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Population diversity of rice bacterial leaf blight isolates in Uganda

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ABSTRACT

The deployment of resistant cultivars is the best option to control bacterial leaf blight (BLB) and requires an understanding of the pathogen diversity. BLB caused by Xanthomonas oryzae pv.oryzae is a destructive disease of rice (Oryza sativa L.) in Uganda. Unfortunately, detailed information on Ugandan pathogen populations is not available. A set of 21 differential rice genotypes that have known resistance genes were used in a split plot design to test for differential virulence of six different isolates of X.oryzae pv.oryzae obtained from seed samples collected from Eastern Uganda. Statistical analysis showed diversity among isolates. The susceptibility of the differentials with two to four-gene combinations and the resistance of IR24 to Ugandan isolates were unusual and, therefore suggested a different pattern between Asian and Ugandan isolates at pathological level. Although the isolates were fewer in number, attempts to classify them into pathogenic races gave rise to 6 different races, as each isolate caused a particular virulence pattern on the differentials. The six races formed two significantly different ($p \leq 0.05$) groups of one race (UX041) and five races (UX051, UX00, UX050, UX058 and UX044). These races would be used in screening rice germplasm available and/or newly introduced to Uganda.

Key words: BLB, isolate, NILs, rice, *Xanthomonas oryzae* pv. *oryzae*

INTRODUCTION

Rice (*Oryza sativa* L.) is one of the most important staple cereal foods for over half of the world's population; it ranks third after wheat and maize in terms of grain production [6]. However, rice production is contrained by several fungal, bacterial and viral diseases. Bacterial leaf blight (BLB), caused by *Xanthomonas oryzae* pv.*oryzae*, is one of the most destructive diseases of rice worldwide. It was first reported in Japan in 1884 [32] and from then it quickly spread across the Asian countries [29]. In Africa, the first report was in the West African region in 1980s [38]. Research studies conducted in West African countries revealed that the disease incidence ranged from 70-85% and yield losses ranged from 50-90% in the severely infected fields [36]. The presence of *X.oryzae* pv.*oryzae* has now been confirmed in Uganda [37] and this pathogen has the potential to cause huge losses mainly because information on the variability of the local pathogen populations is lacking. Although no studies have been conducted to assess the losses caused by the disease in Uganda, the disease occurence at early stages usually causes greater damage due to complete wilting of the seedling or kresek. However, infection of the plants at the tillering stage can sometimes lead up to 100% yield losses [26].

Varietal resistance is the main control measure presently available since no other control method is economically effective [32]. Fortunately, several resistance genes are available for deployment against this disease. However, this depends on the pathogenic diversity of local pathogen population [3]. In fact, new races of the pathogen keep emerging and can potentially overcome deployed resistance [27, 28]. Most of these races differ between regions, sites and even fields within a site [31]. This implies that breeding for resistance to BLB would be locally done using the prevailing races in that particular geographical location. Although the presence of *X.oryzae* pv.*oryzae* has been confirmed in Uganda [37], knowledge on the variability of *X.oryzae* pv.*oryzae* populations is crucial for the selection of disease resistance cultivars and in designing national breeding strategies for BLB.

Several classical and molecular methods are available for analyzing aggressiveness of *X.oryzae* pv.*oryzae* isolates [31]. One such approach is the use of differential rice lines developed at the International Rice Research Institute (IRRI) by backcrossing resistance sources to the recurrent parent IR24, which was susceptible to all known Philippine races of the pathogen [34, 15, 35]. Using these differential lines, 31 resistant genes have been identified against the Asian isolates [25]. This approach also enabled isolates to be grouped into races [44, 27, 1]. However, the reaction of these differential lines against Ugandan isolates is not known. The main objective of this study was therefore to identify the races of *X.oryzae* pv.*oryzae* prevailing in in the key rice rowing areas of Uganda based on their reactions on the differential lines containing known resistance genes.

MATERIALS AND METHODS

Isolation of bacterial strains: Twenty-three seed samples were collected from rice-growing areas of Budaka, Iganga, Kaliro, Kibuku, Mbale and Pallisa Districts in Eastern Uganda. The seed samples were obtained from the local variety “Supa” that showed visible foliar symptoms of BLB (Fig.1).

Figure 1. BLB symptoms in farmer’s field

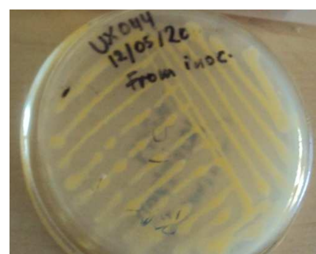

Figure 2. Purification of *X.oryzae* pv.*oryzae* isolate on PSA medium in a Petri plate

The isolation of the bacterial pathogen from seeds was conducted at the Crop Science Laboratory of Makerere University using a methodology described by Singh and Rao [39] and Mortensen [30]. Bacteria that have yellow, viscous and round colonies with entire margins and smooth waxy and shiny surfaces were isolated. The isolated colonies were further purified on peptone sucrose agar (PSA) medium under incubation at 28°C for 3 days [33, 24]. Single colony of each isolate was selected as a representative isolate for the study (Fig.2) and only 6 *X.oryzae* pv.*oryzae* isolates resulted from the pathogenicity tests on the susceptible line IR24 [2]. These isolates were from Kibuku and Pallisa Districts and were named UX050, UX051, UX00, UX041, UX044 and UX058 with the symbol ‘UX’ for “Ugandan *Xanthomonas”*, followed by a corresponding seed sample number. Pure cultures were maintained on PSA slants at +4°C for routine work and in 30% glycerol at -80°C for long-term preservation for further use [16, 3].

Differential rice lines: Twenty-one differential lines obtained from AfricaRice in Benin Republic, were used to characterize the virulence of *X.oryzae* pv.*oryzae* isolates. The differentials included 12 lines with single resistance genes and 8 pyramided lines with two to four-gene combinations, as detailed in Table1. These differentials were the near-isogenic rice lines (NILs) in the background of IR24 [25]. The line IR24 without resistance gene [28] was also used as a susceptible check.

Table 1. Bacterial blight NILs and their respective resistance genes

NILs	*Xa-gene*	NILs	*Xa-gene(s)*
IRBB1	*Xa1*	IRBB21	*Xa21*
IRBB2	*Xa2*	IRBB50	*Xa4+xa5*
IRBB3	*Xa3*	IRBB51	*Xa4+xa13*
IRBB4	*Xa4*	IRBB52	*Xa4+Xa21*
IRBB5	*xa5*	IRBB54	*xa5+Xa21*
IRBB7	*Xa7*	IRBB55	*xa13+Xa21*
IRBB8	*xa8*	IRBB56	*Xa4+xa5+xa13*
IRBB10	*Xa10*	IRBB57	*Xa4+xa5+Xa21*
IRBB11	*Xa11*	IRBB60	*Xa4+xa5+xa13+Xa21*
IRBB13	*xa13*	IR24	-
IRBB14	*Xa14*		

Source: Liu et al., 2007

The differential rice seeds were first sown in Petri plates for 10 days; three better seedlings of each line were transferred to a nine-liter plastic pot in a glasshouse and thinned out to one healthier plant six weeks after transplanting. Standard crop management practices including fertilizer and pesticide applications, watering and hand weeding were employed [4, 26].

Inoculum preparation and inoculation method: A 3-day-old culture of each isolate was used as inoculum. These cultures were retrieved from the PSA plates and incubated at 28°C for 72 hours prior to inoculation. The inoculum was prepared and diluted by suspending separately the bacterial cultures of each isolate in 10 ml of distilled water. Using a spectrophotometer, the absorbance (A) of the inoculum was adjusted to A = 0.05 (620 nm) to make a concentration value of about 10^8 cells per ml [19, 26].

The experiments were conducted from March 2010 to July 2010 in the glasshouse at National Crops Resources Research Institute (NaCRRI)-Namulonge in Central Uganda along latitude 0°32'N and longitude 32°53'E. The experimental design was a split-plot replicated twice, with differential line as main plots and the bacterial isolates as sub-plots [13]. The experimental unit consisted of one plant per pot for 7 pots per cultivar. Top ten or more fully expanded leaves of 70-day-old plants were clip-inoculated with each isolate [18, 5]. A separate pair of sterilized scissors was dipped in the prepared inoculum before clipping 1 to 2 cm off the leaf tip for inoculation such that each plant be inoculated with one strain. Control plants were inoculated similarly with sterile distilled water [26]. Inoculated plants were raised under the day temperature ranging from 24.6 to 33.5°C and 60.15% relative humidity (RH) measured over 3 weeks with a thermo-hygroclock M288CTH. To reduce possible noxious effects of high temperature on the entry of bacteria into infection courts in the presence of sufficient moisture on the leaf surface and subsequent disease aggressiveness, the inoculation was conducted in the evening and inoculated leaves were immediately covered overnight with a polyethylene bag [24, 5]. Because the glasshouse was naturally air-conditioned, the inoculated plants were kept inside the glasshouse under concrete trays permanently filled with water to maintain high the RH required for the disease development. Furthermore, walls and roof were sprinkled with water whenever the RH fell below 50% inside the glasshouse. Plants were sufficiently and regularly watered to avoid any undesirable drought stress which could interfere with bacterial blight symptoms.

Disease assessment: Plant reaction was assessed 21 days after inoculation (DAI) on 10 clipped and inoculated leaves of each line-isolate combination [43, 9]. Results from 21 days were used as virulence reaction. The length of typically yellowish-grey lesions developed below the point of inoculation was measured in centimeter (cm) with a ruler [14]. The average lesion length (LL) of all the 10 leaves in an experimental unit was considered to indicate the virulence level of a particular isolate on a specific host genotype for that replicate. The means of each treatment from the two replications were averaged to obtain the overall mean that was used to assess resistance or susceptibility of rice line. The plant reaction was scored according to the modified Fang's methodology [10]. Therefore, the mean lesion length (MLL) was rated immune, I (0.0 cm); highly resistant, HR (0.1-0.9 cm); resistant, R (1.0-1.9 cm); moderately resistant, MR (2.0-2.9 cm); moderately susceptible, MS (3.0-5.9 cm); susceptible, S (6.0-6.9 cm) and highly susceptible, HS (MLL ≥ 7.0 cm).

Statistical analysis: Original LL data for each line-isolate combination were square-root transformed at $(X+1)^{1/2}$ as suggested by Snedecor and Cochran [41], to increase the precision with which the differences between small means were measured. The transformed data were subjected to the analysis of variance (ANOVA) to test the homogeneity between plots and the significant differential interaction among the isolates, the genotypes and their interactions

using GenStat package [11]. Mean comparisons were performed using the transformed data, but presented as back-transformed means [22]. The Fisher's protected least significant difference (FPLSD) was used for multiple mean comparisons at 5% probability level.

RESULTS

Interaction between differential lines and isolates: Significant difference in disease symtpoms and severity were incited on differential lines depending on isolate used, as shown in Table 2.

Table 2. ANOVA for 21 rice genotypes and their response to infection by six *X.oryzae* pv. *oryzae* isolates

Source	Df	SS	MS	F
Replication	1	0.327	0.327	0.16
Genotype (G)	20	214.835	10.742***	5.15
Main plot error	20	41.71	2.086	1.87
Isolate (I)	6	16.984	2.831*	2.53
Interaction (G x I)	120	183.048	1.525*	1.37
Sub-plot error	126	140.733	1.117	
Total	293	597.638		

** and *** indicate, respectively significance at $p \leq 0.05$ and $p \leq 0.001$,*
Df: degrees of freedom, SS: sum of square, MS: mean sum of squares

Typically the symptoms incited appeared two to three weeks after inoculation. Some of the lines exhibited a resistant reaction marked by delayed initiation of the symptoms and expression of brown necrotic lesions was progressing very slowly (Fig. 3).

Figure 3. Differential rice lines resistant (A) and susceptible (B) to *X. oryzae* pv.*oryzae*

Detailed measurement of the disease showed the mean lesion lengths (MLL) ranging from 0.729 to 9.48 cm, with an overall MLL of 3.63 cm (Table 3). The size of the lesion varied with isolate used.

Surprisingly, the susceptible check IR24 incited the least size of lesion (MLL of 0.729 cm). This line is usually used as a susceptible recurrent parent to build the NILs using Asian isolates. In contrast, the line IRBB56 showed the most susceptible response with an MLL of 9.48 cm (Table 3). Based on lesion sizes, eight genotypes were considered resistant with MLL less than 3.0 cm. These included lines IR24, IRBB7, IRBB14, IRBB4, IRBB1, IRBB21, IRBB55 and IRBB54 (Table 3). The latter two contained pyramided genes for resistant while the rest excluding IR24, were single-gene lines. The following lines had two to four resistant gene combinations but showed a susceptible reaction with an overall MLL ≥ 3.0 cm (Table 3). These are IRBB56, IRBB57, IRBB50, IRBB60, IRBB51 and IRBB52. In general based on MLL rating, all the isolates except UX00 incited the highest MLL on IRBB56 with a MLL ranging from 7.11 to 17.3 cm, with UX00 having a MLL indicating moderate susceptibility (data not shown). The isolates UX051, UX00, UX058 and UX041 were least virulent on IR24 with a MLL varying from 0.26 to 0.77 cm. The isolates UX044 and UX050 were more aggressive to IR24 with MLL of 1.59 cm and 0.96 cm, respectively.

Table 3. Mean lesion lengths (MLL) incited by six *X.oryzae* pv.*oryzae* isolates on leaves of 21 differentials

Differentials	Back-transformed MLL (cm)	Transformed MLL (cm)
IR24	0.729	1.407 a
IRBB7	1.534	2.093 a-b
IRBB14	1.913	2.179 a-c
IRBB55	2.453	2.579 b-d
IRBB1	2.682	2.650 b-d
IRBB21	2.761	2.957 b-e
IRBB4	2.644	2.964 b-e
IRBB2	3.202	2.993 b-e
IRBB54	2.928	3.050 b-e
IRBB52	3.123	3.107 b-f
IRBB5	3.479	3.257 c-g
IRBB10	3.500	3.293 c-h
IRBB51	3.128	3.314 c-h
IRBB3	3.424	3.550 d-h
IRBB8	4.682	3.850 e-h
IRBB11	4.781	3.857 e-h
IRBB13	4.688	4.057 e-i
IRBB60	4.405	4.229 f-i
IRBB50	5.220	4.357 g-i
IRBB57	5.559	4.400 h-i
IRBB56	9.480	5.100 i
Mean	3.63	3.297
%CV	67	32
$LSD_{0.05}$	2.146	1.138

$LSD_{0.05}$: Least significant difference at 0.05 probability level, %CV: coefficient of variation.

Three lines with mulitple resistance genes, namely, IRBB56, IRBB57 and IRBB60, and two with single resistance genes, namely, IRBB11 and IRBB13 exhibited a susceptible reaction against all the isolates (MLL $\geq$ 3 cm). Surprisingly, the line IR24 considered susceptible at IRRI showed a resistant reaction to all isolates.

None of remaining 15 genotypes was resistant to all test isolates; thus they showed differential reaction (Table 4). For example, line IRBB7 was susceptible to only UX051, and lines IRBB14 and IRBB55 were susceptible to UX044; and IRBB10 and IRBB50 were resistant to only UX058. None of the lines evaluated, except the check IR24, was resistant against all the Ugandan isolates of *X.oryzae* pv.*oryzae*.

Table 4. Interaction patterns between Ugandan *X.oryzae* pv.*oryzae* isolates and NILs

No	NILs	Resistance genes (*Xa*)	Isolates					
			UX050	UX051	UX00	UX044	UX058	UX041
1	IRBB 1	*Xa1*	HR	R	MS	MR	HR	S
2	IRBB 2	*Xa2*	MS	MS	MS	R	MR	R
3	IRBB 3	*Xa3*	MS	MR	R	MR	HS	MR
4	IRBB 4	*Xa4*	MR	MR	MS	MS	R	R
5	IRBB 5	*xa5*	HR	MS	MS	MS	MS	HR
6	IRBB 7	*Xa7*	R	MS	R	R	R	R
7	IRBB 8	*xa8*	MR	HS	HS	R	MS	MS
8	IRBB 10	*Xa10*	MS	MS	MS	MS	MR	MS
9	IRBB 11	*Xa11*	HS	S	MS	MS	MS	MS
10	IRBB 13	*xa13*	MS	S	S	MS	S	MS
11	IRBB 14	*Xa14*	R	R	HR	MS	MR	MR
12	IRBB 21	*Xa21*	MR	R	MR	MS	MR	MS
13	IRBB 50	*Xa4+xa5*	MS	MS	HS	MS	MR	HS
14	IRBB 51	*Xa4+xa13*	MS	MR	MS	R	MS	MS
15	IRBB 52	*Xa4+Xa21*	MS	R	MR	MR	MS	MS
16	IRBB 54	*xa5+Xa21*	MR	MS	R	MR	MS	MS
17	IRBB 55	*xa13+Xa21*	MR	MR	R	MS	R	R
18	IRBB 56	*Xa4+xa5+xa13*	HS	HS	MS	HS	HS	HS
19	IRBB 57	*Xa4+xa5+Xa21*	MS	MS	HS	MS	MS	MS
20	IRBB 60	*Xa4+xa5+xa13+Xa21*	MS	MS	MS	MS	MS	HS
21	IR24	-	HR	HR	HR	R	HR	HR

HR: Highly resistant; R: Resistant; MR: Moderately resistant; MS: Moderately susceptible; S: Susceptible; HS: Highly susceptible

The pyramided lines containing resistance (R) genes (*Xa4, xa5, xa13* and *Xa21*) in different combinations exhibited complex genetic effects with resistance levels lower or higher than that of the corresponding monogenic NILs, depending on the nature of cultivar-isolate interactions. For example, the R-gene combination of *Xa4+ xa13* (IRBB51) showed an increased resistance level (quantitative complementation) against the isolate UX044 virulent to both *Xa4* and *xa13*, whereas a decreased resistance level (quantitative compensation) was observed with UX041, UX050, and UX058 all virulent to *xa13* and avirulent to *Xa4* (Table 4). The R-gene pair of *Xa4 + xa5* (IRBB50) maintained a moderate susceptibility level against the isolate UX044, while the resistance level of *Xa4* against UX051 and UX058 was defeated by the quantitative compensation of *xa5* gene (Table 4). Quantitative complementation was observed for *Xa4+Xa21* (IRBB52) and *xa5+Xa21* (IRBB54) with the isolate UX044 virulent to *Xa4, xa5* and *Xa21*. However, the genetic effects of *Xa21* were quantitatively compensated by the gene *xa5* against the isolates UX051 and UX058, but *Xa21* compensated the genetic effects of *Xa4* and *xa5* against UX041 (Table 4). The R-gene of *xa13+Xa21* (IRBB55) was maintained moderately susceptible against UX044, but showed a quantitative complementation against UX00, UX041 and UX058 all virulent to *xa13* and a quantitative compensation against UX051 avirulent to *Xa21* (Table 4). Therefore, the R-gene pairs involving *Xa4, xa5, xa13* and/or *Xa21* suggested that the genetic effects of such pairs were specifically dependent on each tested isolate and were much more complex with three to four R-gene combinations (Table 4).

Based on the number of NILs affected, the isolates UX00 and UX041 were the most virulent. Each one affected a maximum of 13 NILs including 10 in common, namely IRBB1, IRBB8, IRBB10, IRBB11, IRBB13, IRBB50, IRBB51, IRBB56, IRBB57 and IRBB60. The least virulent isolates were UX050 and UX058. Each one affected 11 NILs including 8 in common, namely IRBB3, IRBB11, IRBB13, IRBB51, IRBB52, IRBB56, IRBB57 and IRBB60 (Table 4).

Comparison among isolates and pathotype grouping: The isolate UX041 exhibited the highest virulence with MLL of 4.397 cm, whereas the isolate UX044 exhibited the lowest virulence with MLL of 3.3 cm (Table 5). The mean comparisons among isolates indicated that all 6 isolates were significantly more virulent than the check (distilled sterile water). Based on Fisher's protected least significant difference (FPLSD), the isolates tested were grouped in two categories, namely, the group of isolate UX041 and the group of isolates UX051, UX00, UX050, UX058 and UX044 (Table 5). These results therefore showed the diversity among used isolates though the sample size was limited.

Table 5. Mean lesion lengths (MLL) incited by *X.oryzae* pv.*oryzae* isolates on the leaves of 21 differential rice lines

Isolates	Back-transformed MLL (cm)	Transformed MLL (cm)
Ck	2.311 a	2.838 a
UX044	3.300 ab	3.162 ab
UX058	3.400 bc	3.193 ab
UX050	3.686 bc	3.307 b
UX00	4.131 bc	3.464 b
UX041	4.397 c	3.555 b
UX051	4.214 bc	3.562 b
Mean	3.63	3.297
%CV	67.0	32.1
$LSD_{0.05}$	1.052	0.456

Ck: Check (Distilled sterile water), CV: Coefficient of variation,
$LSD_{0.05}$: Least significant difference at 0.05 probability level,

Comparison of treatment mean values of the same letter indicates insignificant differences ($P < 0.05$), and different letters indicate significant differences ($P < 0.05$), based on FPLSD.

DISCUSSION

The isolation of pure *X.oryzae* pv.*oryzae* colonies from seeds and their ability to incite typical BLB symptoms in host rice lines confirm the presence of the disease in farmers'fields in Eastern Uganda. A previous study also reported the BLB disease in Kibimba rice scheme in Bugiri District of Eastern Uganda [37]. In this study positive detection was in samples of the variety 'Supa' collected from the locations of Pallisa and Kibuku Districts. Taken together, these studies point to a 'hot spot' for BLB in Eastern Uganda.

The 21 differentials that have either single resistance genes or two to four-gene combinations for resistance to *X.oryzae* pv.*oryzae* were used to characterize the virulence of 6 isolates originating in Eastern Uganda. The analysis revealed a significant diversity even within such a limited coverage of Pallisa and Kibuku districts. Such diversity among isolates of *X.oryzae* pv.*oryzae* is reported by Ardales *et al.* [7] who demostrated genetic differences in the population structure of *X.oryzae* pv.*oryzae* at levels of agroecosystems, sites, fields within sites, sampling areas within fields and cultivars. Futhermore, the attempt to classify the isolates collected from Eastern Uganda into specific pathogenic races or pathotypes showed that the 6 tested isolates exhibited different phenotypes of virulence on the set of 21 differential lines since each isolate induced a particular reaction on the differentials. This suggests that 6 different *X.oryzae* pv.*oryzae* races exist in Pallisa and Kibuku region, named, races UX050, UX00, UX041, UX044, UX058 and UX051. However, there is a need to test more isolates from different rice-growing areas of Uganda for a better classification into distinct pathogenic races of *X.oryzae* pv.*oryzae*.

Among the single resistance genes, both *Xa11* and *xa13* were completely overcome by all isolates. A similar reaction of *xa13* tested against the Korean isolates was also reported [17], whereas an effective resistance of *xa13* was found against one Philippine race and six isolates from Punjab in India [40], and against a large part of bacterial population from Niger [8]. Although resistance gene *Xa21* was reported to be effective and stable against multiple isolates of *X. oryzae* pv.*oryzae* from Asia [42, 21] and Mali, in Africa [8], the isolates UX041 and UX044 originating in Uganda were virulent to the line containing *Xa21* gene. Nepalese, Japanese and Korean isolates of *X. oryzae* pv.*oryzae* were also reported to be capable of overcoming the *Xa21* resistance [2].

The susceptibility of the multi-gene lines, namely IRBB56, IRBB57 and IRBB60 against all tested isolates was not expected, given that the genotypes harbor resistance-gene combinations of *Xa4* + *xa5* + *xa13*, *Xa4* + *xa5* + *xa13* and *Xa4* + *xa5* + *xa13* + Xa21, respectively. Various studies showed that the lines with two-, three- and four-gene combinations exhibit more effective and more durable resistance to the *X.oryzae* pv.*oryzae* isolates than the resistance conferred by single genes individually [2, 40, 23]. However, the multi-gene lines IRBB56, IRBB57 and IRBB60 were more susceptible than the single gene lines such as IRBB4, IRBB5 and IRBB21. Similarly, lines IRBB50 (*Xa4* + *xa5*), IRBB51 (*Xa4* + *xa13*), IRBB52 (*Xa4* + *Xa21*), IRBB54 (*xa5* + *Xa21*) and IRBB55 (*xa13* + *Xa21*) showed susceptible reactions to the isolates tested, instead of the expected 'relatively strong resistance to all or most of the isolates' reaction. In a preious study [17], the reaction of lines IRBB50 (*Xa4* and *xa5),* IRBB52 (*Xa4* and *Xa21)* and IRBB54 (*xa5* and *Xa21)* against most of Korean isolates revealed a significantly increased resistance. This was reported to be due to combined effects of the resistance genes or due to one resistance gene effect that masks the susceptibility of the defeated resistance gene [17]. These resistance genes interacted with each other independently and additively, resulting in a quantitative or qualitative complementation. In lines IRBB51 (*Xa4* and *xa13)* and IRBB55 (*xa13* and *Xa21)*, particurlarly, the interactions often resulted in quantitative compensation wherein a susceptible defeated resistance gene has a tendency to reduce the resistance gene effects [17]. Such a phenomenon was observed in the current study, but in a pattern different from that of Jeung *et al.* [17] and dependent on each of the 6 BLB isolates tested.

Similarly, the genotype IR24 that was previously reported to be susceptible [28], and hence universally used as susceptible check against various Asian races of *X.oryzae* pv.*oryzae* [1, 20, 12] was surprisingly resistant to all isolates tested. Line IR24 also tested resistant against the race A3 originating in Mali and different other strains of *X.oryzae pv.oryzae* from W. Africa [14, 8]. This suggests that these African strains possess an avirulent (*avr*) gene that would be specifically recognized by IR24 line. Taken together, these findings reflect a different pattern between Asian and African isolates (including Ugandan isolates) at pathological level. This also highlights the extreme diversity of *X.oryzae* pv.*oryzae* worldwide. The dissimilarity between African and Asian *X.oryzae* pv.*oryzae* races has been reported by Gonzalez *et al*. [14] and Basso *et al*. [8] and gives further credences for regional/local focus to breeding efforts.

CONCLUSION

The population of the BLB pathogen of rice prevailing in Eastern Uganda is diverse. The six Ugandan isolates are similar to most of West African isolates, but different from those from Asian countries [14]. Therefore, the effectiveness of genes varies in locations within the region. A lager collection of isolates is recommended for fully understanding the total BLB population structure, and eventually identifying potential races existing in Uganda. Furthermore, continuously surveying the reactions of the NILs against the pathogen in epidemic hot spots along with

leading cultivars would assist in monitoring changes in the pathogen population followed by prompt development of a new resistant genotype.

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