Genetic determinism of the Rice Yellow Mottle virus (RYMV) in Niger Republic: a particular adaptation of the virus isolates to the genetic background of Oryza sativa and Oryza glaberrima.

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Abstract—The Rice yellow mottle is the main rice viral disease in Africa. It affects all rice fields in Niger Republic were it incidence reached 90 %. The disease was propaged from East to West in Africa where two species of rice are present. These are Asiatic rice (Oryza sativa) throughout Africa and African rice (oryza glaberrima), present only in West Africa. This study evaluated the virus genetic determinism adaptation to the two rice species emblaved. It consisted to inoculate Niger-RYMV pathotype or couple of pathotypes E and T in sensitive varieties IR64 (Oryza sativa) and Tog5673 (Oryza glaberrima). The VPg portion of co-inoculated isolates were amplified and sequenced and their amino acids sequences analyzed. The genetic background effect in the pathotypes selection was evaluated by the disease visual symptoms scores and viral ARN quantification. The results showed that the Oryza glaberrima specie discriminated pathotypes T and E. So, T49 confers a strict selective advantage to pathotype T vs E in O. glaberrima specie. However, the contre-selection of isolate Ng140 (T) vs isolate Ng161 (E) in O. glaberrima specie seems fortuitous, indicating an exception to the pathotypes T selective advantage. What recommends the knowledge deepening on this particular adaptation, in order to control the RYMV propagation in Niger Republic and in whole West Africa.

Index Terms— co-inoculations, genetic determinism, Niger-RYMV, pathotypes T and E and rice

Abbreviations

DNA: deoxyribonucleic acid
cDNA: complementary deoxyribonucleic acid
RNA: ribonucleic acid
CERRA: Regional Centre for Agricultural Research
INRAN: National Institute of Agricultural Research of Niger
IRD: Research Institute for Development
DAI: Day After Inoculation
DAS: Day After Sowing
M-MLV-RT: Moloney- Murin Leukomie Virus – Reverse Transcriptase or reverse transcription enzyme
ORF: Open Reading Frame
PBST: Phosphate Buffer Saline Tween
CP: Capsid protein
PCR: Polymerase Chain Reaction
qPCR: Quantitative Polymerase Chain Reaction
RT: Reverse Transcription
RT-PCR: Reverse Transcription Reaction-Chain Amplification and Polymerization Reaction
RYMV: Rice Yellow Mottle Virus
S 1- CA: Central African strain S1
S1-WA: West African S1 strain
Tog: Tropical Oryza Glaberrima
µL: microliter
VPg: Viral protein genome linked.

INTRODUCTION

Rice yellow mottle is an emerging viral disease of rice in Africa (Rakotamalala et al., 2019). It affects all rice-growing areas of Africa, with increasing crop losses (Kouassi et al., 2005; Ndikumana et al., 2011). The disease is present in all rice-growing areas of Niger with incidences between 5-90% and production losses exceeding 70% (Issaka et al., 2012b). It is transmitted by insect vectors of the Chrysomelidae family and mechanically (Abo et al., 2000). The disease pathogen is Rice Yellow Mottle Virus (RYMV).

RYMV is a virus of the genus Sobemovirus, positive RNA, single-stranded (N'gon A Yassi, 1994) and having four open reading frames or ORFs (Fargette et al., 2004). ORF 4 encodes capsid protein (PC) and ORF P2a drives the synthesis of protease and VPg (Virus-Protein, genome-linked), a genomic protein bound to the 5' end of the viral genome (Pinel et al., 2000; Fargette et al., 2004). The serological properties and nucleotide sequences of the capsid protein or the entire genome have made it possible to distinguish six major strains of the virus including strains S1, S2 and S3 present in West Africa and strains S4, S5 and S6 localized in East Africa (Nguessan et al, 2000; Traoré et al., 2005). The S1 strain is subdivided into clades S1-WA from West Africa and S1-CA from Central Africa (Traoré et al., 2005; Tall et al., 2020).

The control of rice yellow mottle is currently based on cultivation practices and the use of resistance genes. However, these resistance genes are increasingly overcome by virulent isolates and genetic background seem to play a decisive role (Traoré et al, 2006; Pinel et al., 2007; Thiémélé et al., 2010; Issaka et al., 2012a; Hébrard et al., 2018). Resistance to RYMV is controlled by the RYMV1 gene (Ndjiondjop et al., 1999) located on chromosome 4 of rice (Albar et al., 2003) and which codes for a 4G eIF (iso) translation initiation factor (Albar et al, 2006; Hebrard et al, 2010). This gene is the most studied compared to the other two resistance genes (RYMV2 and RYMV3) that have been identified in glaberrima accessions (Pidon et al., 2017). The RYMV1 gene has five alleles including the rymv1-1 sensitivity allele and four recessive resistance alleles rymv1-2, rymv1-3, rymv1-4 and rymv1-5 (Thiémélé et al., 2010).

The sensitivity of a cultivar follows a compatible interaction between the VPg of the RYMV and the central eIF(iso) 4G domain of the cultivar genome (Hébrard, 2010). Thus, the overcoming of rice resistance by the virus is due to a restoration of the compatible interaction between the VPg of the virus resistance breaking isolate and the eIF factor (iso) 4 G of the resistant rice variety encoded by the RYMV1 gene. The VPg-eIF(iso)4G compatible interaction is dependent on the polymorphism T (Threonine) and E (Glutamic acid) of the amino acid 49 of the VPg of RYMV (Pinel et al, 2007; Traoré et al. 2010; Poulicard et al, 2012). This polymorphism divides virus isolates into pathotypes T (possessing Threonine in 49) and E (with Glutamic acid in 49). Also, the nature and role of the amino acid 49 of the VPg amplify the importance of the historical contingencies of the ability of the RYMV to overcome the resistance of the RYMV1 gene (Poulicard et al. 2012). In addition, the resistance breakdown is more or less marked, depending on whether the pathotype is at E or T (Pinel et al., 2016).

The present study aimed to better understand the genetic basis for the adaptation of Niger-RYMV T and E pathotypes to the O. sativa and O. glaberrima rice gene pools. The aim was to test competitions between pathotypes T and E, in order to simulate events that occur in the field. They consisted in co-inoculating a pathotype and / or couple of RYMV pathotypes from Niger in the two rice genetic background, in order to obtain, after RT-PCR and qPCR of the portion of VPg extracted from co-inoculated plants, the possible elimination of one pathotype by the other.

1. MATERIALS ET METHODS

1.1. Plant and viral material

The plant material consists of two susceptible varieties (IR64 and Tog5673) belonging to the two species of rice cultivated in Niger. The viral material, consisting of 5 Niger-RYMV isolates from the Issaka collection (2013), was used in single infections (Table 1), and couple of these isolates were assessed in mixed infections (Table 2).

Table 1: Identity of isolates used in competitions between pathotypes E and T

Isolates	Host	Locality	Pathotype*	Origine
Ng40	IR64	Daïkaina	E	IR64
Ng1	IR64	Say 1	E	IR64
Ng161	IR64	Kirkissoye	E	IR64
Ng36	IR64	Kareygorou	T	IR64
Ng140	IR64	Kollo	T	IR64

* *These pathotypes were identified by Issaka (2013)*

Table 2: Couple of pathotypes co-inoculated to IR64 and Tog5673

T-Isolate	E-Isolate		
	Ng1	Ng40	Ng161
Ng36	Ng36/Ng1/IR 64	Ng36/ Ng40/IR 64	Ng36/Ng161/IR 64
	Ng36/Ng1/ Tog5673	Ng36/ Ng40/Tog5673	Ng36 Ng161/ Tog5673
Ng140	Ng140/Ng1/IR 64	Ng140/Ng40/IR 64	Ng140/Ng161/IR 64
	Ng140/Ng1/Tog5673	Ng140/Ng40 /Tog5673	Ng140/Ng161/Tog5673

2.2. Methodology

Molecular tests and analyses were conducted in greenhouse and laboratory at the IRD based to Montpellier.

2.2.1. Sowing, inoculation and scoring

The susceptible varieties IR64 and Tog5673 were each sown in pots of one liter by volume, at the rate of five grains corresponding to five plants per pot per variety. Two batches of seedlings were carried out: sowing for single inoculations or individual passage of pathotypes on genotypes and mixed inoculations of pathotypes E and T.

The inoculum was prepared by grinding the leaf samples or isolates in phosphate buffer 0.1 M pH 7.2 (KH2Po4 0.1M + Na2HPo4 0.1M), at the rate of 1 g of leaves per 10 ml of inoculation buffer. Carborundum (600 mesh) was added to the grind (at a rate of 0.1 g per 100 µl of grind) to serve as an abrasive during inoculation (N'guessan et al., 2001). The inoculum obtained with each isolate or pair of isolates was standardized by qPCR at a concentration of 1012 copies of viral RNA by pathotype, using the supplier's and Poulicard et al., (2010) method.

Inoculation of the pathotypes to the two sensitive genotypes IR64 and Tog5673 occurred at 14 post-sowing (DAS). One pot of each genotype was inoculated by pathotype in the case of single inoculations and for the case of mixed inoculations, two pots of each variety were inoculated. The inoculation consisted of rubbing the leaves of the plants with the standardized inoculum associated with carborundum (600 mesh). Each plant was inoculated with an amount of 100 µl of inoculum in the case of simple inoculations; 100 µl of inoculum, consisting of 50 µl of pathotype grind and 50 µl of inoculation buffer, were inoculated at each plant. For mixed inoculations, the inoculum was formed of an equimolar mixture of inoculum of the two co-inoculated pathotypes, at a rate of 50 µl of inoculum of each pathotypes couple (Table 2). The equimolar concentration was obtained after appropriate dilutions of the initial inoculum.

The disease was assessed 21 and 42 days after inoculation (DAI), using the severity scale 1-9 (where 1 represents no symptoms and 9 represents leaf destruction) for single infections and 60 DAI for mixed infections.

2.2.2. Quantification, standardization and sequencing of viral RNA

The quantification of viral RNA in competition tests was done according to the method of Poulicard et al., (2010). The standard used to quantify the number of viral RNA copies contained in the inoculum and in infected plants corresponds to the cDNA of a purified virus (CIa, strain S2/S3). It is quantified at nanodrop. Dilution series were performed with a standard DNA range of 5.10 8 to 5.10 5 copies for each qPCR reaction; standard ADNs were aliquoted and stored at -20°C. The number of copies per reaction was estimated using Stratagene MX3005 version 2.02.

2. RESULTS

2.1. Visual symptom scores after single and mixed inoculations

In cases of simple inoculations, the highest score (score 9 = total necrosis of leaf tips with often death of the infected plant) was obtained at 42 days after inoculation (DAI) in the IR64 variety by Ng161 (E) isolate. All other isolates infected the IR64 variety with a score between 3 and 9. In the Tog5673 variety, Ng140 (T) and Ng40 (E) isolates showed no symptoms (score 1) and the other isolates showed scores ranging from 3 and 9 (Figure 1).

In mixed inoculations (Figure 2), the most important symptoms (score 9) were observed at 42 DAI on the IR64 variety inoculated with the couple of isolates Ng140 (T) / Ng161 (E). The lowest scores recorded on the IR64 variety were produced by the couples Ng36 (T) / Ng1 (E) and Ng140 (T) / Ng40 (E); other isolates couples caused moderate or high symptoms. Symptoms were low when the Tog5673 variety was co-inoculate with the all the couples of pathotypes. Thus, they varied between 1 and 3 with half of the co-inoculated couples. The couples Ng140 (T) / Ng40 (E) and Ng140 (T) / Ng161 recorded an average score greater than 1 on the variety Tog5673, while this variety was asymptomatic (score = 1) with the couple Ng140 (T) / Ng1 (E).

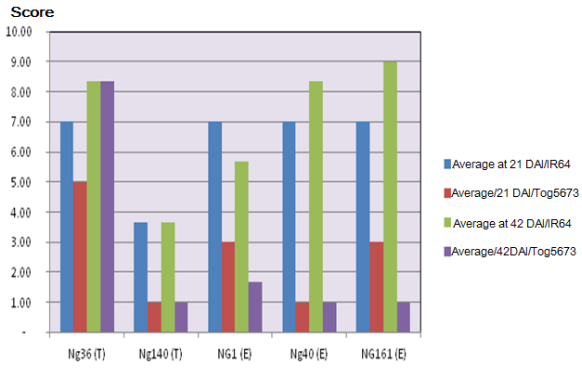

Figure 1: Mean symptom-induced score at 21 and 42 DAI in IR64 and Tog5673 varieties simply inoculated by T&E

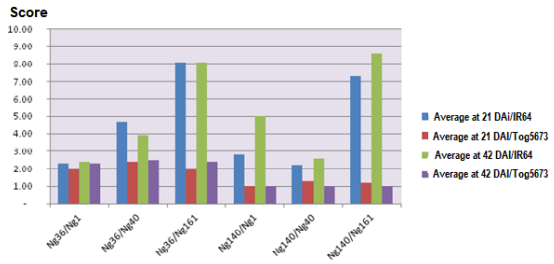

Figure 2: Mean score of induced symptoms at 21 and 42 JHA in co-inoculation of pathotypes T and E to IR64 and Tog5673 varieties.

2.2. Behavior of RYMV pathotypes (T and E) in single infections

Table 3 summarizes the results of the pathotype/genotype interaction of rice during simple inoculations from pathotypes T and E to varieties IR 64 (O. sativa) and Tog5673 (O. glaberrima). The combination of visual scores and the amount of dosed RNA showed that the interaction between the E pathotypes and the sativa genetic background was very strong (more than 10^{11} copies). On the other hand, the interaction between T-pathotypes and varieties varied according to the genetic background. Thus, the Ng36 (T) pathotype strongly infested the glaberrima genetic background (10^{12} copies of RNA) and the Ng140 (T) pathotype reacted weakly with the same genotype, while it accumulated strongly in the sativa genetic background.

Table 3: Effect of rice genotypes on RYMV pathotypes during simply inoculations at 42 DAI

Isolat	IR64		Tog5673		Favorable genetic background**
	Score	Con. en ARN s	Score	Conc. ARN s	
Ng36 (T)	7	$5,4\,10^{11}$	7	$1,2.10^{12}$	Tog5673
Ng140 (T)	3	$1,1.10^{12}$	1	$3,3.10^{7}$	IR64
Ng1 (E)	7	$2,1\,10^{11}$	3	$4,8.10^{11}$	IR64/ Tog5673
Ng40 (E)	7	$3,1\,10^{11}$	1	$2,7.10^{11}$	IR64/ Tog5673
Ng161 (E)	9	$1,4\,10^{11}$	3	$2,9.10^{11}$	IR64/ Tog5673

**Values significantly different from the others in the same column after multiple comparison of the means to the LSD at the 5% threshold; ** Combination of visual score and viral load; Con. RNA= Concentration of RNA (in number of copies); JHA: days after inoculation*

2.3. Selection of the pathotype (T or E) by the genetic background of co-inoculated rice

The analysis of the VPg portions sequences (electrophoregrams and consensus sequences) obtained from the co-inoculated plants made it possible to identify the selected pathotype, according to the genetic background (Table 4). Thus, in the sativa genetic background, the pathotype E was mainly selected (5/6 cases) to the detriment of the T-pathotype. Indeed, in more than half cases of the mixed infections of the IR64 variety the pathotype E was selected while in 2/7 cases, the pathotypes E and T had the same chance to be selected (Ng36/Ng40 and Ng140/Ng1). However, in the Ng140/Ng161 couple, the T-pathotype (Ng140) was selected in 2/3 cases of mixed infections of the IR64 variety.

In the glaberrima genetic background, the adaptation of the pathotypes was variable according to the pathotypes couple. The Ng36 (T) pathotype was absolutely selected (100%) in all mixed infections of the IR64 variety. However, in the interaction Ng140 (T) / Ng161 (E) / Tog5673, the pathotype Ng140 was sometimes selected sometimes counter-selected against the pathotype E (Ng161). Thus, in 5/7 of the cases of mixed Tog5673 infections, the T-pathotype (Ng140) was selected to the detriment of the E-pathotype (Ng161), even though in simple inoculation this isolate was unable to adapt to the Tog5673 variety. At the same time, the T pathotype (Ng140) was counter-selected in 2/7 cases, in favor of the E pathotype (Ng161). However, in mixed inoculations of Tog5673 with the couples Ng40/Ng1 and Ng140/Ng1 the virus did not survive. Because no RNA has been extracted from plants co-inoculated with such pathotypes couples.

Table 4: Selection of pathotypes (T and E) by the rice genotypes tested, at 60 DAI

Couple (T/ E)	IR64 (Tested plants)		Tog5673 (Tested plants)		Sélection pathotype	
	E	T	E	T	IR64	Tog5673
Ng140/Ng1	1	2	-	-	1E vs 2T	0 pathotypes
Ng140/Ng40	2	0	-	-	E	0 pathotypes
Ng140 (T) /Ng161(E)	**1**	**2**	**2**	**5**	**1E vs 2T**	**2E vs 5T**
Ng36/Ng1	3	0	0	2	E	T
Ng36/Ng40	1	1	0	2	1E vs 1T	T
Ng36/Ng161	2	0	0	3	E	T

NB : Les chiffres 0, 1, 2, 3 et 5 indiquent le nombre de plants testés

2.4. Adaptation of T and E pathotypes to rice genetic background

The estimation of the chances of selection of a pathotype (T or E) by either of the two rice genetic background confirmed the results of symptom scoring and viral RNA quantification (Figure 1 and 2, Table 4). Thus, in mixed infection, the behavior of the pathotypes against the genotypes varied, according to the genetic background and the pathotype genetic. All the E-pathotypes evaluated (except Ng161 isolate) were absolutely selected in the sativa genetic background to the detriment of the T pathotypes, while in the glaberrima genetic background, the selection of pathotypes was variable according to the species.

In the glaberrima genetic background, the nNg36 (T) pathotype was absolutely selected. At the same time, the Ng140 (T) pathotype was mostly selected, but counter-selected in 2/7 cases of mixed inoculations of the O. glaberrima species, in favor of the Ng161 (E) pathotype. However, in the Ng140 (T) / Ng1 (E) / Tog5673 and Ng140 (T) / Ng40 (E) / Tog5673 interactions none of the co-inoculated pathotypes were amplified.

4. Discussion

4.1. Behaviour of the pathotypes T and E of Niger-RYMV in the two rice genetic background

Niger-RYMV isolates differ genetically by the amino acid 49 of the VPg. Indeed, Issaka (2013) reported that the amino acid 49 of their VPg discriminates them into pathotypes T and E, depending on whether said amino acid is a Threonine (T) or a glutamic acid (E). The geographical distribution of these RYMV pathotypes is closely associated with the polymorphism of the amino acid 49 of the VPg which appeared under diversifying selection (Pinel et al., 2007; Poulicard et al., 2010). This amino acid would be the basis of the adaptation and pathological diversity of the virus on the continent (Pinel et al., 2016).

In this study, the combination of the mean score of induced symptoms and the quantification of viral RNA extracted from simply inoculated varieties showed a strong interaction between pathotype E and sativa genetic background. On the other hand, it revealed variable interactions between the T-pathotypes and the two genetic backgrounds. Similar results were obtained by Pinel al., (2016), from mixed infections of RYMV pathotypes from different rice-growing areas to the two rice genetic background. Our results indicate that in co-inoculation the E-pathotypes have a selective advantage in the sativa genetic background. However, pathotype E (Ng161) was counter-selected in some cases in mixed infections on the IR64 variety. In the glaberrima genetic background, T-pathotypes were mostly selected. However, the selection of the T pathotype and its counter-selection against the Ng161 pathotype are atypical reactions compared to the genetic background of rice. This suggests that the selection of a pathotype (T or E) by a genetic background is due either to the selective advantage which is an intrinsic aptitude of the selected pathotype or to a fortuitous selection that constitute a particular aptitude. Indeed, if the selective advantage justifies the selection of pathotype T (Ng36) against all pathotypes E in the variety Tog5673, it does not explain the counter-selection of pathotype Ng140 (T), in favour of pathotype Ng161 (E). Similarly, the selective advantage of the pathotype does not explain the selection of the Ng140 (T) pathotype to the detriment of the Ng161 (E) pathotype on the O. sativa species.

These atypical cases of RYMV pathotypes selection by rice genetic background are a novelty compared to the results of Poulicard et al. 2012, and Pinel et al., (2016) who showed on the one hand that pathotypes E and T have the same chance of be select on sativa with, however, a preferential adaptation of pathotypes E to this genetic background and on the other hand, that on the species O. glaberrima the pathotype T has always been selected. Thus, the quasi-selection of RYMV T-pathotypes against E pathotypes in the glaberrima genetic background would be, in the majority of cases of mixed infections, dependent on the genetics (selective value) of the pathotype. Indeed, the presence of threonine at VPg codon 49 is decisive in the adaptive process of the T-pathotype, since only isolates carrying this amino acid were selected almost exclusively in the glaberrima background. New mixed infections of the two genetic background with a wide range of T and E pathotypes from geographically distant rice-growing areas across Africa would complement and certainly help elucidate this hypothesis of fortuitous selection of T and E pathotypes. In the Niger Republic rice-growing area cases of non-multiplication of the Ng140 (T) pathotype in some pathotypes interactions would suggest an absence or a low selective value of T-pathotypes. These results are also explained by the phenomenon of complementation that would have allowed the defective pathotype to complete its replication cycle. Indeed, complementation is an interaction between co-infected pathotypes that would promote the multiplication of one of the pathotypes to the detriment of the other. The selection of pathotype Ng140 (T) against pathotype Ng161 (E) in mixed infection of Tog5673 would corroborate this situation, given that this isolate was the only non-pathogenic on the glaberrima genetic background. These particular adaptation of RYMV pathotypes could also be due to a synergy between two low pathogenic pathotypes, leading to high pathogenicity (Pita et al., 2001).

4.2. Basis of pathotypes T and E adaptation to the two rice genetic background

The adaptation of T and E pathotypes in mixed infections on sativa has shown that it is comparable to a neutral genetic background. Because it did not greatly influence the selective value of pathotypes E and T. Therefore, the selection of T-pathotypes to the detriment of E in the glaberrima specie and their wide distribution in West Africa have been dependent on the genetic background. They have also been

related to the evolutionary history of rice and the virus in Africa (Traoré et al., 2010; Poulicard et al. 2012; Hebrard et al., 2018). Indeed, the dispersion of O. glaberrima rice across the continent was made from the Niger loop to the East as well as to the West (current rice belt of Africa). As for the species O. sativa, it was introduced to East Africa by the Arabs and Indians around the 10th century on the coasts of Tanzania and to the west by the Portuguese in the 15th century. Subsequently, the cultivation of O. sativa rice was intensified in the second half of the 19th century in Central Africa (Portères, 1950; Thiémélé et al., 2010). However, even before the introduction of sativa in West Africa, glaberrima cultivation was practiced there. This culture had diversified in the Niger and Senegal Deltas (Harlan, 1976).
In the rice ecology of western Niger, the dispersion of E pathotypes occurred from East to West, with a cohabitation of the two pathotypes in the West and a contrasting distribution of pathotypes in the East, according to Issaka (2013). The exclusive presence of E-pathotypes in eastern Niger indicates a genetic determinism close to that of isolates from Central Africa (Chad, Cameroon) and East Africa (Pinel et al., 2016). This indicates an inadequacy of T-pathotype in these rice-growing environments; thus confirming the genetic determinism of the pathogenesis related to the amino acid 49 of the RYMV's VPg reported by Pinel et al,. (2007) and Poulicard et al., (2010). As for the rice fields of eastern Niger as well as those of Central Africa, they have been characterized by the intensive cultivation of O. sativa varieties which would be favorable to the maintenance of pathotypes E. The great pathogenic diversity of the virus in the West with cohabitation of the two pathotypes could be explained by the cultivation of the two varieties of rice (Traoré et al., 2010). The maintenance or change of the amino acid 49 of the VPg would have allowed a pathotype to adapt preferentially to one or the other of the two rice species. RYMV was initially present on O. sativa varieties and weeds in East and probably Central Africa before spreading to the west of the continent where glutamic acid (E) mutated to threonine and facilitated passage to O. glaberrima (Fargette et al., 2006; Pinel et al., 2007; Poulicard et al. 2012). Later, with the intensification of sativa cultivation in West Africa, the few E pathotypes that remained subservient to weeds began to colonize the rice fields of this African geographical area again. The estimation of the number, place and date of the events of fixation of the mutations as well as the reconstruction of the associated demographic changes are then necessary to better explain these facts.

CONCLUSION

The results of single and mixed infections showed that the adaptation of RYMV to rice is determined by the amino acid 49 of viral VPg. The presence of this amino acid induced the adaptation of pathotypes T and E to the genetic background of rice. The study showed that the glaberrima genetic background discriminates between the T and E pathotypes of Niger-RYMV. Thus, Threonine in 49 (T49) confers a strict selective advantage to T vs E isolates over the species O. glaberrima. However, the counter-selection of Ng140 (T) vs Ng161 (E) isolate in the glaberrima genetic background appears to be fortuitous (atypical behaviour), given that the selection of Ng161 (E) isolate was made against the selective advantage related to T49; the mechanism of Niger-RYMV pathotypes adaptation to the genetic background of rice being closely related to the evolution of the virus and the history of rice cultivation in Africa. Deepening of knowledge on this particular adaptation of Niger-RYMV isolates would be necessary. The results will ultimately help to better control the spread of the virus in Niger and whole West Africa.

CONFLICTS OF INTEREST

The authors state that there is no conflict of interest.

AUTHORS' CONTRIBUTIONS

IS, NI and SMAM iplemented the protocol, conducted prospecting, sampling and laboratory tests, set up bioassays, collected, analyzed and interpreted the data. DH contributed to sampling, trial conduct, and data interpretation. All authors wrote and corrected the manuscript before and after submission.

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REFERENCES

Abo M.E., Alegbejo M.D., Sy A.A., Misari S.M. 2000. An Overview of the Mode of Transmission, Host Plants and Methods of Detection of Rice Yellow Mottle Virus. Journal of Sustainable Agriculture, Vol. 17 (1) 19-36. DOI : https://doi.org/10.1300/J064v17n01_04

Albar L., Ndjiondjop M.N., Esshak Z., Berger A., Pinel A. Jones M., Fargette D., Ghesquière A. 2003. Fine genetic mapping of a gene required for Rice yellow mottle virus cell-to-cell movement. Theor Appl Genet 107:371–378. DOI: http://dx.doi.org/10.1007/s00122-003-1258-4.

Fargette D., Pinel A., Abubakar Z., Traoré O., Brugidou C., Sorho F., Hébrard E., Choisy M., Séré Y., Fauquet C., Konaté G., 2004. Inferring the evolutionary history of Rice yellow mottle virus from genomic, phylogenetic, and phylogeographic studies. Journal of Virology 78: 3252-3261.

Fargette D., Konaté G., Fauquet C., Muller E., Petershmitt M., Thresh J.2006. Molecular ecology and emergence of tropical plant viruses. Annu.Rev.Phytopath. 44, 235–260. DOI : https://doi.org/10.1146/annurev.phyto.44.120705.104644

Harlan J.R., De West J.M., Stemler A.B. (eds). 1976. Origin of African plant domestication. Mouton publishers, La Hayes.
Hebrard E., Pinel-Galzi A., Oludaré A., Poulicard N., Aribi J., Fabre S., Issaka S., Mariac C., Dereeper A., Albar L., Silue D., Fargette D. 2018. Identification of a Hypervirulent Pathotype of Rice Yellow Mottle Virus: A Threat to Genetic Resistance Deployment in West-Central Africa, Phytopathology, 108: 299–307. DOI : https://doi.org/10.1094/phyto-05-17-0190-r
Issaka S., Onasanya A., Basso A., Sorho, F., Haougui A., Sido A.Y, Ake S., Fargette D. and Sere Y. 2012a. Existence of several pathotypes among Rice Yellow Mottle Virus (RYMV) isolates collected in Niger republic. Trends in Applied Sciences Research 7 (1): 32-45. DOI: 10.3923/tasr. 2012. www.m.elewa.org.
Issaka S., Basso A., Sorho F., Onasanya A., Haougui A., Sido A.Y., Aké S., Fargette D. et Séré, Y. 2012b. Diagnosis and Importance of Rice Yellow Mottle Disease Epidemics in Niger Republic. Journal of Applied Biosciences 50: 3501– 3511. ISSN 1997–5902. DOI : https://doi.org/10.3923/tasr.2012.32.45.
Issaka S. 2013. La panachure jaune du riz en Afrique de l'Ouest: épidémiologie, distribution, pathogénie et variabilité du Rice Yellow Mottle Virus (RYMV) au Niger. Thèse de Doctorat de l'Université Félix Houphouet Boigny de Cocody, Abidjan-Côte d'Ivoire. 156 P.
Kouassi N., N'Guessan P., Albar L., Fauquet C. and Brugidou C. 2005. Distribution and characterization of Rice yellow mottle virus: a threat to African farmers. Plant Dis 89:124–133. DOI: http://dx.doi.org/10.1094/PD-89-0124
Ndikumana I., Gasoré R., Issaka S., Pinel-Galzi G., Onasanya A., Hassani-Mehraban A., Fargette D., Peters D. and Séré Y. 2011. Rice yellow mottle virus in rice in Rwanda: first report and evidence of strain circulation. New Disease Reports 23, 18. DOI : http://dx.doi.org/10.5197/j.2044-0588.2011.023.018.
Ndjiondjop M.N., Albar L., Fargette D., Fauquet C.M. and Ghesquière A. 1999. The genetic basis of high resistance to rice yellow mottle virus (RYMV) in cultivars of two cultivated rice species. Plant Dis. 83:931-935. DOI : http://dx.doi.org/10.1094/PDIS.1999.83.10.931.
Ngon-A-Yassi M., Ritzenthaler C., Brugidou C., Fauquet C. and Beachy R.N. 1994. Nucleotide sequence and genome characterization of rice yellow mottle virus. J. Gen. Virol. 75, 249–257.
N'Guessan P., Pinel A., Caruana M., Frutos R., Sy A., Ghesquière A. and Fargette D. 2000. Evidence of the presence of two serotypes of rice yellow mottle sobemovirus in Côte d'Ivoire. Eur. J. Plant Pathol. 106:167–178. DOI : https://doi.org/10.1023/A:1008792109954.
N'Guessan P., Pinel A., Sy A., Ghesquière A. et Fargette D., 2001. Distribution, pathogenicity and interactions of two strains of Rice yellow mottle virus in forested and savannah zones of West-Africa. Plant Disease 85: 59-64. DOI : https://doi.org/10.1094/PDIS.2001.85.1.59.
Pidon, H. Ghesquière, A. Chéron, S. Issaka, S. Hébrard, E. Sabot, F. Kolade, O. Silué, D. and Albar, L. 2017. Fine mapping of RYMV3: a new resistance gene to Rice yellow mottle virus from Oryzaglaberrima. Theoretical and Applied Genetics, Volume 130, Issue 4, pp 807-818. DOI : https//www.10.1007/s00122-017-2853-0.
Pinel, A., N'Guessan P., Bousalem M. & Fargette D. 2000. Molecular Variability of Geographically Distinct Isolates of Rice Yellow Mottle Virus, Archives of Virology, 145: 1621–38. DOI : https://doi.org/10.1007/s007050070080.
Pinel A., Rakotomalala M., Sangu E., Sorho F., Kanyeka Z., Traoreb O., Sérémé D., Poulicard N., Rabenantoandro Y., Séré Y. et Konaté G. 2007. Theme and Variations in the Evolutionary Pathways to Virulence of an RNA Plant Virus species. PLoS Pathog 3(11).
Pinel-Galzi A., Dubreuil-Tranchant C., Hébrard E., Mariac C., Ghesquière A. and Albar L.2016. Mutations in Rice yellow mottle virus Polyprotein P2a Involved in RYMV2 Gene Resistance Breakdown. Front. Plant Sci. 7:1779. DOI: 10.3389/fpls.2016.01779
Pita, J.S., Fondong, V.N., Sangaré, A., Otin-Napé, G.W., Ogwal, S. and Fauquet, C.M. 2001. Recombinaision, pseudorecombination and synergism of germiniviruses are determinant keys to epidemic of severe cassava mosaic disease in Uganda. Journal of General virology (82) 655-665.
Poulicard N., Pinel-Galzi A., Hébrard E. and Fargette D. 2010. Why Rice yellow mottle virus, a rapidly evolving RNA plant virus, is not efficient at breaking rymv1-2 resistance. Molecular plant pathology 11(1), 145–154.
Poulicard, N., Pinel-Galzi, A., Traoré, O., Vignols, F., Ghesquière, A., Konaté, G., et al. 2012. Historical contingencies modulate the adaptability of Rice yellow mottle virus. PLoS Pathog. 8: e1002482. DOI: 10.1371/journal.ppat. 1002482.
Rakotamala M., Vrancken, B., Pinel-Galzi, A., Ramavovololona, P., Hebrard, E., Randrianangaly, S.J., Dellicour, S., Lemey, P. and Fargette, D. 2019. Comparing patterns and scales of plant virus phylogeography: Rice yellow mottle virus in Madagascar and in continental Africa. Virus Evolution, 5(2): DOI: 10.1093/ve/vez023.
Porteres R. 1950. Vieilles agricultures de l'Afrique subtropicale. Agron. Tropicale 5 :489-507
Tall H., Aribi, J., Camara, S. Pinel-Galzi, A., Poulicard, N., Fargette, D., Hébrard, E. 2020. Nearcomplete genome sequences of Rice yellow mottle virus isolates from Senegal. Microbiol Resour Announc 9:e00937-19. DOI: https://doi.org/ 10.1128/MRA.00937-19.
Thiémélé D., Boisnard A., Ndjiondjop M., Chéron S., Séré Y., Aké S., Ghesquière A. et Albar L., 2010. Identification of a second major resistance gene to Rice yellow mottle virus, RYMV2, in the African cultivated rice species, O. glaberrima. Theor.Appl.Gen. 121,169-179. DOI : https://doi.org/10.1007/s00122-010-1300-2.
Traoré O., Sorho F., Pinel A., Abubakar Z., Banwo O., Maley J., Hébrard E., Winter S., Séré Y., Konaté G. et Fargette D., 2005. Processes of diversification and dispersion of Rice yellow mottle virus inferred from large-scale and high-resolution phylogeographical studies. Molecular Ecology 14:20 97-2110. DOI : https://doi.org/10.1111/j.1365-294X.2005.02578.x
Traoré O., Pinel A., Hébrard E., Gumedzoé Y.D., Fargette D., Traoré A.S. et Konaté G. 2006. Occurrence of Resistance-Breaking Isolates of Rice yellow mottle virus in West and Central Africa. Plant Disease 90: 259-263. DOI : https://doi.org/10.1094/PD-90-0259.
Traoré O., Pinel-Galzi A., Issaka S., Poulicard N., Aribi J. Aké S., Ghesquière A., Séré Y., Konaté G., Hébrard E. et Fargette D. 2010. The adaptation of Rice yellow mottle virus to the eIF(iso) 4G-mediated rice resistance. Virology Virology (408) : 103-108. DOI : https://doi.org/10.1016/j.virol.2010.09.007.