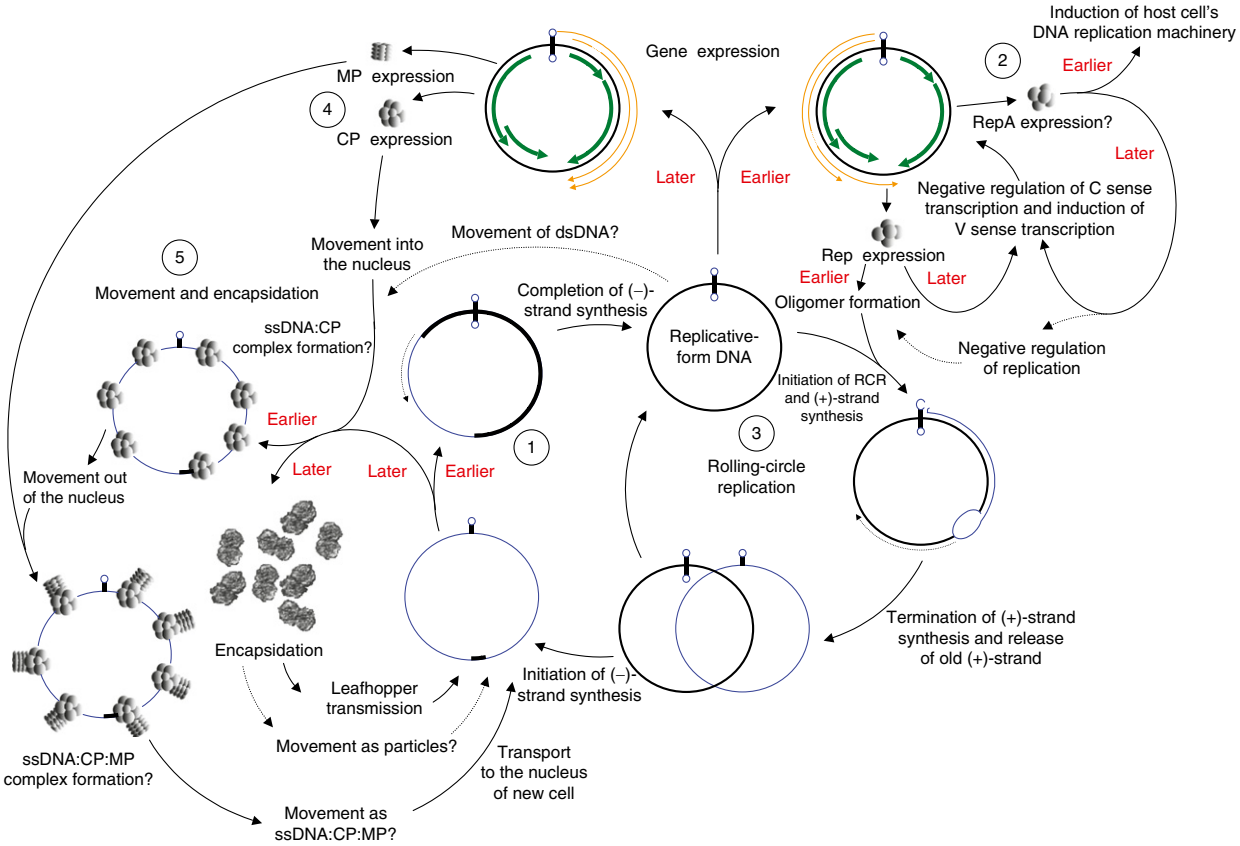

Fig. 6 Summary of the MSV infection process. Early during an infection following the synthesis of a dsDNA replicative form (RF; 1) RepA is most likely expressed and induces a cellular state in which viral DNA replication can occur (2). Rep is also expressed early and RCR begins (3). At a later point in the infection process, following genome amplification and possibly Rep and/or RepA induction of the V sense promoter, MP and CP are expressed (4), and movement and encapsidation occur (5). Represented here is movement of un-encapsidated ssDNA, but it should be noted that it is possible that dsDNA and/or encapsidated ssDNA may also be moved either cell to cell or systemically within the phloem of plants. Whereas the involvement of MSV CP and MP in movement has been demonstrated, the mechanics of the process are obscure. While the probable timing of events is indicated, it is unlikely, for example, that absolutely no MP and CP expression occurs during the earlier stages of the infection process. ssDNA is represented by blue lines, dsDNA by bold black lines, and RNA by orange lines.

replication is discontinuous with virion strand replication being initiated from the hairpin structure in the LIR and complementary strand synthesis being initiated from a short 80 nt primer-like molecule synthesized on the SIR of newly replicated virion strands.

Particle Assembly and Movement

Besides being the primary location of replication, the nucleus is also the site of virus particle assembly. CP molecules in the nucleus nonspecifically bind virion strands released during RCR (there is no known encapsidation signal in mastrevirus genomes), arresting the synthesis of new RF DNAs. Viral ssDNA molecules are packaged into particles that aggregate to form large para-crystalline nuclear inclusions. Crystalline arrays of MSV particles have also been detected outside nuclei within physiologically active phloem companion cells, and inside the vacuoles of dead and dying cells within chlorotic lesions. These lesions are caused by an as yet unexplained degeneration of chloroplasts in infected cells.

The mechanistic details of MSV cell-to-cell movement are still obscure, but it seems to involve an interaction between the CP, MP, and viral DNA. Besides requiring the coordinated interactions of viral gene products and DNA, the successful movement of MSV genomes from infected to uninfected cells is strongly dependent on the extent of plasmodesmatal connections between neighboring cells. Also, in maize it appears as though certain cell types are more sensitive to MSV infection than others. For example, in maize leaves the virus infects all photosynthetic cell types (e.g., mesophyll and bundle sheath cells) but despite abundant plasmodesmatal connections between photosynthetic, epidermal, and parenchyma cells, MSV is only rarely detectable in the latter two cell types.

It is unknown whether systemic movement of geminiviruses within plants simply relies on normal cell-to-cell movement to deliver genomic DNA into the phloem, or whether viral DNA is specifically packaged for long-distance transport. It is possible that

cell-to-cell movement might involve un-encapsidated ss- or dsDNA but that long-distance movement in the phloem might require encapsidation.

Long-distance movement of MSV within infected plants occurs via phloem elements and it is believed that MSV is incapable of invading the root apical, shoot apical, and reproductive meristems due to the absence of developed vasculatures in these tissues. Thus, the virus is not found in tissues that develop into gametes and is therefore not seed-borne. It also does not appear to travel within plants from sites of infection into older uninfected tissues.

Within the shoot apex where most productive MSV replication occurs, MSV first enters developing leaves at approximately plastochron five. While the virus is restricted to the developing leaf vasculature before plastochron 12, it is likely that the development of metaphloem elements at approximately plastochron 12 provides an opportunity for the virus to escape the vasculature into the photosynthetic cells of the leaf. Metaphloem develops with the abundant plasmodesmatal connections required for efficient loading of photoassimilates once the leaf emerges from the whorl. Before emergence, however, the developing photosynthetic tissues are still net importers of photoassimilates and the virus most likely moves into these cells through their plasmodesmatal connections with the metaphloem.

On the leaves, the pattern of chlorotic streak-like lesions that characterizes MSV infections is directly correlated with the pattern of virus accumulation within the leaves and the virus can only be acquired by leafhoppers from these lesions. The degree of chlorosis that occurs within lesions can differ between MSV isolates and is related to the severity of chloroplast malformation that occurs in infected photosynthetic cells.

Control of MSD

Although effective control of MSD in cultivated crops is possible with the use of carbamate insecticides, and it is possible to avoid leafhopper infestations by varying planting dates, the fact that small farmers cannot generally use these options means that the development and use of MSV-resistant crop genotypes is probably the best way to minimize the impact of MSD on African agriculture.

MSV resistance is associated with up to five separate alleles conferring a mixture of both dominant and recessive traits, none of which are sufficient by themselves to prevent MSV infections. Despite great successes achieved in the development of maize genotypes that tolerate MSV infection without significant yield loss, there has been only limited success in the field. For example, severe infections of so-called MSV-tolerant genotypes can occur when they are grown under environmental conditions different from those in which the plants were selected, meaning that distinct geographical growing areas may each require genotypes with MSV resistance that is tailored to those areas.

The problem facing breeders is that natural genetic resistance to MSD is not usually associated with desirable agronomic traits such as good yield. It can therefore be difficult to transfer resistance traits without also transferring undesirable characteristics. Moreover, the number of alleles involved means that successful breeding takes years for each release. Even in the absence of any predictive modeling of sporadic MSD outbreaks, most farmers would still prefer to gamble on the use of higher-yielding MSV-sensitive genotypes.

Efforts are also currently underway to introduce MSV resistance traits into commercial maize genotypes by genetic engineering. This could have the advantage of enabling the direct transfer of single-gene resistance, without linkage to undesirable characteristics, to many different breeding lines suited to different environmental conditions. However, up till now this strategy has been limited by negative public perception of genetically modified organisms, and the expensive and time-consuming risk assessment necessary to ensure a safe feed and food product.

MSD Epidemiology

There are loose correlations between MSD incidence and both environmental conditions and agricultural practices. Environmental influences on MSD epidemiology are mostly driven by a strong correlation between rainfall and leafhopper population densities. For example, drought conditions followed by irregular rains at the beginning of growing seasons tend to be associated with severe MSD outbreaks. Also, maize planted later in the growing season tends to get more severely infected than that planted at the beginning of the season, probably due to steady increases in leafhopper numbers and inoculum sources over the course of the season. As is the case with most insect-borne virus diseases, however, the incidence of MSD is erratic. Whereas MSD can devastate maize production in some years, in others it has only a negligible effect. The reason for this is that, apart from MSD epidemiology being strongly dependent on environmental variables, it is also the product of complex interactions between the various MSV leafhopper vector and host species, and an as yet unknown number of virus strains.

Leafhopper Vectors

Serious MSD outbreaks are absolutely governed by leafhopper acquisition and movement of severe MSV isolates from infected plants (wild grasses or crop plants) to sensitive, uninfected crop plants. The distance that MSV spreads from a source of inoculum

is determined by the movement behavior of leafhoppers. Distinct long- and short-distance flight morphs have been detected among certain *Cicadulina* populations. It is believed that the long-flight morphs are a migratory form and, as such, these may play an important part in the rapid long-distance spread of virulent MSV variants. Migratory movement is more common in certain *Cicadulina* species than in others and it is probably influenced by environmental conditions.

The dynamics of primary infection following leafhopper invasion of a susceptible maize crop are influenced by leafhopper population densities, the proportions of viruliferous individuals in populations, and virus titers within these individuals. Disease spread within individual maize fields is apparently linear when only a few viruliferous leafhoppers are involved in transmission, but becomes exponential once the number of insects exceeds one individual per three plants.

Plant Hosts

Although attempts to understand the dynamics of MSD epidemics have focused primarily on vector population dynamics and behavior, an important component of MSD epidemiology is the population density, turnover, and demographics of the over 80 grass species that are both MSV and vector hosts. Because *Cicadulina* species favor certain annual grass hosts for mating and oviposition, the species composition of grass populations that vary seasonally in any particular area will directly influence leafhopper population densities and feeding behaviors in that area.

The species composition and age distribution of grasses (including cultivated crops) in an area may also affect the amount of MSV inoculum available for transmission in that area. While MSV infects at least 80 of the 138 grass species that leafhoppers feed on, both the susceptibility of these grasses to MSV infection and the severity of symptoms that occur following their infection may be strongly influenced by a number of factors. While sensitivity to infection can vary substantially from species to species, it can also vary within a species with genotype and plant age at the time of inoculation: for example, plants from many species, including maize, generally become more resistant to MSV infection with age, thereby reducing the inoculum available for transmission to other plants.

The Virus

While efforts are underway to promote the widespread cultivation of MSV-resistant maize in Africa, surprisingly little is known about the MSV populations that will confront these new genotypes. Although to date eleven major MSV strain groupings have been discovered, it is unknown whether any other than the maize-adapted MSV-A strain play an important role in the epidemiology of MSD. MSV-B, -C, -D, and -E isolates only produce very mild symptoms in MSV-sensitive maize genotypes and are therefore unlikely to pose any significant direct threat to maize production. Mixed MSV-A and -B infections have, however, been detected in nature and there is also strong evidence of recombination occurring between these strains. It is therefore possible that MSV-B, and possibly other MSV strains, may indirectly influence MSD epidemiology through recombination with MSV-A-type viruses. Recombination has been linked with the emergence of a number of geminivirus diseases and it may also have contributed to the original emergence of MSV-A. It is plausible therefore that it could also eventually contribute to the evolution of MSV-A variants (or even variants of the other MSV strains) with elevated virulence in resistant maize genotypes.

Future Threat

MSV is rightly regarded as a significant potential threat to maize production outside of Africa: while the vectors do not occur outside of Africa, there is no obvious reason that they would not survive in other equatorial or temperate regions of the world such as the southern USA, South America and Eurasia. It is a distinct possibility that they could inadvertently spread, or even be deliberately taken, to these regions. If, following the establishment of a MSV vector species outside of Africa, MSV was then introduced, the virus would inevitably begin spreading within native grasses, cultivated maize and the other cereals that it is known to infect. As none of the maize varieties grown outside of Africa has the slightest resistance to MSV, the probability of severe economic consequences would be very high.

Further Reading

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