

Review on Effect of Maize Lethal Necrosis Disease on Maize Production in Ethiopia

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Abstract: Maize production in Kenya is now under threat from the devastating maize lethal necrosis (MLN) disease in the field. This study was conducted to assess status of MLN in maize seed production fields from both small- and large-scale producers in Kenya. The survey was conducted in five Agro-ecological Zones (AEZ)s in 13 counties in 2015, 2017, 2018, and 2019. Sampling for asymptomatic and non-symptomatic was done using standardized protocol. On-site maize chlorotic mottle virus (MCMV) testing was performed by immunostrips followed by laboratory qRT-PCR test. A total of 2,550 ha was surveyed with 21% having MLN disease and varying levels of severity. The MLN incidence and severity was not significantly different ($P > 0.05$) in the various Kenyan sampled agro-ecological zones, counties, maize varieties, and maize growth stages. Elevated MLN incidences and severities were observed in sub-humid AEZs comprising Embu, Uasin Gishu, Nakuru, and Elegeyo Marakwet counties that form the hotspots for MLN disease. The main MLN-causing viruses detected using q-RT-PCR were MCMV and sugarcane mosaic virus (SCMV). Out of the total samples analyzed using qRT-PCR, 38% were found to have MCMV, 14% with SCMV, and 18% with both MCMV and SCMV. From the 185 sample analyzed with immunostrip from 2017 to 2019, 29 (16%) were positive for MCMV. Phytosanitary programs should be included in seed legislation for legal adoption and strategies to control the spread of MLN disease should focus on high-risk hotspots agro-ecological zones and counties.

Keywords: maize lethal necrosis (MLN) disease, maize chlorotic mottle virus (MCMV), sugarcane mosaic virus (SCMV), hotspots agro-ecological zones, Phytosanitary programs.

1. INTRODUCTION

Maize or corn belongs to the family Poaceae. It is the most important cereal crop in the world (Romay et al., 2013). It grows over a range of agro climatic zones from 58oN to 40 °S below sea level to an altitude higher than 3000m above sea level. The crop grows in areas that receive from 250 mm to 5000mm of rainfall per year (Piperno et al., 2001; Smith et al., 2001).

Despite the role of maize as a source of food in Ethiopia and the effect of MLND in damaging this valuable crop of the country, there is no study that determines the effect of MCMV and SCMV interaction in enhancing the severity of MLN disease in the available maize genotypes of the country. The aim of this study was, therefore, to determine the effect of MCMV and SCMV synergetic interaction in enhancing the severity of MLND in eight maize genotypes from Ethiopia.

2. MAIZE PRODUCTION

Maize is cultivated throughout the year in almost every part of the world. About 875,226,630 tons of maize was produces in 2016 alone and production has increased by 600 million metric tons since 1990 (Incarbon, 2013; Sheets, 1998). World maize production has grown at roughly the same rate as consumption. One mechanism that can be used to increase maize production is increasing the amount of land dedicated to producing it and the area of harvested maize has increased at a rate of 1.32% annually since 1990. Similarly, world maize yield increased at the rate of 1.3% per year from 1990-2016 (FAO, 2017).

In addition to producing maize locally, many African countries import additional maize for food and feed consumption (Trevor et al., 2015). In contrast, some African countries such as South Africa, Uganda, Tanzania, Rwanda and Namibia are important exporters of maize. In 2013, 20% of the worldwide export of maize flour came from Africa, while the USA and France accounted for 14.9% and 10.5%, respectively (Daly et al., 2016).

More than 75 % of maize production in Africa is done by small-scale farmers, while some large-scale farmers mainly work for global export (Nuss and Tanumihardjo, 2010). In Ethiopia, smallholder farms account for more than 95 % and use draft animals for land preparation and cultivation. Approximately 88% of maize produced in Ethiopia is consumed as food, both as green and dry grain (Demeke, 2012).

3. MAIZE PRODUCTION CONSTRAINTS

Socio-economic, technological, policy, abiotic and biotic factors are some of the constraints of maize production (Oscar, 2009). While factors like decline in soil fertility, low soil pH with associated nutrient deficiencies and toxicities make the abiotic factors; insect, viral and fungal infections are the biotic constraints, which affect maize production (Ali et al., 2011).

Viruses are among the most abundant forms of biotic factors in maize production, and exceed the number of host cells by at least one order of magnitude (Cheng et al., 2008; FAO, 2006). Worldwide, more than 50 viruses have been reported in maize causing an array of symptoms in single or mixed infections (Lapierre and Signoret, 2004; Redinbaugh and Zambrano, 2014). Of these, at least dozen viruses have been considered important in terms of prevalence and economic losses in several maize producing areas (Redinbaugh and Pratt, 2008). MLN is one of the most devastating viral diseases reported (Mahuku et al., 2015a).

In Ethiopia, maize production is characterized by variability of yield due to different factors including size of area cultivated, amount and quality of improved seeds, chemical fertilizers and policy environment. There are also different risk factors, which adversely affect maize yield. Weather risk and market risks are the major challenges for farmers. Various biotic, abiotic and socioeconomic, weeds, pest and diseases, erratic rainfall, erosion, low soil fertility, poor infrastructure, and post-harvest crop losses also adversely affect production (Cheng et al., 2008; FAO, 2006). MLN is currently considered as an emerging diseases and a top priority research question. The disease was caused by double infection of maize plants by Maize chlorotic mottle virus (MCMV) in combination with any of the cereal viruses in the family Potyviridae, such as Sugarcane mosaic virus (SCMV), Maize dwarf mosaic virus (MDMV) or Wheat streak mosaic virus (WSMV) (Cabanas, 2013). Among maize production constraints MLND is the main concern of this study.

4. MAIZE LETHAL NECROSIS DISEASE (MLND)

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, respectively. 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 SubSaharan Africa (Mahuku et al., 2015b; Kiruwa et al., 2016).

MLN is a serious disease of maize which is now found in many countries of East Africa where maize is grown. The disease naturally affects varieties of maize resulting in chlorotic mottling of the leaves, severe stunting and necrosis, often leading to plant death.

Most frequently it is SCMV in synergism with MCMV which cause MLND. Single infections of MCMV or SCMV cause only mild mosaic or mottling symptoms and a moderate reduction of growth. In mixed infections, early infected plants appear stunted and show a general chlorosis, leaf bleaching and necrosis (Cabanas et al., 2013).

5. MAIZE CHLOROTIC MOTTLE VIRUS (MCMV)

MCMV is a single-stranded RNA virus with isometric virions, single-component particles and have a smooth spherical or hexagonal shape (King et al., 2011). At least two genetically and geographically distinct strains of MCMV have been reported, MCMV-P (Peru) and MCMV-K (Kansas) (Nyvall 1999).

MCMV transmission occurs through insect vectors, mechanically, and by seed at very low rates (Jensen et al., 1991). It is also possibly transmitted through infested soil, as the virus can survive in maize residue (Nyvall, 1999). In addition, leaf beetles such as *Chaetocnema pulicaria* and *Diabrotica* can transmit this virus over a short period of time. Reports also indicated that it is transmitted at very low rates via infected seed and continuous maize production in a field greatly increases the incidence of maize chlorotic mottle virus (Makone et al., 2014).

MCMV infected young leaf shows fine chlorotic spots that coalesce and develop into broad chlorotic stripes along the veins. These chlorotic stripes contrast with dark green tissue when observed against the light. Leaves showing chlorosis finally die. Plants are stunted because of shortened internodes. Infected plants produce fewer and smaller ears. In most cases, the male inflorescence is malformed.

6. MLND SYMPTOM AND TRANSMISSION

Age at the time of infection, environment, and maize variety or genotype is some of the factors affecting MLND symptoms (Scheets, 2004). Symptoms of MLND include leaf mosaic with fine chlorotic, longitudinal yellow streaks parallel to leaf veins developing about 10 days after inoculation. Streaks may coalesce to create chlorotic mottling. Chlorotic mottling may be followed by leaf necrosis, stunting, shortening of Ears, often with prematurely aged husks and plant death (Nelson et al., 2011).

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 (Incarbon 2013, Sheets 1998; Uyemoto 1981), sterility in male plants and failure to tassel; malformed or no ears, rotting of cobs and failure of cobs to put on grains (Nelson et al., 2011; Makone et al., 2014). 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” describe two conditions. The first portrays a disease that kills infected plant and the second term means a disease which seriously kills infected cells. If the viral pathogens succeed to colonize the host, MLN disease symptoms can develop. Most of the developed symptoms listed above have direct effect on plants growth and development (Mbega et al., 2016).

7. HOST RANGE FOR MCMV

Maize is the only natural host reported for MCMV. Hosts that can be infected experimentally are limited to the grasses in the family Poaceae. Among these grasses, 73 plant species in 35 genera have been tested for susceptibility to virus strains MCMV-Kansas and MCMV-Peru (Scheets, 2004).

8. MECHANISM OF SYNERGISM

Viruses, vectors and susceptible maize cultivars in a suitable environment are the three main components for the MLN disease to occur (Redinbaugh and Zambrano, 2014). For the virus to invade the host it must enter a 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.

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 scrollshaped 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 automatically 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 protein a (NIa), nuclear inclusion protein b (NIb) and coat protein (CP) (Gough et al., 1987; Kreuze, 2002).

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). It 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 cysteine-type proteinase that plays a role in virus cell-to-cell movement (Kasschau and Carrington, 2001; Syller, 2012). It doubtlessly seems 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.

The 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 colonize a susceptible host (Mbegaet al, 2016). In synergism, the presence of one virus leads to the increased replication of another. In MLN, concentration of the Potyvirus in the synergism is similar to that in a single infection whereas the concentration of MCMV is increased clearly (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 Potyvirus known to enhance pathogenicity and accumulation of other viruses (Prusset al., 1997). However, it is also clear that MLN induction is independent of the HC-Pro for a Potyvirus wheat streak mosaic virus (WSMV), suggesting that this virus utilizes a gene other than HC-Pro to suppress posttranscriptional gene silencing (PTGS) and mediate synergistic interactions with MCMV (Stengeret al., 2007).

In the MLN history, 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 (Zhaoet 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, 1989; Cronin et al., 1995).

The most important role of the Potyvirus HC-pro though is to function in a counter defensive 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. Ferredoxins 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 ferredoxins is used to reduce NADP⁺ via a ferredoxin NADP oxidoreductase (FNR). Of the three maize photosynthetic ferredoxinisoproteins (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 driving the Calvin Cycle through electron flow around photosystem I, which directly leads to low yield and (2) Inadequate production of chlorophyll and symptom expression (Mbega et al., 2016).

9. INFECTION CYCLE

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 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 (Molnar 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 stage (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, 2012).

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 (Mbegaet al., 2016).

10. PLANT RESISTANCE FOR VIRAL DISEASE

Virus infection in a host plant activates a defense mechanism related to PTGS that causes degradation of viral RNA and slows down or limits virus accumulation and systemic infection. This process is triggered by double-stranded RNA (dsRNA), produced by replicative intermediates of single-stranded RNA (ssRNA) viruses, or by genomic or defective viral ssRNAs with extensive secondary structure, which is cleaved by Dicerlike enzymes to produce 21–24 nucleotide (nt) fragments called small interfering RNAs (siRNAs) (Cheng et al., 2008).

The siRNAs are incorporated into a large ribonucleoprotein complex (the RNA inducing silencing complex, RISC) that guides sequence-specific cleavage of the target RNA. siRNAs may also play a role in amplification of dsRNA by a plant encoded RNAdependent RNA polymerase (RdRp), that in turn is degraded by Dicer-like enzymes to produce secondary siRNAs (Cheng et al., 2008).

As a counter defence, plant viruses encode proteins that suppress RNA silencing to overcome this defence mechanism (Diaz- Pendon and Ding, 2008). Temperature affects plant-pathogen interactions, and a higher growth temperature may either increase or decrease disease resistance, thus reflecting a differential influence of the same temperature variation on different plant-pathogen systems (Incarbon 2013, Sheet 1998; wangai et al., 2012). Symptoms induced by many plant viruses are attenuated when plants are grown at high temperature, a phenomenon that might be related to different efficiency of the host defense system (Vela et al., 2010).

11. MAIZE RESISTANCE FOR VIRUSES

The most effective means of managing MLN would be the use of tolerant or resistant varieties. A trial performed by Nelson et al. (2011) in Hawaii found many tropical inbred lines and varieties to be highly resistant to MCMV. According to the report, 30 out of 40 (75%) of University of Hawaii bred field maize inbred lines tested positive for resistance; however, no complete immunity was observed. Almost all temperate climate inbred lines and hybrids are highly susceptible to the virus (Nelson et al., 2011). The level of MCMV resistance varies widely among pure lines that have been tested in Hawaii, so it is considered a quantitative trait (Nelson et al., 2011). Preliminary inheritance studies on the inheritance of traits suggest a polygenic control of the disease, with resistance being partially dominant. This encourages the commercial production of hybrids only if both parents are resistant to the pathogen.

In Kenya, varieties are being screened for resistance by KARI and CIMMYT in two sites; Naivasha and Bomet. Preliminary data from 43 pre-commercial maize hybrids and seven commercial hybrids at Bomet, Chepkitwal and Naivasha, and 200 elite inbred lines at Naivasha, during one season of screening under natural disease pressure, suggest that MLN-resistant maize germplasm can be identified and developed quickly. KARI, CIMMYT and other partners will be expected to reconfirm the potential resistance of precommercial hybrids and inbred lines that show the lowest susceptibility to MLN and work urgently to develop resistant varieties (Makumbi and Wangai, 2013).

As MLN is due to the co-infection of two viruses, resistance against any one of the viruses would substantially reduce the damage due to the disease. Results of a trial of elite CIMMYT inbred lines under artificial SCMV inoculation showed several highlyresistant lines (Makumbi and Wangai et al, 2013). In the long run, deployment of varieties that are resistant to both MCMV and SCMV will be the best means of managing MLND (Incarbon, 2013; Sheets, 1998; Uyemoto, 1981).

12. EFFECT OF MLND

MLN is expected to threaten maize production in developing countries. Maize is ranked the third most important cereal crop after wheat and rice (Khaliliet 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 and Amusa, 2004). It has been estimated that highly MLN-affected areas can experience a massive yield loss (Incarbon2013,Sheet 1998;Uyemoto1981). Due to dependence of farmers on maize as their main food crop, shortage in its supply can be synonymous with food insecurity.

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 (Mahukuet al., 2015b). Their widely presence can be indicative of their adaptation and interaction with host plants in areas where MCMV is considered new. Since they are adapted, they have a full 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 have little or lack resistance to the pathogen, and thus the additional weakened effect by the Potyviruses 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 losses are expected in Africa. Estimates made with an ecological niche models using a genetic algorithm (GARP) by Isabirye and Rwomushana (2016) showed that, suitable habitats for MCMV is as high as 662,974 km² in Ethiopia, 625,690 km² in Tanzania, 615,940 km² in D. R. Congo, 361,556 km² in Angola, 298,402 km² in South Africa and 265,564 km² in Madagascar. Swaziland, Burundi, and Rwanda 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 is 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).

In Kenya, in areas where MLND was very serious, farmers experienced extensive or complete crop loss (Incarbon 2013; Wangai et al., 2012). The infected plants are frequently barren; the ears formed are small, deformed and set little or no seeds, drastically reducing the yield. The areas affected constitute major maize production acreage and given the recorded loss of up to 100%, it has become an important food security issue in Kenya. Infection rates and damage can be very high, seriously affecting yields and sometimes causing complete loss of the crop (Adams et al., 2012). Infected plants are frequently barren; ears formed may be small or deformed and set little or no seed.

The impact of the disease can be felt in the whole maize value chain. To control MLND, the maize seeds have to be dressed with an insecticide in addition to a fungicide seed dressing. Seed producers have incurred an extra cost in the production of seed maize.

13. VIRUS DIAGNOSIS

Identification of MLND and the viruses involved in the disease complex is generally by observation of symptoms in the field. However, because single infections of the viruses and early stages of the disease are often inconspicuous and resemble physiological disorders, specific diagnostic tests are to be applied to confirm virus presence and to adequately detect/identify the viruses in the mixed infection (DSMZ, 2014). ELISA tests are the most reliable assays for detection of these viruses in maize as both viruses reach concentrations in maize that can easily be detected by ELISA. Specific polyclonal antibodies developed at the DSMZ Plant Virus Department allow a sensitive and reliable detection and identification of MCMV and SCMV in a standard double antibody sandwich enzyme linked immuno-sorbent assay (DAS-ELISA) (Cabanas et al., 2013).

PCR method is also used in many applications (Doughriet al., 2009) including diagnostics of plant virus diseases (Henson and French, 1993; Hadidiet al., 1995; Lopez et al., 2003) because of its speed, specificity, sensitivity, and versatility (Naidu et al., 2003). Apart from detection of viruses, PCR products can be sequenced to provide further data on strain types (Webster et al., 2004). Sequencing is a very reliable technique for virus identification and has led to development of strain specific probes and primers from extensive sequence data available from many viral isolates. Next-generation sequencing (NGS) is one of the modern techniques that have been used in the diagnosis of new unidentified viral plant diseases (Punja et al., 2007).

14. PREVENTION AND CONTROL MECHANISM

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 Hawaii, 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).

Few studies have determined the incidence of MLND and diversities in SCMV and MCMV in some parts of Ethiopia, the synergistic interaction of SCMV and MCMV, as well as the possible role of such interaction in increasing the severity of MLND received little attention.

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