Part I. General Introduction
Section 4. Importance of Plant Diseases on Rice Production in Africa

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1. Introduction

The history of rice cultivation in Africa is long and varied, probably dating back about 3,000 years. *Oryza glaberrima* is the most ancient rice species grown in Africa, while *Oryza sativa* was introduced about from 500 to 2,000 years ago (Harlan and Stemler 1976). The introduced *O. sativa* was quickly adopted in African rice production systems, overwhelming *O. glaberrima* production, because of its short cooking time (Williams 1978, Anonymous 2005) and the lower grain yield of *O. glaberrima* (Linares 2002). *O. sativa* cultivation in Africa was driven further by the development of irrigation schemes and the introduction, through INGER (International Network for Genetic Evaluation of Rice), of semidwarf high-yielding varieties from IRRI (International Rice Research Institute) and CIAT (Centro International de Agricultur Tropical) in the 1970s.

As rice became more and more important in many countries in sub-Saharan Africa (SSA), the governments developed rice research and development programs. West African countries created WARDA (West Africa Rice Development Association, today known as AfricaRice), an intergovernmental organization, to consolidate individual country's efforts with measures to improve rice-sector productivity, profitability, and sustainability. As WARDA continued to grow internationally, the need to extend its mandate to other SSA regions, including eastern, central, and northeast Africa, increased. In January 2003, the name Africa Rice Center was officially adopted in consideration of the aforementioned regions for inclusion (WARDA 2004).

The rice germplasm research program at WARDA started in 1978 after signing a collaborative agreement with IRRI, IITA, and IRAT. The initial activitities focussed on determining the status of rice germplasm, maintainance of a working collection at WARDA in West Africa, and receiving improved germplasm from IITA and IRAT.These activities culminated into the construction of the first gene bank in Fendall, Liberia, in 1979 to safeguard and maintained both local and introduced germplasm resources. As the need for adapted high-yielding germplasm increased, emphasis was placed on breeding activities, first conducted only by introducing, testing and releasing varieties from other continents. Later on, crosses were made, as introduced varieties faced new challenges mainly susceptibility to biotic and abiotic stresses. The success by AfricaRice in crossing *O. sativa* with *O. glaberrima* offered opportunity to improve the adaptability of high-yielding *O. sativa* varieties to African rice ecosystems (Jones et al 1997). These crosses prompted the development of New Rice for Africa (NERICA), which shifted the rice-production frontier from the traditional irrigated/rainfed lowlands to the uplands and underpinned rapid adoption and expansion of rice production in most sub-Saharan African countries.

Despite these research and development efforts, African rice production remained insufficient to meet demand and nearly 37% of the rice consumed in SSA was imported. Then a rice crisis occurred in 200–08 due to the rice stock reduction. Rice prices hit a record USD1,200/t, as traditional exporting countries in Asia closed their borders to increase their rice stocks.-Recovery from this crisis was relatively slow as most African countries had logistical, technical, and production limitations. By 2010, rice production gradually increased in most SSA countries. The high prices encouraged most governments to develop and implement rice development strategies to address rice shortages. However, the demand and preference for rice continuously grew outpacing production and the year 2011 saw the region's highest ever rice demand, estimated at a rate exceeding 6% per year—faster than any other food staple on the

continent. Concomitantly, mean per capita consumption is projected to increase further, from 44 to 53 kg, between 2011 and 2025 (Fofana et al 2014).

Despite the increase in demand, the overall rice production area has remained relatively modest and still lags behind demand. The average yield has also remained relatively low, averaging 2.1 t/ha, which is 49% below the world average (3.4 t/ha). This discrepancy is an indication that rice production in Africa is still poignant, and vulnerable to world production shocks and price instability.

The International Rice Research Institute (IRRI) and AfricaRice are the leading rice research centers mandated to conduct research on rice production improvement in Africa. In 2010, IRRI and Africa Rice engaged in a Global Rice Science Partnership (GRiSP), bringing together advanced research institutes, national agricultural research systems (NARES), international and regional fora, and development organizations to bring about a lasting and sustainable rice productivity increase in Africa and Asia. This partnership identified potential dynamics and opportunities for improving the rice sector in Africa. Already a great deal of improved varieties and crop management practices had been developed and there were promising prospects to increase rice production, either by increasing the area under rice cultivation or by intensifying rice production. However, underming the first achievements of this partnership have been a wide range of biotic and abiotic constraints that affect rice productivity, including frequent weather shocks in the form of drought and floods, salinity, iron toxicity, cold, weeds, and pests and diseases.

1.1. Rice diseases

Diseases that limit rice productivity in Africa have been identified and documented in West Africa through the IPM project on cereal crops funded by USAID in Sahelian countries in the 1980s (INSAH-UCTRPV-CTA 1987, INSAH 1992). Economically important diseases have been categorized as major ones, including rice blast (*Magnaporthe oryzae*), rice yellow mottle virus disease (RYMV), and bacterial blight (*Xanthomonas oryzae* pv. *oryzae)*. Secondary diseases include brown spot (*Bipolaris oryzae*), leaf scald (*Gerlachia oryzae*), sheath blight (*Rhizoctonia solani*), and bacterial leaf streak (BLS; *X. oryzae* pv. *oryzicola)*. Diseases of minor importance include false smut (*Ustilaginoides virens*), narrow brown spot (*Cercospora jansenea*), sheath rot (*Sarocladium oryzae*), bakanae disease (*Fusarium fujikuroi*), downy mildew (*Sclerophtora macrospora),* bacterial brown stripe (*Acidovorax avenae*), bacterial brown sheath rot (*Pseudomonas fuscovaginae*), and white gall (*Corallocytostroma oryzae)* (Séré et al 2013). The current impact of these diseases is largely variable and the magnitude of the damage depends on the weather patterns, cropping systems, production inputs, rice cultivars, ecology, and geographical location.

The importance and magnitude of disease damage have also been associated with climate change (Onaga et al 2016, Duku et al 2018). However, little work has been done to understand and model the effects of climate change on rice disease epidemics in Africa. Therefore, rigorous studies on the potential effect of climate change on pathogens, host plants, and their interaction, in relation to current and future weather simulations, are urgently needed.

Some rice diseases in Africa also show significant ecological dependence. Rice diseases commonly found in the uplands are of fungal origin, whereas several pathogens, including fungi, bacteria, and viruses are more prevalent in lowland rainfed and irrigated ecologies. A combination of pathogen attacks can also occur in rice production systems in Africa as observed in Rwanda (**AF Figure 1**) and in Tanzania (**AF Figure 2**). The economic impact of this combined infection exceeds the losses associated with a single pathogen depending on the level of interaction between the two or more pathogens and the host plant involved. This has been shown for RYMV and BLS (Tollenaere et al 2017), in which co-infection of the two pathogens was shown to increase the severity of BLS infection in rice.

AF Fig. 1. Mixed infection RYMV and BLS in Rwanda in 2009 (Photo: Y. Séré).

AF Fig. 2. Mixed infection of RYMV and blast in Tanzania, 2018 (Photo: G. Onaga).

Here, we discuss the importance of rice diseases in Africa in relation to the aforementioned factors and provide examples of recent developments in disease management.

2. Rice blast

2.1. History and economic importance in Africa

Rice blast is the most commonly found disease in the rice-growing areas of Africa. It is estimated that blast entered in Africa in the early 20th century, although the disease was probably present long before this. Small (1922) first described symptomatic infections in Uganda. Subsequent studies indicated that the symptoms of the disease began to emerge later in Ghana in 1923; Kenya in 1924; Congo in 1932; Egypt in 1935; Madagascar, Morocco, and Senegal in 1952; and South Africa in 1956 (Asuyama 1965).

Regional and national surveys conducted during the last 30 years of the 20th century documented gradual expansion of the disease in the areas affected and increasing epidemics in new areas (Séré et al 2011). By the 2000s, surveys further showed a shift in blast distribution and severity with most rice-producing areas experiencing multiple outbreaks, leading to yield losses ranging from 20 to 100% in several African countries, including Bukina Faso (36–63%), Nigeria (35–50%), Benin (20–30%), Togo (60%), Sierra Leone and Côte d'Ivoire (80%), Egypt (31%), Kenya (48%), Tanzania (40%), and up to 100% in Ghana, Gambia, and Uganda (Kihoro et al 2009, Séré et al 2013, MAAIF 2014, Hubert et al 2015). An unexpected serious outbreak of rice blast was also reported in Egypt (Sehly and Balal 1994).

Currently, blast epidemics are occasionally reported in rice-producing areas, depending on the occurrence of conditions suitable for the survival and growth of the pathogen, such as susceptible varieties, cloudy days with humidity ranging from 80 to 100% and temperatures ranging from 24 to 30°C. Higher incidence rates and relative frequencies of the disease have been commonly reported in association with upland ecologies, but field observations show that the disease is increasingly becoming important in lowland rainfed and irrigated ecologies mainly

when farmers try to improve their rice cultivation using high-yielding varieties from other continents and nitrogen fertilizers. A few studies have shown the occurrence of the disease, similar to what is observed in Asia with respect to cropping practices (Onaga and Asea 2016). The increasing replacement of the traditional rainfed rice–fallow system of producing one crop per year with intensive double cropping is expected to alter rice–rice blast pathosystems. Thus, an integrated disease management system needs to be devised and adopted to overcome imminent disease problems in the future. Presently, varietal resistance is the most economic and effective way of controlling rice blast, especially among African resource-poor farmers. However, pathogen variability makes the resistance unstable in the field. In the recent past, efforts were deployed to counter this through studies aimed at better understanding the blast population structure and variability.

2.2. Pathogen population structure in Africa

In the 1990s, rice scientists worldwide used the lineage exclusion method (Zeigler et al 1994) to build varieties with durable resistance. In four African countries, *Magnaporthe grisea* repetitive (MGR) sequence described by Hamer et al (1989) and an international set of differential varieties developed by Atkins et al (1967) were used to characterize blast pathogen population genetic diversity and pathogenic variability (Chipili et al 2001, Levy et al 1991) to identify possible lineages/groups. MGR586 fingerprints of more than 300 *M. grisea* isolates from Ghana, Nigeria, Burkina Faso, and Côte d'Ivoire, along with an international reference strain R indicated that blast lineages (genetic groups) varied from two to five per country. Nine distinct West African blast lineages designated WA1–WA9 were documented among the four countries studied. Despite a small number of MGR lineages, high pathotype diversity was observed; 16–25 pathotypes were found in each country.

Studies in other countries also detected multiple pathotypes of *M. oryzae* in rice-growing areas. In Oyo state in Nigeria, seven isolates collected from different cultivars belonged to the same lineage, NI-I, but expressed seven different pathotypes (Chipili et al 2001). A similar pattern was observed in Ghana, Burkina Faso, and Côte d'Ivoire . In another study, Séré et al (2007) classified 55 *M. oryzae* isolates from Burkina Faso into five major groups (*Mg-1* through *5*) using 108 RAPD markers to construct phylogenetic relationships. Some of the isolates originating from the same host plants and from the same localities were found to belong to different groups. Similar results were obtained by Xia et al (1993) who found four lineages from a single cultivar in two fields in a single region in the USA. Sampling for assessing blast pathogen genetic diversity should take such results into consideration in order to ensure that the individuals sampling do reflect the genetic diversity of the entire population.

In Germany, isolates from East Africa showed different severity scores when tested on rice monogenic lines and Co39, a susceptible indica variety from India (Onaga et al 2015). In another study on virulence, Odjo et al (2014) characterized the pathogenic diversity of 96 blast isolates from six West African countries using isogenic and monogenic lines from IRRI. The results showed a great diversity of the pathotype composition of the fungal pathogen populations and a differentiation according to the ecosystems, one being preponderant among upland rice isolates and another one distinguishing isolates from lowland and irrigated ecology. More recently, studies on the disease reaction patterns of isolates from East and West Africa revealed pathogenic variability separating *M. oryzae* isolates from the two regions (Mutiga et al 2017).

However, molecular analysis using calmodulin, actin, and ITS regions have shown minor differences between strains from major rice-growing areas in Tanzania (Chuwa et al 2014). Onaga et al (2015) observed a similar pattern in an AFLP study in which no evidence of population structure was observed in *M. oryzae* isolates from three East African countries, Tanzania, Uganda, and Rwanda. Phylogenetic trees constructed from single-nucleotide polymorphism (SNP) data between populations from East and West Africa showed clustering

largely by major geographic regions (Mutiga et al 2017). More recently, Odjo et al (2018) genotyped a set of 1,058 blast isolates (952 from 12 African countries and 106 from Madagascar) using 10 Simple Sequence Repeat markers and found six genetic clusters in West Africa, four in East Africa, and four in Madagascar, with a differentiation between subregions (East, West, and Madagascar) and also between countries. The authors also found that the two mating types were present in Africa, but no female fertile blast isolates were detected, suggeting the absence of sexual reproduction in Africa. This corroborates with the earlier reports on the potential existence of different mating types in East Africa (Onaga et al 2015).

Together, these studies provide evidence for the potential existance of several pathotypes in Africa and suggest the need for well-designed population-level studies to verify the evolutionary depth of such pathotype variability and to assess further the reproductive mode of the different lineages in the field.

2.3. Management of rice blast

Rice blast outbreaks in Africa are highly dependent on the cultivars grown, weather patterns, and the cropping systems. In recent years, it has been shown that the disease can attack nearly all plant parts, including the roots (Sesma and Osbourn 2004). If this is the case in all rice-growing areas, this could be possibly associated with increased overwintering to survive the intercrop periods. Mutiple hosts have also been suggested to act as platforms facilitating disease emergence via recombination/host shifts (Gladieux et al 2018). Under such conditions potential outbreaks may occur, and existence of several pathotypes could further complicate the management of the disease.

However, several methods can be used to prevent and manage rice blast. Considerable research has been conducted on chemical control and evidence of fungicide effectiveness is well documented (Séré et al 2011). However, the predominance of smallholder-farming systems in Africa economically favors the use of resistant varieties for blast management.

Systematic conventional breeding for blast resistance in Africa started in the 1990s and resistant varieties such as ITA 212, FARO 44, FARO 50, and Cisadane were first identified and released in Nigeria. Within the same period, WITA 1 and WITA 3 were released in Côte d'Ivoire. Subsequent studies conducted in West Africa on host-plant resistance led to the release of several other blast-resistant varieties, including NERICA 9, 12, 15, 16 and 18; ROK-16; FARO 11; IRAT 13; Moroberekan; and LAC 23 (Fomba and Taylor 1994). Similar studies in Egypt identified Sakha 101 and Sakha 103 as highly resistant varieties (Haggag and Tawfik 2014). For most of this resistant gemplasm, resistance genes were not known, except ITA 212, which habors the corresponding *R* gene in Tetep (Singh et al 2000).

By 1992, most of the resistant genotypes, including ITA 123, 212, 239; Moroberekan; Tox 3118-47-1-1-2-3; TCA80-4; WBA 56-50, 56-104; and FKR 33, were included in the breeding program by the first lowland rice-breeding task force in Africa. However, the durability of resistance for most of these varieties was short-lived and some never had a resistance track record after 1994. For instance, ITA 212 was found to be susceptible to rice blast in Nigeria in 1995 and 1996 (Singh et al 1997). Some varieties were location-specific in their resistance; FARO 8 and ITA 306 were resistant to rice blast in Côte d'Ivoire but susceptible in Nigeria. Such differential reactions were associated with the presence of nonexcluded compatible pathotypes, which required sufficient prerelease challenge with a representative pathogen collection from the region.

In the 1990s and 2000s, AfricaRice suggested the implementation of extensive screening methods through adequate characterization of potential screening sites in all countries to ensure an appropriate identification and deployment of resistant cultivars. A system of trapping, which consists of exposing varieties with known resistance genes to natural inoculum, was proposed and was deemed appropriate for determining the factors of virulence present within the pathogenic population, based on the reaction of the varieties, without

prejudging their association into distinct races (Séré et al 2004). Trapping nurseries at two screening sites in Burkina Faso were effective in characterizing the virulence spectrum of blast populations (Séré et al 2007). This study provided evidence for possible identification of the best sites for screening for durable resistance. The method was later extended to five West African countries (Benin, Burkina Faso, Guinea, Mali, and Nigeria) and three East African countries (Tanzania, Rwanda, and Uganda) using near-isogenic lines (NILs) and monogenic lines obtained from IRRI. Some of the hot spots identified in five West African countries, included Ikenna, Onne, Ibadan, and Kadawa in Nigeria; Mbo and Bokle in Cameroon; Adeta in Togo; Korhogo in Côte d'Ivoire; Karfiguela in Burkina Faso; and Rokupr in Sierra Leone. Later studies in East Africa identified Kyela and Dakawa as screening hot spots in Tanzania; Namulonge, Kibimba, and Olweny schemes in Uganda; and Nyagatara and Cyabayaga in Rwanda.

Screening studies on most of these hot spots identified *Pi9* as a broad-spectrum resistance gene. *Pi9* is presently durable in most African countries and would therefore be valuable in rice-improvement programs against blast. Some other genes such as *Pi33*, *Pita2*, *Pizt*, and *Pik-h* (**AF Table 1**) were found to be efficient in many West African countries and were included in the NARES breeding programs for possible pyramiding to provide durable resistance

AF Table 1. Summary of the status of *R*-gene deployment in Africa.

Region	Country	Effective resistance genes	Deployment status
West Africa	Benin	*Pi33, Pi9, Pis, Pit, Pita-2, Piz-5, Pizt, Pii, Pit, Pif*	-
	Burkina Faso	*Pi9, Pit, Piz-5*	-
	Guinea	*Pi9, Pit, Piz-5*	-
	Mali	*Pi9, Pit, Piz-5*	-
	Nigeria	*Pi9, Pit, Piz-5*	-
	Ghana	*Pi9, Pit, Piz-5*	-
	Côte d'Ivoire	*Pi9, Pit, Piz-5*	-
	Niger	*Pi9, Pit, Piz-5*	-
	Senegal	*Pi9, Pit, Piz-5*	-
	Gambia	*Pi9, Pit, Piz-5*	-
East Africa	Tanzania	*Pi9, Pita2, Piz5, Pi11 (t)*	Breeding work in progress at IRRI hub, Burundi
	Uganda	*Pi9, Pita2, Piz5, Piz-t, Pik^h*	Breeding work in progress at IRRI hub, Burundi
	Kenya	*Pi9, Pita2, Piz5, Piz-t, Pik^h*	Breeding work in progress at IRRI hub, Burundi
	Burundi	*Pi9, Pita2, Piz5, Pi40*	Breeding work in progress at IRRI hub, Burundi
	Rwanda	*Pi9, Pita2, Piz-t, Pik^h*	-
	Ethiopia	-	-
	South Sudan	-	-
Central Africa		-	-
Southern Africa	Mozambique	*Pi9, Pita2, Piz5*	Breeding work in progress at IRRI hub, Burundi
Northern Africa	Egypt	-	-

to blast. Some of the *Pi* genes recently discovered in Asia such as *Pi40*, which confers durable resistance to blast (Suh et al 2009), would also be of interest for breeding blast resistance in Africa, considering its effectiveness in Burundi and Mozambique.

Resistance harbored by *O. glaberrima* accessions and AfricaRice-bred NERICA varieties also hold promise for improving blast resistance in Africa. NERICA 15 has demostratred broad-spectrum resistance in Uganda and Kenya (Onaga et al 2016, Mutiga et al 2017). Experiments carried out in the field in Uganda between 2008 and 2014 showed a high level of resistance in NERICA 4; but this variety is presently susceptible to *M. oryzae* in most rice-growing areas of central Uganda, suggesting the need to revisit the durability of field resistance in NERICAs in response to this disease. This also calls for a need to analyze the genetic basis of resistance in the NERICAs that have been found to be resistant in several other studies (Singh et al 2000, Odjo et al 2014).

2.4. Genomic studies on rice blast resistance in Africa

There are presently more than 100 blast resistant genes and 347 QTLs that have been mapped and identified from diverse rice germplasm populations in the world (Wang et al 2012). Some of these R genes have been pyramided in released varieties in Asia (Prasad et al 2011, Singh et al 2013). In Africa, there has been very slow progress in R gene deployment at the farm level despite profound efforts in the development and release of rice varieties. Moreover, efforts to identify and characterize new sources of resistance are relatively inappreciable. A few studies on QTL mapping with populations derived from crosses between *O. glaberrima* and *O. sativa* were initiated in the 1990s at M'be, Bouake. However, this was disrupted by the 2000s civil war in Ivory Coast. Since then, genomic research on rice blast resistance is moving forward at a very slow pace, partly because the rice research programs in SSA turned their attention to more pressing problems, such as drought, iron toxicity and RYMV.

A study by Mgonja et al (2017), which identified 25 genomic regions associated with blast resistance using association mapping population originating from Africa, is the most recent associated with genomics in rice blast resistance in Africa. Considering the large number of pathotypes identified in Africa and the recent possible emergence of new races in Burundi that are capable of overcoming *Pita* and several other *R* genes (IRRI studies, unpubl.), there is a need to focus on gene stacking or pyramiding R genes that are effective in the region (**AF Table 1**), while identifying more genes/QTLs conferring durable resistance. In the future, gene editing could become a useful strategy for creating gene stacks for broad spectrum resistance and to stay one step ahead in the race against *M. oryzae* evolution. Moreover, continuous monitoring of pathogen populations for effector loss/gain and judicious understanding of *R* gene-effector specificity will enable practical predictions that potentially guide effective *R* gene deployment to be made.

3. Rice bacterial leaf blight (BLB)

3.1. History and economic importance in Africa

Bacterial blight (BLB) caused by *Xanthomonas oryzae pv. oryzae* (*Xoo*) was first reported in Africa in Mali in 1979 (Buddenhagen et al 1979). Before 1970, the literature is devoid of empirical evidence on BLB distribution, outbreaks, and yield losses in Africa. But from the 1980s, the disease appeared in most irrigated rice fields in Sahelian and Soudano-Sahelian countries, including Senegal, Cameroon, and Niger (Notteghem and Baudin 1981, Trinh 1980, Reckhaus 1983). By 1984, BLB had been discovered in rice-growing areas of Togo (John et al 1984). The disease subsequently occurred in Burkina Faso, Gabon, and Madagascar and in irrigated rice production areas of Nigeria, Gabon, Mauritania, Benin Cameroon, and Tanzania (Buddenhagen 1985, Jones et al 1991, Ashura et al 1999). Recurrence and severe outbreaks were noticed in Burkina Faso, Niger, Mali, and Togo between 2000 and 2012 with incidence ranging from 70 to 85% (Séré et al 2005, Banito et al 2012, Basso et al 2011). Gambia,

Mozambique, Rwanda, and Uganda are other countries where BLB incidence was reported (Onasanya et al 2009, El-Namaky 2011, Habarurema et al 2012, Jackson 2014). Recent field observations indicate that the disease is increasing in irrigated rice ecologies of the Igunga and Doho rice-growing schemes of Tanzania and Uganda, respectively. A study by AfricaRice indicated that Rwanda apparently has the most complex distribution of variation of BLB. The study further revealed that three pathotypes were present in two of the sites where rice lines were screened. However, the genetic identity of these pathotypes remains unclear.

BLB can cause yield losses of up to 80% in irrigated areas, depending on the rice variety, plant growth stage, geographic location, and season. In 1998, yield losses were reaching 70% in rice fields planted with the Chinese rice variety TCS10 in Bagre, Burkina Faso. Occasional complete crop failures were reported as well (Ouedraogo et al 2007, Wonni et al 2014). Yield losses of 20 to 80% have also occurred in several other West African countries, including Niger, Mali, and Nigeria (Onasanya et al 2009). In 2013, an estimated yield loss ranging from 19 to 63% was reported in Niger, which costed between USD 400 and 1,000/ha. Yield losses in Mali were well above USD 1,000/ha, especially whenever a combination of BLB and bacterial leaf streak occurred in the field (Jackson 2014).

Quantitative information on losses caused by BLB is relatively rare in East Africa. This is partly because BLB is less widespread in East Africa compared to West Africa and has been isolated rarely to establish its importance. BLB also shares many characteristics with other *X. oryzae* pathovars, including *X. oryzae pv. leersiae* and *Pantoea ananatis*, and obtaining a pure bacterial culture has been a hurdle in the region. In countries such as Uganda where most of these bacterial pathovars have been identified, inexperienced technicians may fail or take too long to identify the right colonies in plate cultures. Additional challenges associated with specific identification could be solved by full genome sequencing of all *X. oryzae* pathovars from the region to identify unique DNA signatures for PCR detection.

3.2. Pathogen population structure in Africa

The puzzle of BLB emergence and evolution in Africa is still ambiguous because of the unclear historical records about the origin and distribution of this disease. It is possible that, after introduction, BLB evolved independently in Africa following a significant geographical separation. In fact, studies by Gonzalez et al (2007) highlighted substantial differences between Asian and African Xoo, which further suggests a historically isolated pathogen population in Africa. A similar result was obtained by Bimerew (2010) using multilocus sequence analysis of *X. oryzae* pv. *oryzae* strains isolated from Africa based on *atpD*, *dnaK, gyrB*, and *efp* housekeeping genes. Pathotyping analysis carried out by the same group with 23 strains collected from West Africa and reference strains from Asia also identified two pathogroups, PI and PII, where the latter comprised the more virulent strains of the pathogen, which constituted 61% of the strains tested, including the West African ones. This study suggested that the West African Xoo are not a recent introduction and that geography and exposure to differential selection pressures are apparently the main drivers for divergence of African Xoo from the Asian Xoo.

Three races have been named in West Africa (A1, A2, and A3). These races do not represent any of the known *Xoo* races in Asia (Gonzalez et al 2007, Triplett et al 2011). Moreover, these races have been shown to harbor a reduced set of TAL effectors compared to the Asian ones (for a review see Verdier et al 2012) and that, despite being widespread among Asian isolates, the CRISPR locus is absent in African *Xoo* strains (Triplett et al 2011). Further more, allelic richness in African *Xoo* is far less compared to the Asian Xoo (Poulin et al 2014). This suggests further that African Xoo have a distant phylogenetic relationship with the Asian isolates.

BLB appears to be well established in West Africa and the populations are apparently more diversified compared to East Africa. Accordingly, it remains to be clarified on how East African

Xoo populations have evolved over time. In West Africa, major genetic differences between BLB populations have been reported. Whereas *Xoo* strains from Niger and Burkina Faso were found to be relatively close to each other, Xoo strains from Cameroon and Mali are more distinct. A dynamic population structure was also found in the Malian strains sampled in 2009, 2010, and 2012, which suggested a strong epidemiological structuring. In East Africa, the genetic structure and demographic history of this pathogen is yet to be understood. Population genetic studies on isolates from the East African region will probably detect undescribed evolutionary signatures somewhere in unsampled rice-growing areas that will probably help to gain better understanding of *Xoo* composition and evolutionary dynalics in Africa.

3.3. Management of BLB

BLB is prevalent in irrigated and rainfed lowland rice-growing areas of Africa and higher temperatures, especially in the dry savanna zone, tend to favor epidemics (Onasanya et al 2009). Several management options have been suggested, ranging from chemical control and cultural practices to varietal resistance. However, effective chemical control measures against *Xoo* are rare or lacking in Africa. Some of the chemicals that have been reported include bactericides, such as Kasugamycin, Phenazine, and Streptomycin, but these have not been attractive to farmers due to the costs involved.

Cultural practices and improvement in crop nutrition have naturally been practiced by farmers in the affected areas. For instance, in Niger, farmers have been practicing crop residue burning after harvest (AfricaRice 2010). Overwintering of the pathogen is reduced when the straw is burnt or plowed and buried in the soil. Weeding is recommended as some weed species, including *Brachiaria* sp., *Cyperus esculentus*, *C. rotondus*, *Dactyloctenium aegyptium*, *Echinocloa* sp., *Eulesine indica*, *Kyllinga squamulata*, *Leersia hexandra*, *Oryza barthii*, *O. longistaminata*, *Panicum lactum*, *P. repens,* and *Pennisetum pedicelatum*, have been reported to harbor the pathogen (AfricaRice 2010). Reduced or optimum nitrogen fertilizer use and optimal plant spacing are advocated as management strategies for BLB in Benin. Farmers also claim that potassium fertilizer increases resistance to BLB, but this has not been experimentally proven. Rotating rice with sweet potatoes appears to break the disease cycle in lowland rainfed ecologies of Uganda and this has gradually become a common diversification strategy for sustainable use of lowland rainfed areas in Uganda. Although these cultural measures have shown promise in rainfed lowland and upland areas, these approaches fail to provide complete control of the disease in irrigated areas. Exploiting genetic resistance present in both cultivated and wild rice relatives provides the best management option.

3.4. Breeding for BLB resistance

Breeding for disease resistance is the most relied-upon strategy to control BLB in Africa. Resistance to BLB is both monogenic or oligogenic and polygenic. Promising sources of resistance against BLB have been reported in the NERICA series. NERICA 1 was found to be resistant to *Xoo* isolates from Molodo, Nango, Ndebougou, and Niono in Mali. A similar study by Banito et al (2012) found that NERICA 14, 8, and 4 were moderately resistant to *Xoo* strains from the same region. From the work conducted by the Rwanda Agricultural Board, five cultivars, including NERICA 4, were found to be resistant to all *Xoo* isolates collected in the country. In a parallel study at Uyole Agricultural Research Institute in Tanzania, scientists identified two lines resistant to *Xoo*, although large variations across seasons and sites indicated precarious prospects. In Uganda, screening experiments at the National Crops Resources Research Institute (NaCRRI) found WITA 9 and NERICA 4 to be resistant. With the exception of NERICA 4, most of the other NERICA accessions have not been challenged with *Xoo* strains from East Africa. There is need to screen more NERICA accessions to identify more resitance sources. In West Africa, most accessions derived from *O. glaberima*, including the NERICAs, were found to be more resistant to A3 but susceptible to the A1 lineage (Djedatin et

al 2011). The high susceptibility of several NERICAs and other accessions derived from *O. glaberrima* to Race A1, despite being resistant to race A3, may probably limit the use of the NERICAs as parental lines in breeding against BLB in West Africa.

So far, 40 *R* genes and more than 17 QTLs conferring host resistance against various strains of *Xoo* have been identified (Djedatin et al 2016). In our recent analysis of East African germplasm using low density SNPs, the local accession Nawa Tule Na Bwana carries *xa5* and *xa4* resistance whereas about 38 East African accessions carry *xa4* in their backgrounds. Resistance QTLs *qABB-9*, *qBB-4*, *qBB-5*, and *qABB-10* have been reported to be effective against African *Xoo* strains, but the use of these QTLs in rice breeding in Africa is yet to be realized. Most *R* genes have also been found to provide resistance to African *Xoo* lineages, especially lineage A2 and A3. Recent pathogenicity studies on a differential set of rice lines have shown that Race A3 strains, which originate from Mali, are incompatible on 11 rice NILs (Gonzalez et al 2007). In particular, the resistant genes *xa5, Xa7, Xa14, Xa18, Xa21*, and their associations *Xa4+xa5, xa5+Xa21, xa13+Xa21* were found to be efficient to Malian R3 *Xoo* population. This suggests that Race A3 carries several effectors, which may contribute to virulence on the normal rice host plants. Race A1 strains are present in Niger, Burkina Faso, and Cameroon and are avirulent to only three *R* genes *Xa4*, *xa5*, and *Xa7*. Race A1 is therefore considered a serious threat to rice production and food securitv in Africa. However, *Xa7* and the four pyramided genes (*Xa4+xa5+xa13+Xa21*) as well as *Xa3, Xa10, Xa11, Xa4+xa5, Xa4+xa13,* and *xa5+xa13* have been shown to provide resistance or moderate resistance in Burkina Faso (AfricaRice 2010).

Race A2 from Burkina Faso is incompatible with *Xa3, Xa4, xa5, Xa7, Xa8,* and *Xa11* and intermediate on *Xa10, xa13, Xa14,* and *Xa21*. This might suggest that Race A1 could have originated from race A2 by acquiring virulence to *IR24, xa3, Xa10, Xa11, xa13,* and *Xa14*; and Race A2 from A3 by acquired virulence to IR24. This rate of evolution, although not quite substantial, is the probable cause and trend of *Xoo* evolution leading to epidemics. *Xoo* pathotype differences are frequently found wherever the disease is present in Africa, and is probably the most important factor contributing to significant epidemiological consequences on rice.

In a study conducted by Séré et al (2005), isolates from different regions in Mali reacted differently on four varieties, Bouaké 189, BG90-2, NERICA 1, and NERICA 4. A similar study by Onasanya et al (2009) found that *Xoo* isolates from seven West African countries formed two pathotypes, Pta and Ptb, with three and two sub-groups, respectively. In Niger, pathotyping experiments revealed three major pathogroups (AfricaRice, 2010). Although the genotypes used in these studies were few, these pathotype differences highlight the need for comprehensive studies to clearly delineate pathotypes. Additionally, Africa needs to conduct extensive sampling and analysis to better understand the genetic structure and dynamics of BLB populations in all areas where it has been reported but not characterized.

3.5. Mapping and genome-wide association studies (GWAS) on BLB resistance

Mapping and genome-wide association studies (GWAS) for discovery of BLB resistance trait loci is relatively new in Africa. A recent study at Université Polytechnique d'Abomey in Benin has shown that several QTLs encode for resistance to African *Xoo* strains. A cross between a susceptible tropical japonica landrace, Azucena, and a highly resistant indica cultivar, IR64, identified 12 putative QTLs induced by African *Xoo* strains (Djedatin et al 2016). The same authors have shown that two of the QTLs identified, qABB-7 and qABB-11, have large effects. qABB-11 is within a chromosomal region spanned by several BLB resistance genes (*Xa3/Xa26*, *Xa4, Xa4b, Xa6,* and *Xa9*) of which *Xa4* is the most likely candidate conferring BLB resistance in that region, although genetic interactions could also be possible. Several other QTLs that confer BLB resistance have been detected in Asia. African scientists could take advantage of such QTLs, by evaluating and adapting them to African breeding programs. Investigation into

the basis of resistance in *O. glaberrima* accessions that have shown resistance to *Xoo* would probably identify more genomic regions conferring resistance to Africa *Xoo*. By tapping into the recently discovered biotechnological tools such as genome editing, African scientists could also broaden the resistance spectrum of commercial high yielding varieties by stacking genomic regions/*R* genes conferring resistance to individual races.

4. Rice yellow mottle virus (RYMV) disease

4.1. History and economic importance in Africa

Rice yellow mottle virus (RYMV) is a monopartite positive sence RNA virus of 4,450 nucleotides encoding four open reading frames and belonging to the *Sobemovirus* genus. The virus is specific to the African continent where it has become increasingly important throughout the major rice-growing areas (Abo et al 1998, Kouassi et al 2005, Salaudeen et al 2008). RYMV was first confirmed on the rice cultivar “Sindano” at Otonglo in Kisumu, Kenya, in 1966 (Bakker 1970). However, a similar time-frame of RYMV diversification was estimated for both East and West Africa, which probably calls for further inquiry into the origin of this virus. Nonetheless, historical records show that RYMV was first detected in West Africa in 1975 (Fauquet and Thouvenel 1977). By 1981, RYMV was the most devastating rice disease in Burkina Faso and, in 1982, the disease was reported to cause yield losses of up to 92% on "Supa", the most popular rice variety in Tanzania (Rossel et al 1982, Banwo 2003).

Between 1980 and the late 2000s, the disease had spread to all rice-growing countries of West, Central, and East Africa, including Togo, Benin, Malawi, Uganda, and Rwanda (for reviews, see Kouassi et al 2005, Salaudeen et al 2008). Within the same period, the worst epidemics of RYMV occurred in Niger, Mali, and Madagascar (Reckhaus and Adamou 1986, Sy et al 1994, Reckhaus and Andriamasintseheno 1995). RYMV also re-emerged in the Mkindo irrigation scheme in Morogoro region of Tanzania in 1993 and, by 1995, epidemic proportions were noticed in Mbeya, Mwanza, and Shinyanga regions (Abubakar et al 2003). The negative impact of RYMV has since been felt also in Gambia, Ghana, Guinea, Guinea Bissau, Mauritania, and Senegal (Salaudeen et al 2008). Moreover, recent studies have shown severe outbreaks in Burundi, Zimbabwe, Ethiopia, Central African Republic, Democratic Republic of Congo, and Zanzibar (Abubakar et al 2003, Traore et al 2001, Hubert et al 2013).

The virus is a rapidly evolving type (Fargette et al 2008) and emergence and quick spread of resistance breaking isolates have been reported in countries Mali, Burkina Faso, Cameroon, Chad, and Togo (Konate et al 1997, Sorho et al 2005, Traoré et al 2006). RYMV is particularly devastating in lowland rainfed and irrigated ecologies of Africa (Abo et al 1998, Kouassi et al 2005), although it has also been reported to occur in the uplands (Awoderu 1991). In West Africa, the virus is well adapted to the Savanna and forest ecologies, whereas in East Africa, RYMV occurs mainly around lakes, mountaineous areas, and inland valleys (Abubakar et al 2003. Traoré et al 2005). Yield losses ranging from 20 to 100% have been reported across Africa.

Crop damage largely varies depending on the environment, rice genotypes, presence of vectors, stage of infection, and the strain involved. In Niger, yield losses ranging from 56 to 68% occurred when susceptible high-yielding Asian rice cultivars, IR1529 and BG90-2, were cultivated (Reckhaus and Adamou 1986). In Sierra Leone, losses of 82% were reported on varieties PN 623-3, TOX 516-12-SLR, and ROK 3 (Taylor et al 1990), and in Burkina Faso, losses of 84%, 79%, and 75% were recorded on varieties, FKR56N, FKR62N, and TS2, respectively (Traoré et al 2015). A survey in other West African countries showed yield losses of up to 70% in Mali and from 82 to 97% on varieties PN623-3, Tox 516-12-SLR, ROK3, ROK15, and IR65 in Liberia. In Nigeria, yield losses in excess of 90% were recorded on susceptible cultivars, Bouake 189 and FARO 29, following RYMV infection (Onwughalu et al 2010). In East Africa, disease severity and epidemics have caused yield reductions ranging from 5 to 100%

in Tanzania (Luzi-Kihupi et al 2000), and in Uganda, farmers estimated yield losses ranging from 30 to 80%.

4.2. Pathogen population structure and distribution in Africa

The genetic structure of RYMV populations in Africa is interestingly diverse, well-structured, and associated with geographical distance where closely related isolates are rarely found geogoraphically far apart. This relatively high level of relatedness between isolates from the same geographic region indicates limited long-range dispersal. There are, however, reports where isolates from the same area are quite different from each other and close to isolates from a remote country. For instance, serological studies have shown that isolates from Mwanza and Shinyanga regions in Tanzania were found similar to the isolates from Madagascar and Mali (Ali 1999), which suggests possible occurrence of transmission and probably intra-convergence, although this is rarely reported in RYMV studies.

Both immunological and molecular sequencing studies split RYMV into three lineages (Pinel-Galzi et al 2015) with six major strains currently distinguished from these lineages: three in East Africa (S4, S5, and S6) and three (S1, S2, and S3) in West and Central Africa (Pinel et al 2000, Fargette et al 2004, Traoré et al 2005). S5 lineage is confined to Kilombero, a region along the Indian Ocean at the latitude of the Zanzibar Archipelago, while S6 lineage is reported to be more widespread in East Africa, but more distributed on the coastal zone alongside the Indian Ocean (Pinel-Galzi et al 2015). A recent split of S6 lineage into S6c and S6w has been reported in Tanzania (Hubert et al 2017). The S4-S1 lineage is widespread all over Africa. Interestingly, all the three lineages are present in Kilombero region (Traoré et al 2005), and it is suggested that this region could be the center of origin of RYMV following a recent reconstruction of its phylogeographic history.

The highest diversity of RYMV is indeed found in Eastern Tanzania and decreases towards West Africa. Along the route towards the west, RYMV is said to have spread in the direction of Kenya and Uganda (Trovão et al 2015). RYMV has also been reported in Burundi, Rwanda, and Democratic Republic of Congo (DRC) (Ndikumana et al 2011, 2012, 2015; Hubert et al 2013; Trovão et al 2015). The S4lv strains appear to have spread from the East Africa mainland through Burundi, Rwanda, and the DRC towards the west. Several variants of S4, such as S4lv, are dominant in Kenya, Uganda, Lake Victoria zone of Tanzania and Kigoma, and near Lake Tanganyika (Hubert et al 2017). S4mg is the dorminant variant in Madagascar (Rakotomalala et al 2008). Recent emergence of another strain, S4ug, which is apparently similar to S4mg, has been reported in Uganda, Tanzania, and Kenya (Ochola et al 2015, Hubert et al 2017, Adego et al 2017).

S4ug appears to be displacing S4lv in East Africa, and is more abundant than previously suspected, considering the timeframe it has taken to emerge widely (three countries in three years from the time of its first detection in Uganda). Recombination events between S4lv and S4ug have also been reported recently in Kenya and two strains, Ke101 and Ke105, recognized as emerging types, have been detected (Adego et al 2017). A similar pattern of recombination, between isolates of closely related strains, was previously reported in eastern Tanzania (Pinel-Galzi et al 2009, Rakotomalala et al 2008). As such, S4 variants appear to readily recombine and regular surveillance is required to prevent the occurrence of unexpected epidemics.

In Malawi, where S4lm strain has been dorminant along Lake Tanganyika, emergence of a new strain, Mw10, has been reported (Ndikumana et al 2015, 2017). This strain, although closely similar to S6, is the latest to be identified and differs from the traditional S4lm strains by a nucleotide sequence dissimilarity of about 10% and has been tentatively named S7. Identification of this strain reopens avenues for investigating the genetic factors responsible for RYMV emergence and for revisiting its phylogeography.

In West Africa, profound diversity of RYMV is found in the Inner Niger Delta (Traoré et al 2005), where domestication of *O. glaberrima* is said to have occurred ca. 3,000 years ago

(Porteres 1976). The most recent findings, however, indicate that the West Africa lineage (S1-S3) existed relatively early, dating back to 1840 and was estimated to have originated from Côte d'Ivoire. Diffusion appears to have started within and between Côte d'Ivoire and Mali where S1-S3 strains are diverse and predominant and from where the virus spread to the west, east, and the south. With the exception of Gambia where strains are closely related to the basal lineage found in the Inner Niger Delta in Mali (Séré et al 2008a), westward expansion includes the spread to Sierra Leone and Guinea, where S3 and S2 are abundant (N'Guessan et al 2000), Eastward expansion includes Burkina Faso, Nigeria, Ghana, Chad, and Central African Republic where S1 strains are predominantly present (Trovão et al 2015). Both inter- and intra-region transmissions of RYMV have been attributed to river flow. For instance, the Benue and Niger Rivers apparently facilitated the east-west dissemination of RYMV (Fargette et al 2006).

A similar pattern is predicted for the south-north dispersal through the Chari and Logone rivers in Central Africa, and the westward spread through the Senegal and Gambia Rivers. However, an attempt to link the West and East African populations is constrained by limited sampling in the east-central-west African continuum. Previous studies predict East to West transmission to be in a wave-like pattern proposed for some human and plant viruses, in which vector transmission is punctuated by severe bottlenecks that allows only a small subset of the virus diversity to be transferred from one region to another (Traoré et al 2009). Minimal diversity in the West African region compared to East Africa (Pinel-Galzi et al 2015) is apparently consistent with this. The restricted RYMV genetic diversity in West Africa is attributed to the Dahomey-Gap (Sorho 2006), which probably constrained RYMV diversification and distribution. This was also recently supported by the work of Trovão et al (2015), in which the presence of a long-distance resistant landscape was predicted to limit viral dispersal.

A geographic split of RYMV strains in West Africa in which the Sa serotype is predominantly found in the Inner Niger Delta, S1-wa and S2 in the rest of the west (rice belt), and S1-ca in the West to Central African continuum towards the eastern part of West Africa (yam belt), is congruent with this prediction. Thus, in the event that such a model holds, it is most probable that RYMV entered West Africa through Burundi, Rwanda, or Uganda to the east of the Democratic Republic of Congo where S4 isolates could have undergone a severe bottleneck or *de novo* mutations predating population separation.

4.3. Epidemiology and Management of RYMV

The epidemiology of RYMV is influenced by several factors making it complicated to deploy control strategies across Africa. Epidemics are influenced by the strains involved, vectors present in the area, alternative hosts, cropping practices, and environmental conditions. Among the strains identified so far, S5 strains are very damaging in East Africa (**AF Figure 3**), whereas S2 strains are more aggressive in West Africa.

Analysis of the genome-scale evolution of RYMV indicates that changes in virulence involve recombination in the ORF2a genome-linked viral protein (VPg) domain (Traoré et al 2010). Polymorphism at codon 49 of the VPg protein involves either a glutamic acid residue or a threonine residue (E/T polymorphism) and is a major determinant of virulence (Poulicard et al 2012). Moreover, T/E polymorphism is associated with interspecies adaptation with potential negative consequences on plant resistance (Pinel-Galzi et al 2016). For instance, virus isolates with a “T” residue are able to adapt to *O. glaberrima* background. Mutations at codons 41 and 52 are particularly the cause of resistance break down in *O. glaberrima* accessions carrying rymv1-3 allele, while isolates with a mutation at codon 48 are able to adapt to *O. sativa* accessions carrying rymv1-2. Recent identification of a glutamine instead of the usual T/E in S7 isolate identified in Malawi (Ndikumana et al 2017) indicates possible presence of an alternative genetic route to virulence. Moreover, a hypervirulent pathotype, named T', with

AF Fig. 3. Entire rice field infected with RYMV in Tanzania in 2018 (*Photo: G. Onaga*).

altered central part of the VPg as a result of convergent nonsynonymous substitution, has been recently identified in West-Central Africa (Hébrard et al 2018). Other virulence proteins, such as the sobemovirus P1 protein, that was recently reported as a suppressor of posttranscriptional gene silencing, have been demonstrated to be required for systemic virus spread in rice plants (Nummert et al 2017). Thus, apart from T/E selective fitness advantage, other viral proteins potentially determine RYMV virulence.

Once established in the field, RYMV's ability to successfully infect the rice host on a large scale depends on the effectiveness of transmission. About 12 insect species are known vectors of RYMV. These insect vectors may vary from country to country or even between localities. For instance, insect transmission experiments performed in Uganda indicate that *Coryphosima centralis* appears to be the main vector of RYMV in the rice fields (Uke et al 2014). Vector transmission also occurs through chrysomelid beetles such as *Chaetocnema abyssinica*, *C. pulla*, and *Trichispa sericea* and grasshoppers, *Conocephalus merumontus, C. longipennis, and Oxya* spp., which accelerate secondary spread in most localities. Some of them (*Oxya hyla*, *Cnootriba similes*, and *Locris rubra*) have been used successfully to screen varieties for resistance to RYMV (Séré et al 2008b).

On the other hand, transmission through contaminated soil has been reported (Traoré et al 2008) and is believed to occur through fields that previously contained RYMV-infected crops. In such fields, rice debris, roots, and ratoons infected with RYMV are the main soil contaminants, which serve as sources of primary infection in subsequent years. Persistent perennial gramininaceous hosts may also serve as virus reservoirs and virus adaptation to these perrenial hosts is facilitated by similar levels of susceptibility and viral mutation required to adapt to more than one host. Some studies suggest that RYMV is contact-transmitted by cows, donkeys, rats, man, wind and water (Traoré et al 2009). Cow- and rat-transmissions occur through feeding. Human transmission mainly occurs through handling and transplanting of seedlings and weeding under RYMV-contaminated irrigation water. The flow of irrigation water from infected fields could be the cause of contamination of both within and distant rice fields and infection of alternative hosts at the boundary of irrigated rice fields or rivers sustains the virus cycle. Wind transmission occurs through sand grains and guttation fluid abrasives.

Influence of environmental factors on epidemics mainly depends on the agroecosystem involved. Most epidemics occur in areas where irrigated rice is grown, especially in areas with continuous flooding. Such conditions favor establishment and persistence of insect vectors and alternative host plants, and facilitate the spread of the virus. Besides agroecosystem influence, cropping practices may cause viral emergence through ecological perturbation, including a

change in land-use associated with rice production intensification practices, germplasm characteristics, and husbandry practices.

Several management options have been suggested for limiting yield losses due to RYMV, several reviews have been published (Kouassi et al 2005, Traoré et al 2009, Pinel-Galzi et al 2015). Cultural practices such as early or late planting in order to avoid the peak vector population and removal or roguing and burning of diseased plants and weeds are recommended to limit disease incidence. Removal of diseased plants and volunteers can be combined with a postharvest weeding or plowing under the infected volunteer rice, crop residues, and alternate hosts to limit off-season virus inoculum. This has been demonstrated to be effective in the Bongor region of Chad; Molodo, N'Débougou, and Niono in Mali; and Southern Guinea (Traoré et al 2009). Rotation of rice with sweet potatoes or vegetables to break the life cycle of vectors is being practiced by Ugandan farmers. Insecticide application has been recommended for vector control (Reckhaus and Andriamasintseheno 1995).

4.4. Host plant resistance to RYMV

Management of RYMV by host plant resistance is an effective and economical strategy to limit rice yield losses. However, host plant resistance is constrained by the narrow resistance genetic base, fertility barriers, and recessive and/or polygenic resistance in most donor accessions. Earlier studies on genetics of resistance have identified high resistance characterized by the absence of symptoms in the indica varieties Gigante and Bekarosaka (Ndjiondjop et al 1999, Rakotomalala et al 2008). This resistance is controlled by a recessive gene, *rymv1-2*. Progress in transferring *rymv1-2* to commercial high-yielding rice cultivars has been very slow due to the aforementioned factors. Additional resistance alleles, rymv1-3, rymv1-4 (Albar et al 2006), and rymv1-5 (Thiémélé et al 2010), have been identified on the same locus, RYMV1, mapped on chromosome 4, in *O. glaberrima* accessions Tog5681, Tog5438, and Tog5674, respectively.

By studying more than 300 *O. glaberrima* accessions from AfricaRice and IRRI, Thiémélé et al (2010) found 26 accessions with RYMV resistant genes. Some of these accessions carry more than one resistance gene, for instance, Tog5672 habors rymv1-4 and another resistance locus, RYMV2. RYMV2 is also carried by Tog7291. Therefore, *O. glaberrima* represents a valuable source of RYMV resistance genes. Presently, it is known that RYMV resistance is controlled by three genes, distributed on three loci: RYMV1, RYMV2, and RYMV3. The genes on RYMV1 and RYMV2 encode a translation initiation factor eIF(iso)4G1 (Albar et al 2006) and a transmembrane nucleoporin ortholog (CPR5; Gu et al 2016), respectively.

The resistance conferred by *rymv1-2* and *rymv1-4* is represented by a single mutations due to substitution of glutamic acid residue (E) by lysine residue (K) at amino-acid positions E309K and E321K of eIF(iso)4G, respectively, while *rymv1-3* is due to a tripeptide deletion at positions 322-324. Resitance conferred by *rymv1-5* is associated with a tripeptide deletion at positions 313-315 (mutation named K312N). RYMV2 confers resistance by expressing its null allele, characterized by a 1-base deletion leading to a truncation in *CPR5* (Orjuela et al 2013). The resistance gene at the RYMV3 locus is carried by both Tog5307 and Tog5672 and encodes a dorminant CC-NBS-LRR domain-containing protein (Pidon et al 2017). RYMV3 mediates resistance in both a codominant and incomplete penetrant manner. Two candidate genes, *ORGLA11g0175700* and *ORGLA11g0175800*, are present within the fine-mapped genomic region spanned by RYMV3. However, their functional contribution to RYMV resistance mediated by RYMV3 still needs verification.

The three resistance genes (*rymv1*, *rymv2*, and *rymv3*) interact differently with RYMV. *Rymv1* and *2* interact with the central domain of the VPg and the membrane anchor domain of polyprotein P2a (Pinel-Galzi et al 2016), respectively, while *rymv3* interacts, through its central LRR domain, with VPg. The dominant gene *rymv3* easily succumbs to T49 isolates, while *rymv1-2* is resistant to T49 isolates (Pinel-Galzi et al 2016). Tog5681 and Tog7291 are both resistant to E49 isolates. E49 isolates are prevalent in East Africa, while both E49 and T49 are

present in Central and West Africa. Thus, *rymv1-3*- and *rymv2*-mediated resistance is probably more effective in East Africa, while *rymv3* apparently confers resistance against most West African isolates, owing to the presence of an alternative polymorphism (T49 Cia) at amino acid 49, which aids *rymv3*-mediated resistance against T49 isolates. This evolutionary shift is apparently a selective disadvantage on RYMV. However, given the frequent nonsynonymous mutations within this region, it is too soon to conclude that *rymv3*-mediated host resistance against T49 isolates will last.

As mentioned earlier, a recent shift from the classical E/T to a glutamine in S7 isolate identified in Malawi (Ndikumana et al 2017) indicates that development of alternative genetic routes to virulence is possible in RYMV. Although the frequency of this mutation and its survival and distribution are not yet known, it is a signal of continuing evolutionary "arms race" between RYMV and rice. Therefore, other resistance genes that contribute to durable resistances are needed. A number of genotypes are being screened by IRRI scientists in Kenya and Uganda to select those that can tolerate and resist the pathogen. Preliminary results show that Tog lines and wild accessions are promising, and may be included in future RYMV resistance breeding programs. There have also been some reports of effective partial resistance, and resistance obtained through genetic transformation (Sorho et al 2005). Partial resistance, characterized by a delay in virus accumulation and in symptom development, has been associated with several QTLs in japonica cultivar (cv.) Azucena and upland NERICA varieties (Albar et al 1998, Boisnard et al 2007). Combining such resistance with other sources currently present in rice would probably provide durable resistance. A number of japonica cultivars with polygenic resistance QTLs on chromosomes 7 and 12 were also previously identified (Albar et al 1998, Pressoir et al 1998).

A high degree of sequence conservation in RYMV *ORF2* also provides opportunities for developing effective transgenic resistance across the spectrum of strains (Pinto et al 1999). Some level of success was reported by the same authors, and lines with a partial RYMV resistance were developed. Durable resistance can also be developed through recombinant DNA techniques such as the clustered regularly interspaced short palindromic repeat (CRISPR)/CRISPR-associated protein 9 (Cas9) systems. The practical use of this technique in improving RYMV resistance is yet to be explored. The starting step requires identification and validation of rice proteins that directly interact with viral RNA, followed by identification/validation of protein interaction sites to provide possible genetic modification targets in the plant genome which can be altered using CRISPR technique. In addition, efficient transformation protocols and a critical mass of well trained scientists and technicians are required in Africa to efficiently exploit CRISPR techniques.

5. Emerging and minor diseases affecting rice production in Africa

5.1. Bacterial leaf streak (BLS)

Among the diseases classified as minor, bacterial leaf streak (BLS) caused by *Xanthomonas oryzae* pv. *oryzicola* (*Xoc*), a once-overlooked foliar pathogen, is causing substantial yield losses in Asia and Africa. In Africa, BLS was first identified in Madagascar, Nigeria, and Senegal in the 1980s (Diop 1980, Nino-Liu et al 2006). After two decades, the disease was identified in Mali (Gonzalez et al 2007) and Burkina Faso (Wonni et al 2011). In 2014, BLS re-emerged in the Antananarivo and Antsirabe districts of Madagascar, where it was considered a threat to rice production (Poulin et al 2014). Since then, repeated outbreaks have continued to occur and the disease has spread to several rice-growing countries of West and East Africa, including Burundi, Uganda and Kenya (Afolabi et al 2014a,b; Onaga et al 2018). Field observations indicate that BLS is also present in Tanzania and Rwanda (**AF Figure 1**) and appears to be spreading in West Africa to Ghana, Benin, Togo, Côte d'Ivoire Coast, Sierra Leone, Niger, Senegal, Gambia, and Guinea although the prevalence and severity there are most of the time light.

A few studies on pathotyping have shown virulence variability between African *Xoc* strains from Burkina Faso (Wonni et al 2014). Virulence studies in other parts of Africa where the pathogen has been detected are missing. Therefore, studies are needed to better understand the virulence structure and divergence of this pathogen in the affected areas.

The precise ancestry of BLS in Africa is still quite controversial, but high genetic relatedness with Asian strains (Gonzalez et al 2007) points to Asian origin. Transcription activator like (TAL) effectors and TAL effector-binding sites are highly conserved between African and Asian Xoc (Wilkins et al 2015). This high level of conservation provides supporting evidence for Asian ancestry. Phylogenetic seperation between African and Asian strains could be associated with differences or recombination acting in other regions of the genome. The first genetic evidence to infer seperation was shown by Hajri et al (2012) in a multilocus sequence analysis and type-III effector repertoire mining study. Subsequent research has supported this split (Wonni et al 2014). This geographic separation is also mirrored in a few genes, e.g., *Tal3c* and some pseudogenes, that are absent in African *Xoc* but present in Asian *Xoc* and suggests that the exceptions observed in TAL effectors and TAL effector-binding sites is a result of their preferential retention for pathogen adaptation and survival.

Quantitative yield losses caused by BLS are extremely rare in Africa, despite the occurrence of severe infections in rice fields. Isolated cases of yield losses range from 10 to 32% in Burkina Faso (Wonni et al 2014). The spread of BLS in Africa is attributed to germplasm exchange across borders, and this has probably contributed to its rapid increase from 2010 to the present. Within a given locality, seed, especially home saved rice seeds harvested from infected fields, is the most important source of primary inoculum. The pathogen can also multiply and persist on perennial weeds, grasses, infested debris and wild rice (Wonni et al 2014) and spread through irrigation water. Insects and birds may occasionally transmit the disease, especially when bacterial ooze on leaf surfaces stick to their bodies.

Even though resistant cultivars had been identified as early as 2000, little is known about novel host resistance to *Xoc* in Africa. Currently, two putative resistance genes, *a* heterologous Xa27 and *Rxo1,* from maize and *bls1* from DP3, a rice line derived from *Oryza rufipogon* are single major genes that have been identified as effective against African *Xoc* strains (Zhao et al 2004, He et al 2012). Some highly resistant rice varieties have also been identified in Burkina Faso, such as FKR14, 19, 28, and FKR43; upland NERICA 12,13, and 17; and lowland NERICA-L-19 (Wonni et al 2014), but the genetic basis of their resistance is yet to be determined. Introduced varieties such as Zenith, Tetep, IR20, DZ60, Jagannath, H4 BJ1, Co4, and Krishna show field resistance and could provide durable resistance sources against African *Xoc* strains, but these have not been widely tested.

A number of QTLs, including major QTLs, qBLSR-11-1 from a resistant variety, Dular, and qBlsr5a from Acc8558, also confer resistance to BLS. Recently, more than 12 QTLs conferring BLS resistance were identified in rice (Xie et al 2014). However, the effectiveness of these QTLs is yet to be tested against more diverse strains. A major susceptibility (S) BLS TAL-effector target gene, a sulfate transporter (*OsSULTR3;6*), has also been recently identified (Cernadas et al 2014). A better understanding of *OsSULTR3;6* and identification of other susceptibility (S) genes could potentially provide targets for building resistance in susceptible varieties through genome editing.

5.2. Other rice diseases and their importance in Africa

There are relatively few reports and little research regarding other rice diseases in Africa. Some of the most commonly encountered diseases include brown spot (*Bipolaris oryzae*), leaf scald (*Gerlachia oryzae*), sheath blight (*Rhizoctonia solani*), false smut (*Ustilaginoides virens*), narrow brown spot (*Cercospora jansenea*), sheath rot (*Sarocladium oryzae*), bakanae disease (*Fusarium fujikuroi*), downy mildew (*Sclerophtora macrospora*), bacterial brown stripe

(*Acidovorax avenae*), bacterial brown sheath rot (*Pseudomonas fuscovaginae*), bacterial panicle blight (*Burkholderia glumae),* and white gall (*Corallocytostroma oryzae)* (Séré et al 2013). Field visits by AfricaRice in Uganda, Benin, and Burkina Faso indicated frequent occurrence of narrow brown spot due to *Cercospora jansenea* (**AF Figure 4**) as well as sheath rot (**AF Figure 5**), sheath spot caused by *Rhizoctonia solani* (**AF Figure 6**), and bacterial brown stripe caused by *Acidovorax avenae* (**AF Figure 7**).

AF Fig. 4. Heavy infection of narrow brown leaf spot (*Cercospora oryzae*) at Dévé in Benin in 2006.

AF Fig. 5. Lesions of Sheath rot at Mwea, Kenya *(Photo: G. Onaga).*

AF Fig. 6. *Rhizoctonia* sheath blight in Benin in 2009 (Photo: Y. Séré).

AF Fig. 7. *Acidovorax avenae* lesions at NaCRRI, Namulonge, Uganda 2015 (Photo: G. Onaga).

These diseases are sporadic and are probably under-diagnosed or under-reported in comparison to the aforementioned major diseases. Brown spot and narrow brown spot are often encountered in areas of low soil fertility and high humidity. Brown spot occurs in widespread in Africa and has been reported in Nigeria, Ghana, Cameroon, and Sahelian countries mainly Burkina Faso, Tanzania, and Uganda (Aluko et al 1975, Notteghem and Baudin 1981, Jones et al 1993, Nutsugah et al 2003, Kawube et al 2005, Ouedraogo 2008, Mwalyego et al 2011). The disease frequently occurs in upland ecologies and occasionally in lowlands. Successful infections often occur under drought stress and still-air environments, which facilitate retention of dew or water droplets on the leaves. Yield losses ranging from 12 to 43% have been reported in Tanzania (Mwalyego et al 2011). In Uganda and Egypt, brown spot is ranked the second most important fungal disease of rice after rice blast (Odogola 2006, Shabana et al 2008). Investigations conducted in Burkina Faso have shown significant yield losses due to brown spot (Ouedraogo et al 2005). Genetic evidence for high molecular and pathogenic diversity has also been reported (Ouedraogo 2008).

Most of the rice cultivars grown commercially are susceptible to brown spot. Resistance screening experiments have however identified some accessions with high levels of polygenic resistance, including Tadukan, Tetep, and CH45 (Sato et al 2008). Field screening of East African rice accessions at NaCRRI, Uganda, identified IR64, IR 79511, Namche 3, and TXD 306 with high levels of resistance (G. Onaga, unpubl.). No major genes have been shown to confer immunity to this pathogen (Sato et al 2008). Recently, Matsumoto et al (2017) identified a number of QTLs, including *qBS2*, *qBS9*, and *qBS11* for *B. oryzae* resistance in a cross between Tadukan (indica) and Hinohikari (japonica). Additional QTLs from a cross between CH45 and Koshishikari identified *qBSR11-kc*, a major QTL, which explained 23.0–25.9% of resistance to *B. oryzae*. Moreover, a quantitative and polygenic resistance was described in some varieties such as FKR14 grown in Burkina Faso. However, none of these QTLs have been deployed or

validated, despite the widespread occurrence of *B. oryzae*. There is need to undertake validation of these QTLs to support breeding work.

Narrow brown spot is rarely reported in Africa. Where present, the disease often appears late in the rice season and the severity varies from year to year. Disease symptoms on susceptible varieties are often confused with brown spot. Severely attacked rice plants often ripen prematurely and have reduced milling quality. The sporadic occurrence of brown spot and narrow brown spot makes their importance to be largely variable in Africa. If overlooked, there is a possibility of substantial yield losses, considering the recent severe damage observed in Uganda, Benin, and Burkina Faso (Y. Séré, unpubl.).

Leaf scald and sheath blight have been reported in the humid tropics of West Africa on both upland and lowland rice fields. Both diseases, although not adequately reported, appear to be present in East Africa. Cool wet weather and high nitrogen fertilization often favor their development. Although the precise extent of damage is not known, most pathologists in Africa regard these diseases as potentially destructive, especially when conditions are favorable for their growth and survival.

Pseudomonas fuscovaginae and *Sarocladium oryzae* have occasionally caused severe infections in recent years. *P. fuscovaginae* was first reported in Burundi in 1988 (Duveiller et al 1988) mainly in cold regions (Detry 1994). In 1989, high incidence and severity of *P. fuscovaginae* was reported in Madagascar (Rott 1987). Currently, *P. fuscovaginae* is occasionally reported in rice-producing areas of the Democratic Republic of Congo, Rwanda, and Tanzania (Bigirimana et al 2015). *Sarocladium oryzae* has been reported in Rwanda, Cameroon, Niger, Côte d'Ivoire, Kenya, Gambia, Senegal, Nigeria, Tanzania, and Madagascar (Bigirimana et al 2015). The disease is mostly severe in the lowlands and disease outbreaks occur during the rainy season, especially when the weather is hot and humid. Both *S. oryzae* and *P. fuscovaginae* are seed-transmitted, making them more difficult to control.

Other bacterial diseases that occationally cause rice yield losses include *Pantoea ananatis*, a sheath rot pathogen, which is often mistaken for BLB. *P. ananatis* attacks appear to be increasing in Africa. The disease was recently reported to cause severe symptoms on rice in Togo (Kini et al 2017). *Acidovorax avenae*, the causal agent of bacterial brown stripe on rice, is also frequently observed in rice fields. More recently, the disease apparently caused severe infections at Namulonge in Uganda (G. Onaga et al unpubl.).

Burkholderia glumae and other grain rot pathogens also appear to be increasing in Africa, owing to rice production intensification. Although under-reported, this disease is a potential threat to rice production in the face of climate change and a better understanding of its epidemiology is an essential step towards management.

6. Effect of climate change and rice intensification on rice diseases

Climate change is considered to impact on agriculture, in general, and the relationship between plants and pathogens, in particular. There is already evidence that high temperatures occur in most West African countries throughout the year and recent predictions show that temperatures will continue to increase by from 3 to 6°C by 2100 (Cotillon 2017). Curently, maximum temperatures in the Sahel can reach above 40°C, making this region the hottest rice-growing area in Africa. The interactions of these temperature ranges with plants, pathogen biology, vectors, farming practice, and land use are important considerations in forecasting how rice diseases may be affected in Africa. Increasing atmospheric CO_2, O_3, and UV-B radiation also directly impact on plant disease dynamics (Manning and von Tiedemann 1995). By impacting on both pests and host plants (Chancellor and Kubiriba 2006, Baillis 2006) climatic change factors may enable some pest and diseases to expand beyond their current locations.

The role of climate change on the evolution of the interaction between rice varieties and pathogen races is not well documented in Africa. Thus, it is not currently possible to predict the behavior and efficiency of the genetic control of resistance or susceptibility under climate

change. However, recent studies by AfricaRice in collaboration with IRRI have demonstrated that high temperature is less likely to increase rice blast epidemics with climate change, while BLB epidemics may increase and could reduce rice yields by between 0.47 and 0.67 t/ha by 2050 (Duku et al 2016). This is consistent with earlier predictions in which a higher incidence of BLB and BLS diseases in the field was associated with high temperature in combination with high humidity in Burkina Faso, Mali, and Niger (Basso et al 2011). However, both temperature and genetic background tend to affect infection and plant colonization and this has been shown in studies involving *O. japonica* cultivar, Nipponbare, and contrasting genetic backgrounds introgressed with *R* genes (Onaga et al 2016, 2017). High temperature also positively modulated *Xa7*-mediated resistance in indica rice (Webb et al 2010). Conversely, *Xa3/Xa6* is apparently susceptible in some indica backgrounds (Cao et al 2007), which suggests that the genetic background substantially impacts on plant reaction to pathogens, despite climate change.

Drought is another climatic variable with increasing recurrence in most SSA countries. Drought negatively affects crop nutrient uptake, water balance, and the biophysical environment, in general. Drought is predicted to increase in East Africa and frequent periods of drought are already being felt in the region (Carty 2017). Genotypes carrying drought-resistant QTLs were recently found to be more susceptible to BLB than those without them. Moreover, a severe disease susceptibility reaction was observed when both drought QTLs and BLB resistance genes were combined in the same genetic background, suggesting that resistance to BLB under drought conditions appears to be more complicated and may require more studies to identify an effective synergistic combination of BLB resistance genes and drought-resistance QTLs.

In other studies, episodes of blast epidemics observed in Mwea, Kenya in 2009 correlated with climate change-associated variability in weather patterns (Kihoro et al 2009). Moreover, emergence of new variants of RYMV in West-Central Africa (Hébrard et al 2018) and increasing pressure from BLS observed in Uganda, Kenya, Tanzania, and Malawi was associated with the changing climate. Several other pathogens are expected to be directly influenced by global warming, including sheath blight, brown spot, sheath rot, sheat spot, narrow brown spot, and grain rot diseases (Mizobuchi et al 2016, Bigirimana et al 2015). Therefore, climate change studies to better understand how present and future climatic variables influence rice pathogens and pathogen plant interactions are needed to guide implementation of actions that prevent/reduce their impact.

The link between disease pressure and intensification of rice cultivation is also predicted to contribute to emergence of new rice pathogens in Africa (Séré et al 2011). Agroecological changes are critical for disease occurrence and emergence of new pathogen variants. Reliance of modern agricultural systems on synthetic nutrient inputs, improved exotic varieties that cover large cultivable areas, and chemical control methods may potentially contribute to emergence of virulent pathotypes in rice agroecosystems. With time, further increase of rice cultivation is expected in Africa. Thus, rice stakeholders in Africa will have to brace for the arms race between pathogens and the rice crop. Modelling disease occurrence using present and future climatic and cropping pattern data and linking such information to temperal and spatial variation of virulence would be helpful in mitigating against future epidemics.

7. Future perspective and considerations

Rice has become an important crop for farmers in Africa and will continue to be an important component of African farming systems as long as humanity exists. The fundamental problem is on designing management priorities that can maximize grain yields and quality in both smallholder and intensive farming systems. For more than 5 decades, Africa has built a promising rice germplasm base, coming from local and exotic accessions. The international agriculture research institutes that comprise AfricaRice, IRRI, and CIAT have played a great role

in expanding and improving African rice germplasm. However, Africa is extremely vulnerable to unfavorable weather and depredation from pests and diseases. Moreover, diseases present one of the most direct threats to rice production and livelihoods of thousands of farmers who depend on rice. The newly identified pathogens such as those that cause BLS and rice sheath rots and more virulent strains of blast, RYMV, and BLB continually arise and often overcome the resistance in rice cultivars. Although conventional methods have shown some success, more research is needed to develop control methods to reduce yield losses due to these diseases.

Ways to reduce yield losses associated with rice disease epidemics involve the following steps.

- Map and classify the rice production environments according to biophysical characteristics and potential suitability for both rice production and disease occurrence.
- Develop, evaluate, and validate rapid diagnostic platforms and procedures that are specific, sensitive, and reproducible.
- Understand the biology, ecology, epidemiology, and the potential spread of rice pathogens and their relationships with hosts and vectors.
- Characterize rice pathogens to classify and understand evolutionary relationships and diversity that make the pathogen biology agriculturally relevant. Race/lineage identification has remained relatively weak in Africa and hampers tracking of virulence dynamics; there is need to systematically conduct race typing and delineation to support breeding efforts.
- Track the temporal and spatial virulence dynamics of pathogen populations attacking rice in Africa.
- Customize pathogen management and decision tools accompanied by systematic and coordinated improvement of existing models, and/or development of new ones using biophysical and socioeconomic data.

These steps will form the first building blocks for efficient monitoring and early warning systems for forecasting disease epidemics, and will enable early control of rice diseases in Africa. While they are fundamental in disease management, adaptation to pathogen stresses will require harnessing the right rice genetic diversity that can impose divergent selection pressure on the pathogen population into the local breeding programs. The recently developed molecular breeding tools that modify and track specific genes and QTLs, including genome editing, and simplified genomics toolkits that provide optimized marker sets for marker-assisted breeding, would facilitate breeding of resistant varieties. Accompanying these technologies with efficient phenotyping platforms to accelerate suitable germplasm selection, and long- and short-term trainings on genomics-enabled breeding, disease diagnostics, characterization, surveillance, forecasting and general strengthening of laboratory capacity, would effectively minimize disease pressures, and ultimately improve rice productivity in Africa.

The probability of disease occurrence also depends on seed health. Seedborne pathogens are often overlooked, yet the diseases they cause continue to be the principal production constraint in many localities, especially for smallholder farmers who have insufficient resources to apply seed treatment measures. This problem is aggravated by lack of strong institutions and political will to effectively monitor seed exchange, seed production and disease status every season. Designing simple testing tools for seed borne pathogens, and judicious monitoring of their field occurrence will be necessary to control the occurrence and spread of seed born pathogens. Modern tools based on nanobiosensors and unmanned aerial vehicle (UAVs) which detect the presence of pathogenic agents in crops provide a quick and real-time data for rapid responses. These tools are cost-effective and can be modified to detect the pathogen of interest or the presence of disease and disease causing organisms at the seed

level. Rice disease management programs need to look into these sensors and identify the best options for early detection of pathogens and prevention of yield and crop losses.

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