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Role of Potyviruses in Synergistic Interaction Leading to Maize Lethal Necrotic Disease on Maize

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A B S T R A C T

Synergistic interactions among pathogenic viruses are common in plants. Though not all, but a number of reported cases involving such interactions have a potyvirus partner during co-infection. The presence of the potyvirus group seems to favor its own multiplication on host and multiplication of a co-infecting partner. In this review, some characteristics favoring higher pathogenesis have been discussed using maize lethal necrosis (MLN)-causing viruses namely *Maize chlorotic mottle virus* (MCMV) and its synergistic potyvirus, *Sugarcane mosaic virus* (SCMV). A comprehensive discussion on the role of potyvirus in the synergism has been presented to show that in MLN and similar synergisms - the machinery for induction, transmission and colonization of the host is catalysed by the potyvirus and not the co-infecting viral partner which otherwise seems to be the most virulent in the synergism-based diseases such as MLN disease in maize. Furthermore, the effect of MLN to food security and areas for future research for Africa has been discussed in this review.

Introduction

Synergistic diseases in plants have been described since late 1920s (Blood, 1928; Rochow and Ross, 1955; Garces-Orejuela and Pound, 1956; Lee and Ross, 1972; Kuhn and Dawson, 1973; Clark *et al*., 1980; Calvert and Ghabriel, 1983; Poolpol and Inouye, 1986; Uyemoto *et al*., 1981). There are two types of synergist interaction: the *potyvirus-associated synergisms*, in which one of the virus is a member of the potyvirus group and *non-potyvirus synergisms*, in which neither virus is a member of the same group. There is evidence to suggest that in many reported cases, a potyvirus group of plant viruses has been involved as one of the synergistic pair.

For examples the interaction of *Maize chlorotic mottle virus* (MCMV) and a potyvirus such as *Sugarcane mosaic virus* (SCMV), *Maize dwarf mosaic virus* (MDMV) or *Wheat streak mosaic virus* (WSMV) (Hebert and Castillo, 1974; Uyemoto *et al*., 1981, Wangai *et al*., 2012); a potyvirus known as *Sweet potato feathery mottle virus* (SPFMV) with *Sweet potato chlorotic stunt virus* (SPCSV) (Kreuze, 2002); *Bean pod mottle virus* (BPMV) with a potyvirus known as *Soybean mosaic virus* (SMV) (Lee and Ross, 1972; Calvert and Ghabriel, 1983; Anjos *et al*., 1992) and the classic interaction of *Potato virus X* (PVX) and a potyvirus: *Potato virus Y* (PVY) (Goodman and Ross, 1974a; Goodman and Ross, 1974b; Vance, 1991).

This review describes weaponry machinery behind potyvirus synergism and how it influences its co-infecting partner in maize lethal necrosis (MLN) disease on maize and the effect of the disease in Africa. It is well known that MLN is caused by a synergistic co-infection with MCMV and a potyvirus such as MDMV, WSMV or SCMV (Goldberg and Brakke, 1987; Uyemoto *et al*., 1981, Wangai *et al.,* 2012). MLN, also termed corn lethal necrosis (CLN) was first reported in Peru in 1973 (Hebert and Castillo, 1974) with losses of 10 and 15% in floury and sweet corn varieties. Yield reduction of up to 59% has been recorded in experimental plots (Castillo-Loayza, 1977). Later on in 1977, the disease was described in Kansas, United States of America causing losses of between 50% to 90% depending on the variety of maize and the season of the year (Niblett and Claflin, 1978). MLN was then reported in Nebraska (Doupnik, 1979), Hawaii (Jensen *et al*. 1990; Jiang *et al*., 1992), China (Xie *et al*., 2011), Kenya and Tanzania (Wangai *et al*., 2012), Uganda, Rwanda and Democratic Republic of Congo (Adams *et al.,* 2014; Lukanda *et al.,* 2014) and Ethiopia (Mahuku *et al*. 2015a). Since its first record in East Africa, MLN has spread and emerged as a threat to maize based food security in Sub-Saharan Africa (Mahuku *et al*., 2015b, Kiruwa *et al*., 2016).

Although causative agents of MLN have been well described, there is limited literature on the role played by the viral pathogens involved in the synergistic interaction that leads to MLN. Understanding the role of each individual virus particularly the potyviruses will improve the efforts towards controlling MLN including breeding for tolerance/resistance against the viruses and in developing an integrated crop and pest management options. Thus in this review, a comprehensive discussion on the role of such viruses in MLN disease development has been made as a guide in understanding a synergistic interaction in diseases development.

The Infection Cycle by Plant Viruses

The classical infection cycle of plant viruses includes entry into the cell, disassembly of the virus capsids, genome replication and transcription, and the translation of the viral RNA (Kasschau and Carrington, 2001). Resistance of maize plants to virus infection primarily owes to posttranscriptional gene silencing (PTGS). PTGS is a conserved sequence-specific RNA degradation mechanism in most eukaryotic organisms (Incarbone and Donoyer, 2013). It is often associated with methylation of the transcribed region of the silenced gene and with accumulation of small RNAs (21 to 25 nucleotides) homologous to the silenced gene (Molnár *et al.,* 2005). In order for viruses to infect and cause disease in plants they have to suppress this gene silencing strategy. One strategy used by plant viruses to affect this silencing machinery is by

expressing viral suppressors of RNA silencing (VSRs) at a multiple stages (Pumplin and Voinnet, 2013). Those VSRs are among a major requirement for successful colonization of the host plant by the virus. For viral infection to occur there must be cell-to-cell movement as well as long distance transport of the virus through vascular tissues, which requires one or more viral proteins that supply the dedicated movement functions (Syller, 2006). In the interaction between a potyvirus and MCMV, the main causative components of MLN, this dedicated movement function seems to be carried out by the potyvirus. In the recent study by Xia *et al.*, (2016), the accumulations of both MCMV and MCMV-derived siRNAs in maize seemed to be higher during the synergistic infection (with SCMV and MCMV) compared to single infection. This implies that the presence of potyvirus was not only in favor of its own multiplication within the host but also catalyzing the multiplication of the partner co-infecting virus.

Characteristics of MLN-causing Viruses

MCMV from Tombusvirideae family is the only species in the genus *Machlomovirus* (King *et al*., 2011). It has an icosahedral particle with 30 nm in diameter and composed of a single 25 kDa capsid protein subunit encapsidating 4.4 kb single-stranded positive-sense genomic RNA (Nutter *et al.*, 1989; Lommel *et al*., 1991; Scheets 2004; Xie *et al.*, 2010;).

The MLN-causing potyviruses i.e. SCMV, WSMV or MDMV are single stranded, positive-sense RNA genome. They are characterized by induction of pinwheel or scroll-shaped inclusion bodies in the cytoplasm of the infected cells (Edwardson, 1974). These viruses contain a single large open reading frame (ORF) in their genome that is translated into a single polyprotein, which is then autocatalytically digested into about 10 functional proteins: the first protein (P1), helper component proteinase (HC-pro), the third protein (P3), the first 6K protein (6K1), cylindrical inclusion protein (CI), the second 6K protein (6K2), viral genome-linked protein (VPg), nuclear inclusion a protein (NIa), nuclear inclusion protein b (NIb) and coat protein (CP) (Gough *et al*., 1987; Guo *et al*.; Kreuze, 2002).

The Role of Potyvirus in the Synergistic Interaction with MCMV

The mechanism behind synergism between MCMV with a potyvirus is inconclusive in the literature. It is only known that, the region of the potyviral genome that mediates synergism encodes a polyprotein comprising of two potyviral gene products; P1 and HC-Pro, which are both multifunctional (Verchot *et al*., 1991; Verchot and Carrington, 1995; Brantley and Hunt, 1993). The HC-Pro of potyviruses is involved in viral vascular movement and suppression of an antiviral defense mechanism in plants (Savenkov and Valkonen, 2001). In synergism, the presence of one virus leads to the increased replication of another otherwise economically less important virus. For instance in the MLN, concentration of the potyvirus in the synergism is similar to that in a single infection whereas the concentration of MCMV is increased markedly (Xie *et al*., 2016). Two Potyvirus genes, the helper component gene and the gene for nuclear inclusion proteins are potentially avirulent in that they reduce the capacity of maize plants to inhibit the replication of MCMV (Rajamaki and Valkonen, 2009). HC-pro of potyviruses is known to enhace pathogenicity and accumulation of other heterogenous viruses (Pruss *et al.*, 1997). However it is also clear

that MLN induction is independent of the HC-Pro from a potyvirus WSMV, suggesting that this virus utilizes a gene other than HC-Pro to suppress PTGS and mediate synergistic interactions with MCMV (Stenger *et al*., 2007). Such argument provides a clear understanding that more factors are involved in the potyvirus-MCMV synergistic interaction. For instance, in the MLN saga, SCMV offers two proteins that aggravate MCMV replication and severity of symptoms: HC-Pro and nuclear inclusion protein-a and viral genome-linked protein (NIa/VPg) (Kreuze, 2002). SCMV VPg is known to interact with maize elongin C protein (ZmElc) leading to its reduced production as detected in all maize organs, but most highly in leaves and pistil extracts (Zhu, *et al*., 2014). The reduction in the expression of *ZmELc* gene that produces ZmElc protein causes increased replication of MCMV. SCMV VPg is also believed to enhance cell to cell and long distance (systemic) movement of its own virus particles as well as those of MCMV (Barker, H. 1989; Cronin *et al*., 1995). The most important role of the potyvirusHC-pro though is to function in a counterdefensive capacity as a suppressor of PTGS (Kasschau and Carrington, 2001). Furthermore and in similarity with NIa/VPg, HC-Pro of the potyvirus SCMV interacts with ferredoxin-5 (FdV) of maize (Cheng *et al*., 2008) resulting into disturbance in its posttranslational import into maize bundle-sheath cell (BSC) chloroplasts. Ferrodoxins play a key role in the distribution of electrons transferred from photosystem I of photosynthesis to a range of electron acceptors. In leaves under optimal conditions the majority of electron flux through ferrodoxins is used to reduce $NADP^+$ via a ferrodoxin NADP oxidoreductase (FNR). Of the three maize photosynthetic ferredoxin isoproteins (FdI, FdII and FdV), HC-Pro interacts specifically with FdV. The disruption of chloroplast function in maize due to concurrent infection by MCMV and SCMV leads to two things: (1) Production of less ATP required to drive the Calvin cycle through electron flow around photosystem I, which directly leads to low yield and (2) Inadequate production of chlorophyll and symptom expression.

Transmission and Development of MLN

Three main components are important for the MLN disease to occur; the viruses, vectors and a susceptible maize cultivar in a suitable environment (Redinbaugh and Zambrono-Mendoza 2014). For the virus to invade the host it must enter plant cell, replicate in primarily infected cell and move within cells i.e. cell to cell through plasmodesmata and long-distance (leaf to leaf) movement through the vascular system (phloem). Movement of viruses from cell to cell in plants involves one or more viral proteins with special functions. For instance, the case of MLN, the Potyviral HC-Pro has an N-proximal (100 aa) which not only controls virus transmission by aphid vectors but also virulence, genome amplification and virus accumulation. The HC-pro also has a central domain (200 aa) which affects long-distance movement and replication-maintenance functions of the virus, and a C-proximal (150 aa) domains which is a cystein-type proteinase that plays a role in virus cell-to-cell movement (Cronin *et al*., 1995; Kasschau and Carrington, 2001; Syller, 2006). It thus seems doubtlessly that the presence of a potyvirus in the synergistic interaction is very important for development of the disease that seems to be primarily resulting from MCMV.

A model illustrating main stages in disease transmission and development on host is shown in Fig 1. Except for seed transmission

(Jensen *et al.*, 1991 and Zhang *et al.*, 2011, Li *et al* 2011) where the viruses are carried by the seed, the illustration shown here mainly describes other means of viral transmission. Thus, in this model, insect vectors or other viral reservoirs introduce the viruses into plant cell. Penetration of viruses into non-infected plant cells takes place in wounds created by the feeding insect vectors or in other openings in plants including those caused by mechanical injury by human activities. Some insects vectors such as viruliferous beetles spread a layer of pre-digestive materials known as regargitant on host leaves and deposit virus particles in the wound at the feeding site (Trigiano *et al*., 2008). The deposited virus removes the protein coat and nucleic acid enters the nuclear membrane and alters the host DNA replication process by changing its RNA to complementary DNA (cDNA) to mimic its host maize DNA so as to produce many of its copies. When more copies of viral particles have been created, they can move between cells through plasmadermata and the whole maize plant through phloem then colonizes a susceptible host. In a resistant host, the virus colonization cannot be possible thus no MLN symptoms.

Brault *et al*., (2010) indicated that Maize-infecting potyviruses can be transmitted in a non-persistent manner by about 25 aphid species. Seed transmission of SCMV is possible at a rate of 0.4 to 3.9% depending on the genotype (Li *et al*., 2011). Potyviruses cause mosaic symptoms and dwarfing in susceptible maize cultivars.

The main vectors identified through experiments which can transmit MCMV in a semi-persistent manner include Chrysomelid beetles including Diabrotica species (Nault *et al.*, 1978), maize thrips (Frankliniella williamsi) and western flower thrips (*F.occidentialis*) (Cabanas *et al*., 2013; Zhao *et al*., 2014). Mahuku *et al*., (2015b) indicate that there are possibilities that other vectors among thrips, beetles or others insects that are associated with maize can transmit MCMV. Further research is needed to find out whether or not other insects commonly found in maize can transmit MCMV. Seed transmission of the virus is inconclusive. Earlier report by Jensen *et al*., (1991) indicated a rate ranging from 0% to 0.33% for MCMV in 17 lots of maize seed originating from MCMV-infected plant. In a recent study by Mahuku *et al*.,(2015b), 72% of seeds (18 out of 25 seeds) originating from MCMV-infected maize plants and 12 out of 26 ten seed samples pooled from 26 lots of locally purchased seeds were positive with real time polymerase chain reaction (RT-PCR). However, further research is needed to find out whether contaminated seeds can transmit MCMV to progenies. MCMV causes chlorotic mottling to severe mosaic stunting, premature plant death, yellowing and necrosis, sterility of male flowers and shortened, malformed, and partially filled ears depending on developmental stage at the time infection, prevailing environmental condition and genetic background (Wangai *et al.*, 2012, Mahuku *et al.*, 2015a).

Effect of MLN Symptoms on Host

MLN has been identified as the most devastating foliar disease responsible for highest yield loss in maize (Ochieng *et al*., 2012). The two catch terms; 'lethal' and 'necrosis' describes two conditions. The first portrays a disease that 'kills infected plant' and the second term 'necrosis' means a disease which 'seriously kills infected cells'. If the viral pathogens succeed to colonize the host (Fig 1), MLN disease symptoms can develop. Most of the developed symptoms have direct effect to plants growth and development (Table 1). Main symptoms

incited by MLN-interacting viruses in a susceptible host include; yellow streaks parallel to leaf veins, chlorotic mottling, leaf necrosis which, may lead to "dead heart" symptom and plant death, premature aging of the plants (Gordon *et al*., 1984), sterility in male plants and failure to tassel, malformed or no ears, rotting of cobs and failure of cobs to put on grains and (Nelson *et al*., 2011; Wangai *et al*., 2012,; Makone *et al*., 2014).

Potential Impact of MLN on Food Security

MLN is expected to threaten maize production especially in developing countries. We know that, maize is ranked the third most important cereal crop after wheat and rice (Khalili *et al*., 2013) and that more than 1.2 billion small scale farmers in Latin America and Sub-Saharan Africa depend on it as their main staple food and livestock feed (Iken & Amusa, 2004, Onasanya *et al*., 2009). It has been estimated that highly MLN-affected areas can experience a massive yield loss (Wangai *et al*., 2012). Due to dependence of farmers to maize as their main food crop, shortage in its supply can be synonymous with food insecurity. The potential risk of MLN in Africa is high. Considering each individual MLN-causing viruses, Mahuku *et al*., (2015b) indicated that MCMV is considered a primary disease-causing virus behind almost all MLN cases. The MCMV alone has a big potential to establish in warm arid, semi arid and sub-humid tropics (Isabirye and Rwomushana, 2016).

Of the identified potyviruses, MDMV and SCMV are wide spread and cause diseases in maize worldwide (Mahuku *et al*., 2015b). Their widely presence can be indicate their adaptation to their interaction to host plants in areas where MCMV is considered new. Since they are adapted, they have a fully machinery to attack the host, and the host has ways of resisting attack from the virus (Redinbaugh and Zambrano 2014). Since MCMV is new to the crop system, plants seems not to be prepared for attack i.e. plants have little or lacks resistance to the pathogen, and thus the additional weakened effect by the potyviruses or other viruses such as *Maize mosaic virus* and *Maize rayado fino virus* and/or abiotic stress favor their full colonization to maize host (Nelson *et al.*, 2011). As no single germplasm has been identified as resistant to the synergistic-interacting viruses as whole, serious maize loses are expected in Africa, for instance, estimates made with an ecological niche models using a genetic algorithm (GARP) by Isabirye and Rwomushana (2016) show showed that, suitable habitats for MCMV is as high as 662,974 km^2 in Ethiopia, 625,690 km^2 in Tanzania, 615,940 km^2 in D. R. Congo, 361,556 km^2 in Angola, 298,402 km^2 in South Africa and 265,564 km^2 in Madagascar. Swaziland, Burundi, and Rwanda will lose 100% each and Uganda 88.1% in terms of national maize production area. In a synergistic interaction of MCMV with a potyvirus, higher damage to maize crop are expected as it is clear that effects are higher when in combination compared to when MCMV or a potyvirus infects the host individually (Xia *et al.*, 2016).

Management of MLN

Some management principles such as plant quarantine, pathogen eradication, avoidance, plant protection and use of plant resistance has been reviewed by Kiruwa *et al*. (2016). In Africa where MCMV is considered new, scarce information is available on management of MLN. In other countries such as Hawii, integration of cultural practice, host tolerance and suitable

insecticides has been used (Nelson *et al.*, 2011). Work in developing suitable management options such as screening for MLN-tolerant/resistant germplasm and vector control is going on in countries heavily attacked by MLN in Africa (Mahuku *et al*., 2015b).

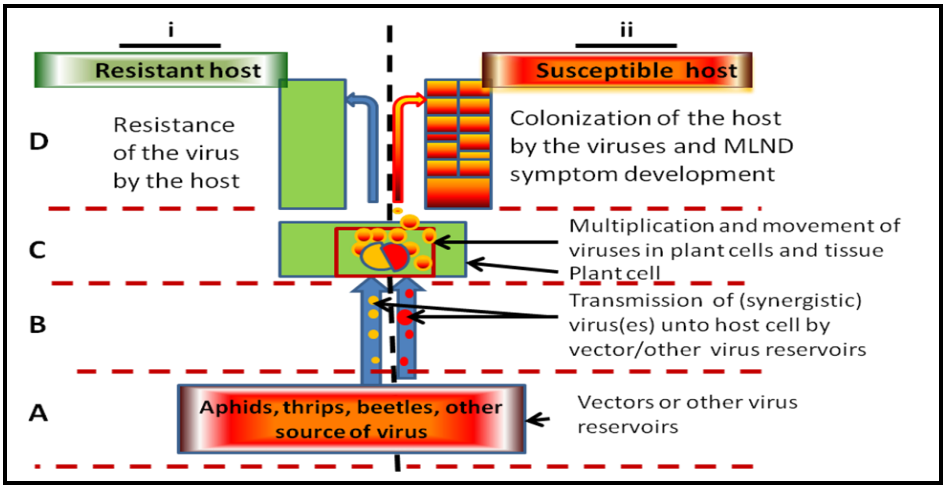
Conclusion and Future Research Needs

In conclusion, this paper discussed the role of potyvirus during its synergism interaction with its co-infecting partner MCMV. It seemed interestingly that in such synergisms, the potyvirus is likely to possess important mechanism for enhanced infection by the co-infecting partner virus which is viewed as the primary disease behind MLN development. One of important proteins playing a significant role in the potyvirus infectivity is the HC-pro, which is multifunctional and possesses counter-defensive capacity to suppress the PTGS of the host. In the synergistic interaction with SCMV and MCMV, the accumulations of both MCMV and MCMV-derived siRNAs in maize is reported to be increased remarkably compared to single infection implying that the presence of potyvirus was not only in favor of its own multiplication within the host but also catalyzing the multiplication of the partner co-infecting virus.

Table.1 Effect of MLN symptoms on host

No	Symptom	Effect
1	Chlorosis	Plant has insufficient photosynthesis
2	Premature aging	Plant receives insufficient heat units required for grain filling
3	Necrosis	reduces total number of photosynthetic cells of the leaf
4	Sterility of male flowers	Leads to infected plants having barren; having small or deformed ears and which set little or no seed.
5	Dying of plant	Total plant failure to reach maturity

Fig.1 A model describes four stages (A-D) of virus synergistic infection on a host plant. A: Virus reservation stage., B: Invasion stage., C: Multiplication and establishment on host and D: Colonization stage. In stage A., vector(s) or other virus reservoir carries the viruses before it becomes in contact with the host then., in stage B., the viruses i.e. MCMV., part (i) and/or a potyvirus part (ii) are transmitted unto the host plant cell where they multiply and establish in a susceptible host (stage C). In stage D., the viruses fail to colonize a resistant host., part (i) or succeed to colonize the host and develop MLN symptoms in a susceptible host., part (ii).



This observation is lightening a way towards developing MLN-resistant maize varieties. The genetics of maize resistance against MDMV and other potyviruses has been described (Jones *et al*., 2007; Redinbaugh and Pratt, 2009; Redinbaugh and Zambrano, 2014). There is need to use such information in initiating marker assisted breeding to halt MLN in areas where maize is the most important crop such as most African countries including Tanzania, Kenya, Rwanda, DRC, Ethiopia and Uganda where MLN has been reported. Though there is no maize genotypes reported to be resistant to MCMV, some tolerant lines have been developed (Redinbaugh and Zambrano, 2014). We know that there is potential for transgenic resistance against MCMV (Murry *et al*., 1993). This potential could also be determined. Since abiotic/environmental stress can exacerbate MCMV infections, there is need to explore environmental conditions and molecular mechanisms behind vector-host-virus interactions to guide designing the MLN-response strategies.

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