RICE YELLOW MOTTLE SOBOMOVIRUS: A LIMITING FACTOR IN RICE PRODUCTION IN AFRICA

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Abstract: Rice is the primary and secondary host of many viruses, but the most important in Africa is the rice yellow mottle virus (RYMV) genus Sobemovirus. It is endemic in the continent and became important after the introduction of new high yielding exotic varieties from Asia that are susceptible to the virus. The pathogen was first noticed in 1966 in Kenya but it has since spread to other parts of Africa countries. It is environmentally stable, highly infectious and about six strains of the virus now exist. Transmitted both mechanically and by insect vectors belonging to the families Chrysomelidae (*Chaetocnema* spp., *Dactylispa* spp., *Hispa unsambarica* Weise, *Sesselia pusilla* Gartucker, *Trichispa sericea* Guerin); Tettigonidae (*Conocephalus longipennis* de Haan, *C. merumontamus* Sjostedt) and Coccinelidane (*Chnootriba similis* Thunberg). Yield losses caused by the virus range from 25 to 100%. Integrated pest management and breeding for resistant varieties are the best strategies so far suggested to reduce havoc caused by this pathogen to rice. This paper reviews the economic importance, distribution, host range, symptom, transmission, varietal reaction, yield loss assessment, epidemiology, molecular characteristics, management strategies and future research needs of the virus.

Keywords: Epidemiology, host range, molecular characterization, symptomatology

Introduction

Rice (*Oryza sativa* L.) plant is annual grasses belonging to the genus *Oryzeae* in the family Poaceae. It can grow for more than a year under favourable conditions and are well adapted to aquatic habitats. Rice is produced in about 10% of all cropland (144 million ha) in over 110 countries. It has the ability to adapt to diverse environmental conditions and is therefore, grown in varied climatic conditions ranging from Sahel to the Rainforest ecological zone (Luh, 1980) to varying degrees in every country in West Africa (WARDA, 1995). The main production ecologies are rainfed lowland, rainfed upland, irrigated lowland, deep water/flooding and mangrove swamp (Onyishi *et al.*, 2010).

Rice is a staple food for about one half of people in the world and is mainly harvested and consumed in Asia and Africa (Anonymous, 2005; Sun *et al.*, 2006). In many regions it is eaten with every meal and provides more calories than any other single food (Hynes, 2007). It is not only consumed by humans and fed to livestock, it is also useful industrially for processing into acetic acid, glucose and starch. Rice straw and stems are used by local farmers as bedding for animals and for weaving roofs, hats, baskets and sandals;while the inedible rice hull is used as fuel, fertilizer and insulation (Schalbroeck, 2001; Hynes, 2007). Two species of rice were domesticated; *Oryzae sativa* L. and *O. glaberrima* Steud. Cultivar of the rice species *O. sativa* are widely grown in Asia, whereas *O. glaberrima* have been grow for ages in Africa (Sarra, 2001). In the last century several *O. glaberrima* cultivars were replaced by higher yielding *O. sativa* cultivars in Africa, resulted in the emergence of RYMV (Abo *et al.*, 1998) causing a highly infectious disease in most rice growing regions with epidemics proportions in irrigated and lowland cultivated rice in the 1990s (Konate *et al.*, 2002).

Rice is the primary and secondary host of many viruses (Koklu and Yilmaz, 2004) but the most important is the rice yellow mottle virus (RYMV) (Awoderu, 1991a; Fargette *et al.*, 2002). The pathogen was first noticed in 1966 in Kenya (Bakker, 1970). However, it has since spread to other parts of Africa (Koklu and Yilmaz, 2004).The virus is characterized by high genetic variability due to its intrinsic high rates of mutation which is evident from the detection of several strains of the pathogen (N'Guessan *et al.*, 2001; Pinel- Galzi and Fargette, 2006). Six strains of the virus have been reported so far: S1, S2 and S3 in West and Central Africa, S4, S5 and S6 in East Africa (Pinel *et al.*, 2000; Fargette *et al.*, 2002; Banwo *et al.*, 2004b). Both virulent and avirulent isolates were found among the strains, indicating that phytopathological traits of an isolate are not hereditary or confined to a specific strain or a particular region, but occurs naturally in different lineages in different parts of Africa (Sorha *et al.*, 2005). Generally, RYMV infection is characterized by mottle and yellowing symptoms of varying intensities, depending on rice genotype and age at infection (Bakker, 1970; Thottappilly and Rossel, 1993a), as well as the virulence of the invading virus strain. Symptoms occur at any stage from transplanting to booting (Abo *et al.*, 1998). It is environmentally stable and is transmitted mechanically from rice to rice (Bakker, 1971; Fauquet and Thouvenel, 1977) and by insect vectors belonging to the families Chrysomelidae (*Chaetocnema* spp., *Dactylispa* spp., *Hispa unsambarica* Weise, *Sesselia pusilla* Gartucker, *Trichispa sericea* Guerin); Tettigonidae (*Conocephalus longipennis* de Haan, *C. merumontamus* Sjostedt) and Coccinelidane (*Chnootriba similis* Thunberg) (Bakker, 1971; Reckhaus and Adamou, 1986; Abo, 1998; Banwo, 2002).

The disease has fast become a major limiting factor to rice production for lowland and irrigation ecosystems in Africa. It is now found in virtually all rice growing countries in West Africa (Fomba, 1990; Aworedu *et al.*, 1991a). According to Traore *et al.* (2005), RYMV occurs in diverse cultivated rice system (rainfed, irrigated lowland mangrove), in all kinds of ecosystems (forest, woodland Savannah, grass Savannah up to the Sahelian region as well as upland Savannah), in countries where rice is either a main or a minor crop and also where epidemics are widespread or localized. Diversification and spread of RYMV has been concomitant with an extension of rice cultivation in Africa since the 19 century. This major agro- ecological change increased the encounter between primary hosts of RYMV and the cultivated rice; it also modified the landscape ecology in ways that facilitated virus spread (Traore *et al.*, 2009). It is known to be naturally very destructive and therefore threatening the rice production in the African continent on a large scale (Banwo *et al.*, 2004a) rapidly spreading among and within the major rice growing areas and cause severe yield losses ranging from 25 to 100% depending on the date and time of infection as well as type of

rice variety (Rossel *et al.*, 1982b; Taylor *et al.*, 1990; Sy, 1994; Konate *et al.*, 1997). This paper reports some of the achievements of earlier works on the economic importance, distribution, host range, symptom, transmission, varietal reaction, yield loss assessment, epidemiology, molecular characteristics, management strategies adopted so far, and suggestions on future research needs of the virus.

Importance of rice yellow mottle virus genus sobemovirus (RYMV)

The increasing incidence and importance of RYMV in East and West Africa are attributed to the cultivation of new highly susceptible exotic rice varieties, mostly from Asia and the availability of water through irrigation, which allows for sequential planting and maintenance of higher crop intensity without dry season gaps (IITA, 1981, 1982; Rossel *et al.*, 1982b). In Africa five viruses, *Rice necrosis stripe* (RNSV) Genus *Furovirus*,*Maize Streak virus* (MSV)Genus *Mastrevirus, African cereal streak virus*, Rice crinkle disease and *Rice yellow mottle virus(*RYMV) GenusSobemovirus (Harder and Bakker, 1973; Bakker, 1975; Thottappilly, 1992) have been reported to infect rice. The latter is the only economically important virus disease (Rossel and Thottappilly, 1985). The disease is fast becoming a major limiting factor to rice production for lowland and irrigated ecosystems in Africa. It is now found in virtually all rice growing countries in Eastern, Western and Southern Africa (Fomba, 1990; Awoderu *et al.*, 1991b; Banwo *et al.*, 2004a). It has reached epidemic proportions in some African countries and is spreading fast in others (Reckhaus and Adamou, 1986; John *et al.*, 1984*;* Sy, 1994; Abo *et al.*, 1998).

Losses caused by RYMV in paddy fields have reached alarming proportions and some farmers have suffered complete crop failure (Yaboue, 1989; Alegbejo *et al*., 2006). RYMV causes a severe disease of rice in most rice growing countries in Africa, yield losses of 58-67% were reported in Niger (Reckhaus and Adamou, 1986), 54-97% in Sierra Leone (Taylor, 1989), 20 -45% in Burkina Faso (Sere, 1991), 67-84% in Cote d'Ivoire and of 64-100% in Mali (Sy *et al.*, 1993), in Kenya the yield reduction of variety Sindano was estimated at 50% (Nwilene *et al*., 2009a). In Tanzania it was reported that some farmers could not harvest any rice while others completely abandoned their fields (Banwo *et al.*, 2004a). Crop abandonment due to the infection of rice fields by RYMV was also reported in Zamfara state, Nigeria (Alegbejo *et al.*, 2006). It causes no damage at all in some years but then reach epidemic proportions when climatic and conditions are exactly right for their development (Nwilene *et al*., 2009a).

Distribution ofRYMV

Incidence of RYMV was first reported in 1966 in Kenya (Bakker, 1970) but has now been reported in many different countries in Central, East and West Africa. It was reported in 1976 in Liberia, Sierra Leone and Tanzania (Raymundo and Buddengan, 1976; Rossel *et al*., 1982). In 1977 it was reported in Cote d'Ivoire (Fauquet and Thouvenel, 1977) and in 1980 in Ghana and Guinea (Raymundo and Konteh, 1980). By 1984 it was identified in Burkina Faso, Malawi, Mali, Niger and Senegal (John *et al*., 1984) and in Rwanda in 1987 (Awoderu *et al*., 1987). Later in 1989 it was described in Madagascar (Reckhaus and Randrianangaly, 1990). In 2001 it was noticed in Cameroun and Chad (Traure *et al*, 2001).The first incidence of RYMV in Nigeria was reported in Niger and Oyo states in 1975 (Raymundo and Buddengan, 1976). The virus was later found in Anambara and Imo states (Awoderu, 1991), Akwa Ibom, Ebonyi and Sokoto states (Singh *et al*., 1997), Zamfara state (Alegbejo *et al*., 2001), Bauchi, Gombe and Kano states (Abo *et al*., 2002) Borno state (Alkali *et al*., 2014a).

Host range of RYMV

Host range is narrow, outside the genus *Oryza.* Some *Graminaceous* species particularly in the tribe *Eragrostideae* were found to be systemic hosts of RYMV (Bakker, 1974, 1975). The grass species reported as diagnostic hosts include *Eleusine indica* Gaertiner, *Echinochloa crus-galli* L.P. Beak, *Dinebra retroflexa,* (Wahl), Ponzer, *Eragrostis tenifolia,* (*A.* Rich)Steud., *Panicummaximum* Jacq (Bakker, 1974, 1975; Raymundo *et al.*, 1979; Okiome *et al.*, 1983; Taylor *et al.*, 1986; Awoderu, 1991a; Nwilene *et al*., 2009a). Abo (1998) found systemic infection of RYMV on rice only and infections of localized nature was obtained with *O. longistaminata, Eragrostis ciliaris, (L) R.* Br., *Echinochloa crus-pavonis* Scultes and *E. Pyramidalis* Hiche&Chase. RYMV was also found in *O longsstaminata* A.Chev.andRoehr.growing in marshy areas and rice fields in Niger and Mali (John *et al.*, 1984; Sy, 1994, Hamadoun *et al.*, 1995), Senegal (Mbodj *et al.*, 1984), and Burkina faso (Konate, 1995). Sy (1994) reported that RYMV – like symptoms were observed visually on *O. longistaminata, O. glaberrina* and I*mperata* sp. in lowland in Mali. *Cynodon dactylon, Cyprus esculentus* L., *C. rotundus* L., *Eleocharis complanata, Eleusine indica, E. cofana, Fuirena umbellata, Imperata cylindrica, Kyllinga pumila* and *Paspalum vaginatum* were reported by Salaudeen *et al*. (2008c) as weed hosts of RYMV in Nigeria.

No infection was obtained with cultivated cereals such as *Sorghumbicolor* (L) Moench, *Zea mays* L., *Pennisetum americanum (L.)* Lecke *and Triticum aestinum* L; neither with some sedges and broad leaved weeds (Abo, 1998). Konate *et al.* (1997) and Nwilene *et al.* (2009a) reported that most of the field samples were from *Oryza* species and NERICA progenies and from the wild grasses belonging to the *Poaceae* family. Thus it is evident that the host range of RYMV is narrow.

RYMV is highly stable in crude, dry and frozen leaves and highly infectious (Bakker, 1974; Fauquet and Thouvenel, 1977). Sap derived from young infected rice leaves (2 – 3 weeks old) is still infective at a dilution of 10^{-11}; however, the dilution end point varies with the origin of the inoculum (Fauquet and Thouvenel, 1987). The virus remains infective for 35 days at 27 to 29^{0}C but when stored at 9^{0}C, the sap remains infective for 71 days (Bakker, 1970, 1974) and at 4^{0}C for 84 days (Fauquet and Thouvenel, 1987). However, the inoculation progressively loses its capacity for infection from 55 to 70^{0}C, although in crude sap the virus remains viable for at least 34 days at 27^{0}C (Fauquet and Thouvenel, 1987). It can replicate at a surprisingly rapid rate (Fauquet *et al.*, 1995).

Symptoms of RYMV

The unique characteristic symptoms of RYMV are leaf mottling and yellowing, grain discoloration, grain or spikelet sterility and stunting of the plants (Bakker, 1974; Raymundo and Bunddenhagen, 1976; Fauquet and Thouvenel, 1977; Rossel *et al*., 1982a; IITA, 1983; John *et al.*, 1984; Rossel and Thottappilly, 1985; Taylor *et al.*, 1990; Thottappilly and Rossel, 1993b). Raymundo and Buddenhagen (1976) stated that infected plants had pale yellow mottle leaves, conspicuous bronze or orange colouration, rolling of leaf margins, leaf desiccation, stunted, reduced tillering, non-synchronous flowering, poor panicle exertion and malformation, spikelet discolouration and sterility, erect panicles, compact appearance of spikelet, brown to dark-brown discolouration of grains (a consequence of secondary infection by Fungi) and crinkling of new growth.

Infected rice plants are found first near the bunds and thereafter whole fields may be infected (Bakker, 1975). Also Fauquet and Thouvenel (1977) reported that the disease appeared as a yellow and green mottle at the leaf tip. In an infested field, yellow-orange discolourations of leaves were

seen on plants scattered throughout the field but such plants were more prevalent at field margins. Leaf mottling and yellowing symptoms of varying intensities may be developed depending on the genotype. It could be mistaken for iron or nitrogen-deficiency (Thottappilly and Rossel, 1993b) as well as iron toxicity damage (Bakker, 1975). Bakker (1974) reported yellowish leaves on cultivar Sindano, mild yellowish green on cultivar Basmati 217 or orange on IR8. Raymundo and Buddenhagen (1976) stated that some varieties continued to produce relatively green leaves with faint, hardly discernable streaking and mottling but with conspicuous stunting, others with narrow and very chlorotic leaves and severe stunting, and still some showed pronounced chlorosis but minimal stunting. Others developed conspicuous bronze or orange pigmentation followed by rolling of leaf margins and subsequent leaf desiccation, malformation and incomplete emergence of panicles and sterility are observation infected rice plants. Symptoms occur at any stage from transplanting to booting (Fauquet and Thouvenel, 1977; Nwilene *et al*., 2009b) and are more pronounced on ratoons (Raymundo *et al.*, 1979). In severe cases, affected plants may die (Bakker, 1974; Fomba, 1988; Thottappilly and Rossel, 1993b).

Symptom expression of RYMV may be strongly influenced by light intensity, day length, humidity, temperature, growth stage of plant, nutrient status, among other factors (Bakker, 1974; Albar *et al*., 1995; Nwilene *et al*., 2009a). According to Bakker (1974) "Sindano variety at the 3 – 4 leaf stage showed at high temperature (30^0C or more) the first systemic symptoms after 4 – 5 days, at 25^0C the symptoms were noticed after 6 days, and at 20^0C after 7 – 8 days. During prolonged periods of temperatures below 20^0C, the yellow dots enlarged more slowly parallel to the veins that at 25^0C, while at higher temperatures the period in which only yellow dots were seen was very short. Inoculation of "Sindano" at increasing age delayed symptom appearance and the symptoms were more pronounced and necrotic in the glass-house than in the field (Bakker, 1974). Awoderu, (1991) and Albar *et al*. (2005) stated that temperature affected symptom expression differently in host plant. At low temperature RYMV show only mild symptom while at high temperature severe symptom developed and also greater expression of RYMV symptoms when the relative humidity was low.

Transmission of RYMV

Rice is known to be attacked by more than 80 species of insects (Alam *et al.*, 1984). The insects infest rice at all stages and feed on all parts of the plant (Banwo *et al.*, 2004a). RYMV is transmitted by insects with biting and chewing mouthparts (Hull, 1988). However, a few insects belonging mainly to the family Chrysomelidae have been found to transmit RYMV in East Africa (Bakker, 1971, 1975) and Madagascar (Reckhaus and Andriamasintsheneno, 1995). These insects include *Trichisp*a *sericea*Guerin.,*Sesselia pussila, Dicladispa* spp., *Chaetocnema* spp. and those in the genus near Apophylia (Bakker, 1974, 1975). *T. sericea*. is identified as the major vector in Niger and Mali (Reckhaus and Adamou, 1986; Sy, 1994), and *Chaetocnema* spp. were suspected by Raymundo and Buddenhagen (1976), Raymundo (1980), Fomba (1990) to be responsible for the spread of RYMV in West Africa. Nwilene *et al*. (2009a) reported new insect vectors of RYMV including; *Zonocerus variegatus, Paratettix sp. Cheilomenes lunata, Euscyrtus sp., Cofana spectra, Cofana unimaculata and Locris rubra.* The long horned grasshopper (*Conocephalus merumontame* Syostedt) (Tettigomidae) and mites from the families Eriophydidae and Tarsonemidae have been indicated as transmitting the virus in Kenya at low levels but possible contamination of the plant by these vectors was not over-ruled (Bakker, 1974).

RYMV can also be transmitted through other sources. The virus is transmitted mechanically through sap (Bakker, 1974, 1975; Fauquet and Thouvenel, 1977). It could also be transmitted by farm implements such as the sickle used in harvesting rice, hoe and through the cultural practice of trimming shoots of rice seedlings at transplanting (Tsuboi *et al.*, 1995), through leaf contact in closely spaced plants, by contaminated hands, rice stubbles incorporated into the soil, and through rice roots inter-twined together (Abo, 1998; Nwilene *et al*., 2009b). Cow dung and soil containing insufficiently decomposed plant material can also contribute to infection with RYMV (Reckhaus and Andriamasintseheno, 1995).

The virus was found in leaf debris and empty rice spikelets *(*Abo and Sy, 1998). RYMV could then be transported in rice sacks containing contaminated and partially winnowed rice seeds to new areas far from its origin and spread there. In Mali 100% infections were reported to occur in transplanted rice fields but not in directly seeded fields thus indicating a possible connection with the infection from the nursery (Sy, 1994). It could also be transmitted through guttation fluid and in the irrigation water of heavily infected rice fields, cow dung, rice straw, rice ratoons, weeds and wild rice (Bakker, 1974; Nwilene *et al*., 2009b). Most likely where insects feed and caused injury and damage to the plants and their dead bodies might have contaminated the water with virus particles (Banwo *et al*., 2004a).

Epidemiology

Many factors influence the development of epidemics and it is simplistic to assume that there can be any single underlying cause of the serious epidemics of diseases (Thresh, 1989). Onasanya *et al*. (2012) reported that epidemics of RYMV in natural condition, insect vectors played a significant role in disease epidemic as they were able to transfer RYMV from surrounding infected field and contaminated weed to the new rice fields. Many cultural practices favour RYMV epidemics: uprooting and bundling of seedlings from nurseries to permanent field provides a means of transmission through contact between healthy and diseased seedlings (Sarra, 2005; Traoré *et al*., 2006); farmers own livestock and after harvesting, they allow cattle to graze on regenerating stubbles so that the dung deposited adds manure to fertilise the subsequent crops; this enhances the virus build-up for cattle, beetles, donkeys and grass rats which all contribute to the epidemic of the virus (Absa, 2010); harvesting using sickle without distinguishing the healthy from the diseased plants, increases the incidence of disease in the subsequent rice regrowth from the remaining stubbles and thus build-up RYMV inoculum reservoir (Sarra, 2005); Traoré *et al*. (2009) reported that most epidemic occur in areas where irrigated rice is grown and also to a lesser extent when water is available for several months during the rainy season where lowlandrice is grown. The worst epidemics of RYMV disease were experienced in Niger (Reckhaus and Adamou, 1986; John *et al.*, 1986), Mali (Sy, 1994) and Madagascar (Reckhaus and Andriamassintseheno, 1995). The disease is still persisting in these countries. The disease was also severe in Cote d'Ivoire (Singh, 1995). It is increasing in other countries of Africa (Abo *et al.*, 1995). The disease has come to the foreground due to intensification and modification of rice culture which has led to major epidemics of rice viruses revealed by growing susceptible varieties, fertilizer and pesticides uses (Thresh, 1989). It is on record that any country where RYMV disease is a serious problem has one or more highly susceptible varieties on a large acreage. For example, Sindano, Basmati 217 and IR 22 in East Africa, BG 90-2 in Mali, Bouake 189 in Cote d'ivoire, IR 1529-680-3 in Niger, Jaya in Senegal, ITA 123 in Burkina Faso, ITA 212 (FARO 35) and FARO 29 (BG 90-2) in Nigeria and IR8 and IR20 in Madagascar (Bakker, 1974; Singh, 1995). Disease outbreaks have been reported mainly from irrigated and rainfed lowland

production systems. Also it does occur in both the highly susceptible exotic and indigenous varieties under rainfed upland production ecosystems (Awoderu, 1991b). It is gradually building up in other countries of Africa (Abo *et al.,* 1995; Anno-Nyako and Twumasi, 1995; Singh, 1995). Bakker (1974) stated that expression of the virus is strongly influenced by light intensity, day length, humidity, temperature, growth stage of the plant and other environmental factors. The area originally affected in Kenya by RYMV was part of a new irrigation project which had led to an increase in rice cultivation due to availability of water throughout the year. Similar circumstances have been associated with high incidence of RYMV in many other parts of Africa (Rossel *et al.,* 1982b). The increased use of fertilizer and irrigation have influenced plant growth in favour of pest population build up, survival and perennation of pathogens by leading to enhanced growth and greater continuity of weed hosts, rice stubble/ratoons, and seedlings volunteers (Abo and Sy, 1998).

Climatic factor such as rainfall, relative humidity, temperature and sunshine hours play important part in outbreaks of epidemic of virus diseases (Shaner, 1981). Many diseases are favoured by humid or rainy weather and may be more common and severe in years favouring good crop production (Moore, 1993). Similarly, the incidence and development of all the insect vectors are much dependent upon the prevailing environmental factors such as temperature, relative humidity and precipitation (Aheer *et al*., 1994). Most emerging infections appear to be caused by pathogens already present in the environment, brought out of obscurity or given a selective advantage by changing conditions and afforded an opportunity to infect new host populations (Moore, 1993).Variation in incidence and severity of RYMV depending on climatic zones and the local environment in which rice is grown (Awoderu, 1991a). Some rice cultivar planted in the rainy season suffers most from RYMV while rice cultivar grown mainly under irrigation is the most productive (Reckhaus and Andriamasintsebeno, 1997). Many and often contradictory results were generated most probably because of the use of different virus isolates and infection conditions. Awoderu (1991a) reported higher RYMV incidence in areas poorer in rainfalls and in areas with low relative humidity while Heinrichs *et al*. (1997) reported no correlations between RYMV and both windspeed and rainfall. Similarly Banwo (2001) reported no correlation between the weather factors and the vector population/disease incidence.

The comparative epidemiology of rice virus diseases showed that the *Oryza sativa* Japonica type varieties are very susceptible to rice viruses from Asia, Americas and the Caribbean while the *O. sativa* indica type varieties are either resistant or tolerant of the viruses (Chen, 1991). On the other hand the *O. sativa* indica type varieties are very susceptible to the Africa specific RYMV and the *O. sativa* Japonica types are either resistant or tolerant of the virus (Abo and Sy, 1998). For example IR8 and IR20, which are resistant to *Rice stripe virus (*RSTV*) Genus Tenuivirus* in Asia (Chiu *et al.,* 1986) are highly susceptible to RYMV in Africa and the neighbouring islands (Bakker, 1974; Thottappilly and Rossel, 1993; Reckhaus and Andriamasintsheheno, 1995).

A survey of incidence and severity of RYMV carried out in four rice growing environments and vegetation zones between 1983 and 1986 in West Africa showed that the incidence was 75% in tropical rainforest, while lowland rice environment had higher incidence than the upland environments (Awoderu, 1991a). Epidemics caused by virus on any varieties of crop plant in the field are influenced not only by the genetic background of host varieties, but also by initial disease intensity and weather (Luo and Zeng, 1995). Poor understanding of the epidemiology and transmission of the disease was reported to have increased the incidence and severity levels at an alarming rate from the late sixties to the nineties, predominantly affecting rice in the lowland ecosystems (Pinto, 2000).

Varietal reaction and yield loss assessment

Studies conducted at IITA on the reaction of hundreds of *O. glaberrima* Steud, has a higher level of resistance than *O. sativa* L. (Alam, 1988; Ng, 1988). Screening of *Oryza* germplasm to identify the potential sources of resistance to the virus that could be used in breeding programmes for resistance to the virus. These sources of resistance are mainly from *O. glaberrima, O. longistaminata,* and *O. barthi* as well as traditional African upland Japonica varieties that are highly tolerant, resistant or immune to the virus. Good progress has been made in identifying valuable sources of tolerant/resistant germplasm (Abo and Sy, 1998). So far immunity to RYMV was only reported in *O. barthii* and *O. longistaminata* accessions (IITA, 1984; Rossel and Thottappilly, 1985).

Screening for RYMV resistance which started in the late 70's at Rokupr, Sierra Leone and IITA, Ibadan, Nigeria indicated the following upland tropical japonica cultivars as resistant: 0S6, LAC 23, Moroberekan. Iguape, Cateto, 63-83, IRAT 13, NSO2 Gbongosy, Taminahum, marlie and many traditional farmer' landrace. Varietal reactions in field and greenhouse tests confirmed high level of susceptibility to RYMV in BG 90-2, Bouake 189, Jaya, IR 1529-680-3, IR5, IR29, ITA 212, Gambiaka, ITA 123, FARO 15, IR46, and ROK5 among others (Sy and Akator, 1993). Varieties found resistant to RYMV in West Africa include IRAT 133, IRAT 156, IR 47686-15051-1, WABIS18, FARO 300 and ITA 305 (Awoderu. 1991a). In East Africa, the resistant varieties include IR47686 -15–1–1, Gigante, IR 53734-27, FARO 300, TGR 78, ITA 305, IRAT 302, CT 7244-9-1-52, Supa SSD1 and Supa SSD5 (Luzi-Kihupi, 2001). Also reported to be resistant to RYMV are FARO 11, ITA 235, IRAT 133, FARO 399 and ITA 195) (Kanyeka *et al.,* 1996). In Nigeria, field experiment showed that BOUAKE 189, FARO 29, FARO 35, FARO 44, FARO 52, FARO 54 and GIGATE were moderately resistant (Salaudeen, 2007).

A study of yield reduction from inoculated and naturally infected rice plants showed a yield reduction rate of 84% for Bouaké 189, 67% for BG 90-2 and 4% for Moroberakan (Sy *et al.,* 1993). Yield losses of 58-67% were reported in Niger (Reckhaus and Adamou, 1986), of 54-97% of Sierra Leone (Taylor, 1989) of 20-45% in Burkina Faso (Sere, 1991). In Tanzania, the Yield loss estimates ranged from 25 – 100% (Luzi-Kihupi *et al.,* 2001). Yield losses of 17-100% and 6.59 – 79.11% were obtained under screenhouse conditions in Nigeria (Onwughalu *et al*., 2011; Alkali *et al*., 2016)). Under natural infection by RYMV disease, Bakker (1974) reported yield reduction of 50% in Kenya. While Taylor *et al.* (1990) reported 82% yield reduction in Sierra Leone. During a special monitoring tour of the WARDA constituted Rice Integrated Pest Management Task Force (IPM-TF) estimated yield losses on BG 90-2 to be as high as 60% under farmers' conditions in Niger (Sy, 1994).

Molecular characteristics of RYMV

RYMV belongs to the Sobemovirus group (Hull, 1988). It is a single stranded positive sense RNA and a distinctive spherical virus measuring about 28 $\pm$ 3 nm in diameter (Bakker, 1974; Rossel *et al.,* 1982b; Rossel and Thottappilly, 1985; Fauquet and Thouvenel, 1987; Opalka *et al.,* 1985). Sobemoviruses are characterized by being mechanically transmissible, except blueberry shoestring virus (BSSV), having relatively narrow host ranges, and having isometric particles that sediment between 110-1205 and form one band in $CSCl_2$ (Some form several bands in CS_2 SO_2), and having a single viral coat protein species of molecular weight of about 30,000 (30kb) (Hull, 1988). Most Sobemoviruses lack immunological

reactions with other Sobemoviruses (Thottappilly *et al.*, 1992). Most of them occur at relatively a high concentration in infected plant and the particles are found in the cytoplasm of most cells **(**Edwardson *et al.*, 1966; Hartman *et al.*, 1973; Bakker, 1974; Mohammed and Mossop, 1981; Hull, 1988) and in the nuclei (Mohammed and Mossop, 1981).

The percentage of nucleic acid is estimated to be about 18% RNA while the molecular weight of the coat protein is 28 $\pm$ 1 kd (Fauquet and Thouvenel, 1987). The percentage of nucleic acid in the infective virus particle is estimated to be 23.6 $\pm$ 0.5% (Bakker, 1974). The genome is a single stranded RNA (ssRNA) of molecular weight of about 1.4 x 10^6 daltons (about 4,500 nucleotides) and has a plus or messenger sense polarity (Hull, 1988). Furthermore, according to Brugidou *et al.* (1995), the genomic RNA consists of 4,550 nucleotides and is not polyadenylated. It also consists of 4 open reading frames (ORFs). ORFI (nt. 80 to 553) encodes a protein called P1 of 17.8 Kd and presumed to be the movement protein of the virus. ORF^2 (nt. 608 to 3607) encodes for a polyprotein of 110 Kd and contains a putative linked protein (VPg), helicase, protease and polymerase. ORF_3 (nt. 2092 to 2467) encodes a small protein of an unknown function. The ORF_4 (nt.3477 to 4116) codes for the coat protein (CP) of 26 Kd.

RYMV variability has been studied in several countries of Africa (Pinel *et al.*, 2000; Traore *et al.*, 2005; Alkali *et al*., 2015). Six major strains (S1 – S6) were found and phylogenetic separation was apparent among strains from East, Central and West Africa (Abubakar *et al.*, 2003). Considerable genetic divergence across a narrow geographical range occurred in East Africa, whereas shallow genetic differences were found across larger distances in west and central Africa (Traore *et al.*, 2005). Pinel *et al.* (2000) and Fargette *et al.* (2002) carried out molecular assays of coat protein gene sequences of 40 and 52 isolates of RYMV in Africa. In all, they differentiated five major strains, three (S1, S2 and S3) from Central and West Africa and two (S4 and S5) from East Africa, with a spatial overlap of strains within each of these two regions showing that geographical isolation alone cannot explain the genetic diversity of RYMV. Fargette *et al.* (2002) using four isolates showed similarities to the six from Madagascar and the only one from Kenya (S4). In a comparison of isolates from Central, East and West Africa and Madagascar, in the evaluation of some molecular characteristics of RYMV isolates occurring on rice in Tanzania, Banwo *et al.* (2002b) reported a completely new group/strain of RYMV referred to as S6. Pinel *et al.* (2000) and Fargette *et al.* (2002) reported that in general, variability was twofold and higher among the East African isolates. Fargette *et al.* (2002) reported that East African strains S4 and S5 were more distantly related to each other and to the Central/West African strains (S1 – S3) with an average nucleotide divergence of about 11%. Isolates collected from a few kilometres apart in the same country could belong to different strains as it was observed in both West and East Africa (Pinel *et al.*, 2000; Banwo *et al.*, 2002b). About 3 strains are now known to exist in both West Africa (S1 – S3) and East Africa (S4, S5 and S6) (Fargette *et al.*, 2002; Banwo *et al.*, 2002b; Alkali *et al.*, 2014b). Different serotypes (Sere *et al*., 2007; Alkali *et al*., 2012) and pathotypes (Ochola and Tuslime 2011b) of RYMV isolate are known to exist. Serological study using 20 RYMV isolates revealed two major Nigeria serogroup (NSg1 and NSg2) and four subgroup (NSg1a, NSg1b, NSg2a and NSg2b) in Southwest Nigeria (Onasanya *et al*., 2011). Similarly, RYMV serotypes SA and SB were found in Northern Nigeria (Salaudeen *et al.*, 2008b).

Management strategies

The prospects for reducing the threat of RYMV as increasingly serious disease of rice specific in most rice growing area of Africa have been investigated (Singh, 1995; Abo *et al*., 1998). Resistant cultivar, cultural practice, and application of insecticide have been widely used for RYMV control in practice (Kouassi *et al*., 2005; Sorho *et al*., 2005). Breeding for resistance is the most promising mechanism of limiting infection and yield losses caused by RYMV (Sorho *et al*., 2008; Wopereis *et al*., 2008; Zhang *et al*., 2009). During the past years, several improved lines obtained from the introgression of *rymv1-2* resistant gene from cultivar Gigante were developed by Biotechnology Unit of Africa Rice Centre, evaluated in the field for RYMV resistance (Absa, 2010). Control of the disease through the development of transgenic plants has been investigated and some transgenic lines with high levels of RYMV resistance have been obtained (Pinto *et al*., 2000). Some rare accessions of African cultivated (*O. sativa*, Gigante) and traditional rice species (*O. glaberrima series* Tog) were found to be highly resistant to RYMV (Ndjiondjop *et al*., 1999). In lowland ecosystems, Moroberekan variety, of Japonica type, appeared to be highly resistant whereas, WITA 9 and WAT 316 varieties, of Indica type, were resistant (Zouzou *et al*., 2008). In Nigeria, the rice cultivars FARO 11, 37, 38, 50, IDSA 10, 25, LAC 23, ITA 150, IRAT 144, Moroberekan and Gigante are resistant to the virus (Abo *et al.*, 2001). Resistance was also observed in Moroberekan and NERICA-L42 under both mechanical and natural viral infestation (Onwuaghalu *et al.*, 2011; Alkali *et al*.,2016), moderately resistance was found in Gigante, WAB 189 838HB, WAB 450 38HB and WAB 450 160HB under screenhouse conditions (Saluadeen *et al.*, 2008). In addition, genomic studies with combined transcriptome and proteome approaches were developed to identify plant genes and proteins involved in the virus cycle (Kouassi *et al*., 2005). Control by genetic engineering was applied to widely grown, RYMV-susceptible varieties; Bouake 189, ITA 212, BG90-2 have been transformed with construct encoding a fragment of the RNA-dependent RNA polymerase of RYMV (Kouassi *et al*., 2005). Deployment of resistant varieties as replacement of the known susceptible local varieties, based on cultivar resistance ranking, three *O. sativa* subspecies japonica (NERICA6, ITA257 and ITA325) and two *O. sativa* subspecies indica (WAC116 and WAC117) were identified as the potential sources of resistance for improving susceptible local varieties (Ochola and Tuslime, 2011a). The confirmed resistance breakdown in Gigante (Traore *et al*., 2006) has important implications for plant breeding and disease control strategies. Therefore, pyramiding different sources of resistance is recommended for enhanced durability of resistance. In addition, the use of sufficient pre-release challenge by the combination of isolates with different levels of aggressiveness will delay the rise of susceptibility in deployed resistant cultivars.

Several cultural practices have been recommended by WARDA's Integrated Pest Management Task Force (IPM-TF) involving International and National rice scientists to farmers in combating the disease in member countries affected by the virus. Namely, destruction of rice residues after harvest and ratoons that harbour the virus and insect vectors (Reckhaus and Adamous, 1986), diversification of varieties on a single plot, change of site for the next nursery beds, change the mode of sowing from wet to dry seeding (raising nurseries under rainfed conditions in lieu of irrigation), flooding of tilled plots until transplanting is carried out in order to limit the spread of the disease, early transplanting before the outbreak of *T. sericea* and reduction of spacing plants, rouging of infected plants and immediate replanting, early and double weeding to reduce the weed reservoir of the virus and insect vectors, withholding irrigation between planting to provide a rice free period and to restrict the build-up of the virus infection and insect population, and reduction of fertilizer application (e.g. urea) on infected

plants (Reckhaus and Adamous, 1986: Thresh, 1991; Coulibaly,1995; Reckhaus and Andriamasintsebeno, 1997; Abo *et al.*, 1998 ; Nwilene *et al.*, 2009b).

Different insecticides such as Malathion, Decis, Carbaryl, and a host of others have been used to control rice insect vectors population (Abo and Sy, 1998). Usually when the insect population is low, chemical control of the vector reduces virus disease incidence effectively, but when the population is very high the use of an insecticide may not give any control of the disease (Kissimoto, 1969). According to Breniere (1983) spreading a light layer of insecticides or petrol on the surface of water and thus causing insects to drop on it, as well as using granulated insecticides in water of the paddy against the larvae are effective strategies for the control of hispid beetles.

Conclusion and Recommendations

In view of the increasing incidence and importance of RYMV on rice production, more information on the epidemiology and dynamics of the virus spread and the roles of vectors found in and around rice fields under the prevailing weather condition is needed in each of the RYMV endemic or hotspot in the Africa continent. To provided sufficient and high quality epidemiological data, couple with technological advances in capacity and speed of computers, and availability and sophistication of computer models, will make it increasingly easy to incorporate and simulate weather factors to predict localized RYMV epidemics effectively.

More research on rice varieties with durable and broad-spectrum resistance against localized strains of the virus into the farmer preferred varieties that possess wonderful culinary traits is recommended for enhanced durability of resistance.

More information on the periods represents the most vulnerable phase in RYMV infection on different rice cultivars growth stages on yield losses is needed under different localities and ecologies. So that the stage at which plant developing tolerance to the pathogen is determined, thereby protection of the plant before that stage of growth against RYMV vectors in the fields would reduce the effect of the disease on yields.

More information on molecular characterization and strain differentiation of RYMV in different parts of Africa are need for identification of isolates/strains in pathogenicity and breeding studies of RYMV. However, for these to have a practical meaning for the rice grower, specific distinct pattern for each isolate must be related to the degree of virulence to rice cultivar in that particular ecology. Hence, coat protein (CP) genes of representative isolates of the different serotypes in each country should be sequenced to verify the serology-based conclusions, for routine test in comprehensive epidemiological studies.

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