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# Pathogenicity of Rice Yellow Mottle Virus and the Potential Sources of Resistance against the Disease in Eastern Uganda

[1,2]D. Ochola and [2]G. Tusiime

[1]Cereals Research Program, National Crops Resources Research Institute (NaCRRI), Uganda
[2]Department of Crop Science, Faculty of Agriculture, Makerere University, Uganda

*Corresponding Author: D. Ochola, Cereals Research Program, National Crops Resources Research Institute (NaCRRI), Uganda Tel: +256712301290*

## ABSTRACT

In the present study, we assessed the pathogenicity of the prevalent strains of Rice Yellow Mottle Virus (RYMV) present in Eastern Uganda and identified suitable donors for durable resistance among rice cultivars. Screenhouse studies were conducted using eight isolates of the virus against 16 rice cultivars. Isolate aggressiveness and cultivar resistance were assessed in terms of disease severity and percentage stunting following mechanical inoculation by the finger-rub technique. Highly significant differences ($p<0.001$) between rice cultivars for both parameters revealed the occurrence of different genetic factors for resistance among the rice cultivars. Mean disease severity and percentage stunting ranged between 0.9-9.0 and -1.1-32.4%, respectively for individual cultivars. The absence of significant isolate-by-cultivar interaction suggests a race-non-specific resistance. No significant differences in aggressiveness were obtained between isolates, indicating limited pathotype diversity in Eastern Uganda as evidenced by the absence of mega-environments among the prevalent strains. Based on cultivar resistance ranking, three *O. sativa* subspecies japonica (NERICA6, ITA257 and ITA325) and two *O. sativa* subspecies indica (WAC116 and WAC117) were identified as the potential sources of resistance for improving susceptible local varieties. The confirmed resistance breakdown in GIGANTE has important implications for plant breeding and disease control strategies. Therefore, pyramiding different sources of resistance is recommended for enhanced durability of resistance. In addition, the use of sufficient prerelease challenge by the combination of isolates with different levels of aggressiveness will delay the rise of susceptibility in deployed resistant cultivars.

**Key words:** Rice yellow mottle virus, pathogenicity, percentage stunting, disease severity resistance breakdown

## INTRODUCTION

Rice Yellow Mottle Virus (RYMV) belonging to the genus *Sobemovirus* is a major rice health constraint endemic to the African continent (Abo *et al*., 1998; Banwo *et al*., 2004). The virus was first detected in 1966 at Otonglo along the shores of the Kivorondo Gulf of Lake Victoria (Bakker, 1974; Abo *et al*., 2000; Banwo, 2003). Numerous reports of RYMV incidence have emerged from all the major lowland rice growing countries in Sub-Sahara Africa and on the islands of Zanzibar and Madagascar (Reckhaus and Randrianangaly, 1990; Traore *et al*., 2001; Kouassi *et al*., 2005). Most recently, it was confirmed in Uganda (Pinel-Galzi *et al*., 2006). RYMV has a narrow host range limited to grasses of the Oryzeae and Eragrostidae families

(Konatè *et al.*, 1997; N'Guessan *et al.*, 2001). Natural transmission is in a semi-persistent manner by coleopterous insects (Reckhaus and Adriamasintseheno, 1995; Allarangaye *et al.*, 2007; Nwilene *et al.*, 2009) and by mammals like cattle, rodents and donkeys (Sarra and Peters, 2003). Mechanically it is disseminated through agronomic operations like transplantation and soil incorporation of re-growths during field preparation (Abo *et al.*, 2000; Fargette *et al.*, 2006) and abiotically by wind and irrigation water (Sarra *et al.*, 2004; Sarra, 2005; Abo *et al.*, 1998). There is no evidence of seed-borne transmission of RYMV and of mechanisms that sufficiently explain its long-distance dispersal across Africa (Abo *et al.*, 1998; Allarangaye *et al.*, 2006; Konate *et al.*, 2001). Diseased rice plants are normally characterized by mottle and yellowing symptoms that are often likened to iron toxicity and nitrogen deficiency (N'Guessan *et al.*, 2001; Abo *et al.*, 2005). Necrosis of susceptible cultivars often leads to death (Fauquet and Thouvenel, 1977). Crop damage by RYMV can be devastating causing variable yield losses that may reach 100% depending on the rice genotypes, infectious strain, stage of infection and environment (Paul *et al.*, 2003; Kouassi *et al.*, 2005). Subsequent effects of infection leading to yield loss include stunting, reduced tillering, asynchronous flowering, poor panicle exertion, spikelet discoloration and sterility. Breeding for resistance is the most promising mechanism of limiting infection and yield losses caused by RYMV (Wopereis *et al.*, 2008; Sere at al., 2008). High resistance controlled by a recessive gene *Rymv 1-2* is present in a few *O. glaberrima* accessions and in the *O. sativa* indica cultivars Gigante (Albar *et al.*, 2003; Ndjiondjop *et al.*, 1999) and recently identified in Bekarosaka, a local variety from Madagascar (Albar *et al.*, 2007). Resistance was also observed in Moroberekan and NERICA-L42 under both mechanical and natural viral infestation (Onwughalu *et al.*, 2011). The narrow genetic base of the available germplasm in Uganda (Mogga *et al.*, 2010) is a major bottleneck to pyramiding genes for durable RYMV resistance into the widely preferred but highly susceptible local varieties, such as K5, K85 and K98. In this study, we sought to assess the pathogenicity of the different strains of RYMV present in Eastern Uganda and to identify suitable donors for durable resistance among rice cultivars. The generated information from this study is useful to breeders in developing mechanisms that can effectively counter the widespread of RYMV through the deployment of resistant varieties as replacement of the known susceptible local varieties.

## MATERIALS AND METHODS

**Study area:** The experiment was conducted under artificial conditions in a screen house at the National Crops Resources Research Institute (NaCRRI), Namulonge, Uganda from July-September 2010.

**Rice genotypes:** Sixteen differential rice genotypes (Table 1) used in this study were obtained from Africa Rice Center (WARDA) and from the germplasm collection of the National Crops Resources Research Institute (NaCRRI).

**RYMV isolates:** Eight RYMV isolates used for this study were among the isolates collected from rice fields in Eastern Uganda in 2009 (Table 2). The prevalent strain (isolates Ug 4, Ug 5, Ug 7, Ug 8, Ug 9 and Ug 10) and a newer strain (Isolates Ug 2 and Ug 3) in the country were identified by molecular typing following the protocol by Pinel *et al.* (2000). Before use, isolates were propagated in the screenhouse on the highly susceptible cultivar IR64 by mechanical inoculation of two-week-old seedlings using the finger-rub technique (Raymundo *et al.*, 1979). Four weeks after inoculation, leaves of each RYMV isolate bearing typical yellow mottle symptoms were harvested and used for inoculating rice genotypes (Onasanya *et al.*, 2004).

Table 1: Identity of 16 rice genotypes used to study pathological properties of RYMV isolates and identify potential donors of resistance to the disease

| Code | Genotypes | Type | Ecologies |
|---|---|---|---|
| $V_1$ | NERICA 1 | *O. sativa javanica* | Upland |
| $V_2$ | NERICA 4 | *O. sativa javanica* | Upland |
| $V_3$ | NERICA 6 | *O. sativa javanica* | Upland |
| $V_4$ | NERICA 10 | *O. sativa javanica* | Upland |
| $V_5$ | ITA257 | *O. sativa japonica* | Upland |
| $V_6$ | ITA325 | *O. sativa japonica* | Upland |
| $V_7$ | TOG5682 | *O. glaberrima* | Upland |
| $V_8$ | CG14 | *O. glaberrima* | Upland |
| $V_9$ | WITA9 | *O. sativa indica* | Lowland |
| $V_{10}$ | PNA647F4-56 | *O. sativa japonica* | Lowland |
| $V_{11}$ | IR47686-15-1-1 | *O. sativa japonica* | Lowland |
| $V_{12}$ | GIGANTE | *O. sativa indica* | Lowland |
| $V_{13}$ | WAC116 | *O. sativa japonica* | Lowland |
| $V_{14}$ | WAC117 | *O. sativa japonica* | Lowland |
| $V_{15}$ | K85 | *O. sativa indica* | Lowland |
| $V_{16}$ | IR64 | *O. sativa indica* | Lowland |

Table 2: Identity of RYMV isolates used for pathological studies

| Isolates | District of origin | Collection site | Ecologies |
|---|---|---|---|
| Ug 2 | Butaleja | Doho | Irrigated lowland |
| Ug 3 | Butaleja | Doho | Irrigated lowland |
| Ug 4 | Butaleja | Doho | Irrigated lowland |
| Ug 5 | Butaleja | Doho | Irrigated lowland |
| Ug 7 | Mbale | Musoto | Rainfed lowland |
| Ug 8 | Mbale | Musoto | Rainfed lowland |
| Ug 9 | Mbale | Musoto | Rainfed lowland |
| Ug 10 | Mbale | Musoto | Rainfed lowland |

**Experimental design and treatment allocation:** The experiment was arranged according to a randomized block split plot design with three replicates. The isolates and un-inoculated control represented the Main-plots while the 16 cultivars represented the sub-plots. The cultivar IR64 was used as the highly susceptible check while GIGANTE was the highly resistant check. Six seeds of each cultivar were directly sowed in 25 cm-diameter pots and later thinned to three-plants per pot after one week. Furadan (5 G) was applied to each pot in order to control insect damage. Pots were constantly supplied with fresh tap water in the mornings and when necessary in the afternoons. Two grams of NPK fertilizer was applied to the plants before inoculation.

**Inoculation of rice genotypes:** Mechanically inoculation was achieved using the finger-rub technique. One gram of sliced infected leaf tissue of each isolate was squashed in a drop of double distilled water using sterile mortars and pestles until 80% were crushed. The leaf extract was diluted 10 times by addition of 10 mL of double distilled water. The resulting homogenate was allowed to settle, decanted and preserved in a refrigerator. Inoculum was mixed with fine sand and subsequently rubbed onto leaves using pieces of cotton wool. Care was taken to ensure maximum wetting and formation of mild bruises that acted as infection passageways.

**Disease severity and stunting measurements:** Appearance of symptoms and disease progress was monitored for each inoculated genotype from 7 to 32 days post inoculation (dpi). Disease severity was scored with a modified version of the scale developed by John and Thottappilly (1987) where 0 was equivalent to no infection and 9 was 100% infection. Plant height of inoculated plants and uninoculated controls were measured from the ground level to the tip of the tallest leaf. The impact of RYMV on growth of each genotype was calculated using the following formulae: $(N_i\text{-}I)/N_i\times100$, whereby $N_i$ and I are the mean values of uninoculated controls and inoculated plants, respectively.

**Data analysis:** Severity scores and plant height data from both the inoculated and control genotypes were used to determine the disease severity and percentage stunting for each genotype. Variance and mean comparison of disease severity and percentage stunting were analyzed using GENSTAT® 13 Edition statistical software. By the harmonization of severity scores and percentage stunting, varietal resistance of rice genotypes was categorized into five classes: highly resistant (score 0 and 0%), resistant (1-3 and <5%), moderately resistant (3-5 and 5.1-25%), susceptible (5-7 and 25.1-45%) and highly susceptible (7-9 and >45%). Genotype (cultivar) by environment (isolate) interaction effects of RYMV disease on rice growth was carried out using Additive Main effect and Multiplicative Interaction (AMMI) analysis (Ebdon and Gauch, 2002). The GGE biplot were constructed used to depict and ascertain whether there is any meaningful cultivar by isolate interactions and whether resistance was race-specific or race-non-specific.

## RESULTS

**Effect of rice yellow mottle virus on symptom development in rice cultivars:** The results of the analysis of variance for disease severity scores at 32 days post inoculation (dpi) (Table 3) revealed that all 16 rice cultivars varied significantly ($F = 44.68$, $df = 15$, $p\leq0.001$). However, no significant cultivar-isolate interactions ($F = 0.8$, $df = 105$, $p = 0.05$) were observed which suggests that the observed disease responses to RYMV isolates were strongly dependent on the race-non-specific resistance of individual rice cultivars. Disease progress on the 16 rice cultivars caused by the eight representative RYMV isolates (Fig. 1) shows that early disease initiation at 8 dpi, rapid disease development and highest mean disease severities, characterized RYMV infection of the highly susceptible check IR64 and the local variety K85 (Fig. 1). By contrast, delayed disease initiation, slow disease development and lower mean disease severities occurred in NERICA6, ITA257, WAC116 and WAC117 (Fig. 1). The locally preferred rice variety in Uganda K85 was among the susceptible varieties. Generally, average disease severity scores were lowest (0.9) in WAC116 and highest (9) in IR64 plants (Table 5). Amongst the upland rice cultivars evaluated ($V_1$-$V_8$) the overall mean severities were between 2.2-6.8, whereby the lowest disease severity score (2.2) and the highest disease severity score (6.8) were observed in ITA257 and NERICA1, respectively. All the eight RYMV isolates from Eastern Uganda used in this study significantly resulted into different disease levels ($F = 2.98$, $df = 7$, $p\leq0.05$). A few isolates were able to circumvent to some extent the high resistance in GIGANTE by inducing disease severities in the range 1.9-6. Although all isolates affected the evaluated cultivars, the progress of RYMV symptoms was much slower on plants inoculated with isolate Ug 7. The isolates Ug 2 and Ug 4 induced the least amount of symptoms on the local variety K85 while isolates Ug 5 and Ug 9 did not induce any observable symptoms on cultivars WAC116 and WAC117.

Table 3: Analysis of variance for severity scores of 16 rice genotypes affected by the inoculation with eight RYMV isolates from Eastern Uganda

| Source of variation | df | ss | ms | F † |
|---|---|---|---|---|
| Blocks | 2 | 112.04 | 56.02 | 16.86 ns |
| Sub-plot (Variety) | 15 | 1938.67 | 129.24 | 44.68 ** |
| Main-plot (Isolates) | 7 | 69.25 | 9.89 | 2.98 * |
| Variety * Isolate | 105 | 242.73 | 2.31 | 0.80 ns |
| Main-plot Error | 14 | 46.52 | 3.32 | |
| Sub-plot Error | 240 | 694.17 | 2.89 | |
| Total | 383 | 3103.37 | | |

†ns: Not significant, *: Significant at 5% level, **: Significant at 1% level

Table 4: Analysis of variance for percentage stunting of 16 rice genotypes affected by the inoculation with eight RYMV isolates from Eastern Uganda

| Source of variation | df | ss | ms | F † |
|---|---|---|---|---|
| Blocks | 2 | 22743.1 | 11371.5 | 54.18 ns |
| Sub-plot (Variety) | 15 | 24527.8 | 1635.2 | 12.79 ** |
| Main-plot (Isolates) | 7 | 978.2 | 139.7 | 0.67 ns |
| Variety * Isolate | 105 | 6001.7 | 57.2 | 0.45 ns |
| Main-plot Error | 14 | 2938.2 | 209.9 | |
| Sub-plot Error | 240 | 30687.5 | 127.9 | |
| Total | 383 | 87876.0 | | |

†ns: Not significant, *: Significant at 5% level, **: Significant at 1% level

**Effect of rice yellow mottle virus on plant height of rice cultivars:** Results of analyses of variance presented in Table 4 revealed significant cultivar effects for percent stunting ($F = 12.79$, $df = 15$, $p \leq 0.001$) following infection with the different RYMV isolates from Eastern Uganda. However, it failed to identify any significant effect of isolate aggressiveness and cultivar-isolate interaction. The observed range -6.4 to 36.2% stunting among cultivars suggested the high variability in resistance to RYMV disease. The lowest average stunting (-1.1%) was in cultivar WAC117 while the highest average stunting (32.4%) was in the cultivar IR64. The least stunting observed in WAC117 suggests high levels of resistance to RYMV in this cultivar (Table 6). All isolates studied reduced plant height of the globally acclaimed RYMV resistant cultivar GIGANTE by between 6.8-17.8%. The cultivar WAC117 expressed stable resistance to all isolates except for Ug 7. The isolates Ug 5 and Ug 4 caused the highest (13.7%) and the least (8.8%) stunting of rice cultivars tested (Table 6). Ug 4 and Ug 8 did not affect plant height of local variety K85 despite its high mean severity of 7.6 presented in Table 5. Similarly, Ug 3 did not substantially affect height of PNA647F4-56 plants. Amongst the evaluated upland rice cultivars, ITA257 and ITA325 had the lowest stunting of 0.9 and 2.3, respectively while the NERICA cultivars were most affected in the order NERICA10>NERICA1>NERICA4>NERICA6.

**Assessment of genotype and environment interactions using additive main effects and multiplicative interaction (AMMI):** Differential responses of varying genotypes under changes in the environment are difficult to understand using ordinary ANOVA. The AMMI model used to generate Genotype and Genotype by Environment (GGE) interaction biplots explained 94.74 and 88.04% of the variation in disease severity and percentage stunting that was due to GGE, respectively (Fig. 2, 3). A convex-hull drawn on cultivars from the biplot origin gave four sectors

Table 5: Disease severity scores due to eight rice yellow mottle virus isolates on 16 varieties

| Isolate | V1 | V2 | V3 | V4 | V5 | V6 | V7 | V8 | V9 | V10 | V11 | V12 | V13 | V14 | V15 | V16 | I-Mean |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ug2 | 7.2$^{g}$ | 5.2$^{def}$ | 2.6$^{b}$ | 5.2$^{def}$ | 3.4$^{bc}$ | 2.1$^{ab}$ | 5.0$^{de}$ | 4.8$^{de}$ | 5.4$^{def}$ | 6.8$^{fg}$ | 6.1$^{f}$ | 6.0$^{f}$ | 1.6$^{a}$ | 1.4$^{a}$ | 7.0$^{g}$ | 9.0$^{h}$ | 4.9$^{b}$ |
| Ug3 | 6.8$^{fg}$ | 5.5$^{def}$ | 2.5$^{ab}$ | 5.7$^{ef}$ | 3.4$^{bc}$ | 3.0$^{bc}$ | 4.1$^{cd}$ | 4.8$^{de}$ | 5.2$^{def}$ | 4.1$^{cde}$ | 5.0$^{de}$ | 2.2$^{ab}$ | 0.7$^{a}$ | 1.0$^{a}$ | 7.2$^{g}$ | 9.0$^{h}$ | 4.4$^{ab}$ |
| Ug4 | 6.5$^{f}$ | 3.3$^{bc}$ | 1.1$^{a}$ | 5.9$^{efg}$ | 1.3$^{a}$ | 1.4$^{a}$ | 3.2$^{bc}$ | 3.1$^{bc}$ | 5.7$^{efg}$ | 6.3$^{f}$ | 5.5$^{def}$ | 3.0$^{bc}$ | 3.7$^{bcd}$ | 3.9$^{bcd}$ | 6.0$^{f}$ | 9.0$^{h}$ | 4.3$^{ab}$ |
| Ug5 | 6.1$^{f}$ | 5.5$^{def}$ | 2.8$^{bc}$ | 5.9$^{efg}$ | 1.8$^{ab}$ | 2.3$^{ab}$ | 3.9$^{bcd}$ | 4.5$^{cde}$ | 5.0$^{def}$ | 5.0$^{de}$ | 5.5$^{def}$ | 1.9$^{ab}$ | 0.1$^{a}$ | 0.0$^{a}$ | 7.7$^{gh}$ | 9.0$^{h}$ | 4.2$^{ab}$ |
| Ug7 | 5.4$^{def}$ | 4.3$^{cde}$ | 1.5$^{a}$ | 5.3$^{def}$ | 1.2$^{a}$ | 1.8$^{ab}$ | 2.3$^{ab}$ | 0.3$^{a}$ | 4.3$^{cde}$ | 4.6$^{de}$ | 5.2$^{def}$ | 2.3$^{ab}$ | 0.0$^{a}$ | 0.2$^{a}$ | 7.4$^{g}$ | 9.0$^{h}$ | 3.5$^{a}$ |
| Ug8 | 7.7$^{gh}$ | 5.2$^{def}$ | 2.8$^{bc}$ | 5.7$^{efg}$ | 1.6$^{ab}$ | 3.4$^{bc}$ | 5.4$^{def}$ | 4.3$^{cde}$ | 6.1$^{f}$ | 5.4$^{def}$ | 6.5$^{f}$ | 2.3$^{ab}$ | 0.6$^{a}$ | 0.8$^{a}$ | 8.3$^{gh}$ | 9.0$^{h}$ | 4.7$^{b}$ |
| Ug9 | 7.7$^{gh}$ | 5.0$^{de}$ | 3.0$^{bc}$ | 6.0$^{fg}$ | 2.6$^{b}$ | 2.2$^{ab}$ | 5.2$^{def}$ | 3.3$^{bc}$ | 6.3$^{f}$ | 4.2$^{cde}$ | 7.6$^{gh}$ | 3.0$^{bc}$ | 0.0$^{a}$ | 0.0$^{a}$ | 9.0$^{h}$ | 9.0$^{h}$ | 4.6$^{b}$ |
| Ug10 | 7.2$^{g}$ | 4.6$^{de}$ | 2.6$^{b}$ | 5.9$^{efg}$ | 2.6$^{b}$ | 3.9$^{bcd}$ | 5.4$^{def}$ | 5.0$^{de}$ | 4.3$^{cde}$ | 5.2$^{def}$ | 5.7$^{efg}$ | 3.8$^{bcd}$ | 0.2$^{a}$ | 1.3$^{a}$ | 7.9$^{gh}$ | 9.0$^{h}$ | 4.7$^{b}$ |
| V-Mean | 6.8$^{fg}$ | 4.8$^{de}$ | 2.4$^{ab}$ | 5.7$^{ef}$ | 2.2$^{ab}$ | 2.5$^{ab}$ | 4.3$^{cde}$ | 3.8$^{bcd}$ | 5.3$^{def}$ | 5.2$^{def}$ | 5.9$^{efg}$ | 3.1$^{bc}$ | 0.9$^{a}$ | 1.1$^{a}$ | 7.6$^{gh}$ | 9.0$^{h}$ | |

In a column, means followed by a common letter are not significant at the 5% level by Tukey's HSD test. Data illustrated was collected at 32 days post inoculation. The genotypes: $V_1$: NERICA 1, $V_2$: NERICA 4, $V_3$: NERICA 6, $V_4$: NERICA 10, $V_5$: ITA 257 (NARIC 1), $V_6$: ITA 325 (NARIC 2), $V_7$: TOG 5682, $V_8$: CG 14, $V_9$: WITA 9, $V_{10}$: PNA647F4-56, $V_{11}$: IR47686-15-1-1, $V_{12}$: GIGANTE, $V_{13}$: WAC 116, $V_{14}$: WAC 117, $V_{15}$: K 85, $V_{16}$: IR 64

Table 6: Varietal percentage (%) stunting due to eight rice yellow mottle virus isolates on 16 varieties

| Isolate | V1 | V2 | V3 | V4 | V5 | V6 | V7 | V8 | V9 | V10 | V11 | V12 | V13 | V14 | V15 | V16 | I-Mean |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ug2 | 11.5$^{bcde}$ | 12.5$^{cde}$ | 22.5$^{fg}$ | 18.7$^{f}$ | -1.9$^{a}$ | 7.7$^{abcd}$ | 5.2$^{abcd}$ | 21.4$^{fg}$ | 13.6$^{cde}$ | 15.9$^{def}$ | 3.6$^{abc}$ | 6.8$^{abcd}$ | 9.7$^{abcde}$ | -0.7$^{a}$ | 0.5$^{ab}$ | 32.1$^{g}$ | 11.2$^{a}$ |
| Ug3 | 16.5ef | 21.6fg | 10.1$^{bcde}$ | 18.4f | -0.4a | 6.3$^{abcd}$ | 2.8$^{abc}$ | 14.5$^{def}$ | 14.6$^{def}$ | 0.9$^{ab}$ | -3.8$^{a}$ | 10.6$^{bcde}$ | 10.1$^{bcde}$ | -3.9$^{a}$ | 5.3$^{abcd}$ | 27.1$^{fg}$ | 9.4$^{a}$ |
| Ug4 | 11.9$^{bcde}$ | 10.8$^{bcde}$ | 8.7$^{abcde}$ | 17.1$^{f}$ | -5.7$^{a}$ | -0.4$^{a}$ | 0.6$^{ab}$ | 11.4$^{bcde}$ | 13.3$^{cde}$ | 7.5$^{abcd}$ | 5.2$^{abcd}$ | 16.2$^{ef}$ | 9.7$^{abcde}$ | 0.1$^{ab}$ | -1.6$^{a}$ | 35.5$^{g}$ | 8.8$^{a}$ |
| Ug5 | 17.6$^{f}$ | 15.7$^{def}$ | 15$^{ef}$ | 24.9$^{fg}$ | 10.2$^{bcde}$ | 9.1$^{abcde}$ | 6.1$^{abcd}$ | 10.3$^{bcde}$ | 16$^{ef}$ | 11.7$^{bcde}$ | 3.6$^{abc}$ | 17.8$^{f}$ | 11.1$^{bcde}$ | 3.2$^{abcd}$ | 10.2$^{bcde}$ | 36.2$^{g}$ | 13.7$^{a}$ |
| Ug7 | 18.5$^{f}$ | 14$^{def}$ | 9.9$^{abcde}$ | 14$^{def}$ | 4.3$^{abcd}$ | 0$^{ab}$ | 12.4$^{cde}$ | 11.6$^{abcd}$ | 19.1$^{f}$ | 6.6$^{abcd}$ | 0.7$^{ab}$ | 8.6$^{abcde}$ | 16.5$^{ef}$ | 4.7$^{abcd}$ | 12.6$^{cde}$ | 29.9$^{fg}$ | 11.5$^{a}$ |
| Ug8 | 14.8$^{def}$ | 13.5$^{cdef}$ | 15.8$^{def}$ | 17.3$^{f}$ | -1.3$^{a}$ | -2.5$^{a}$ | 9.2$^{abcde}$ | 15$^{def}$ | 23.3$^{fg}$ | 11.1$^{bcde}$ | 12.5$^{cde}$ | 11.9$^{bcde}$ | 12.2$^{cde}$ | -6.4$^{a}$ | -5.4$^{a}$ | 35.8$^{g}$ | 11.1$^{a}$ |
| Ug9 | 21.6$^{fg}$ | 17.9$^{f}$ | 13.2$^{cde}$ | 20$^{fg}$ | 1.1$^{ab}$ | -1.5$^{a}$ | 18$^{f}$ | 18$^{f}$ | 22.5$^{fg}$ | 11.1$^{bcde}$ | 1.1$^{abc}$ | 16$^{ef}$ | 8.5$^{abcde}$ | 0.1$^{ab}$ | 17.3$^{f}$ | 30.5$^{g}$ | 13.5$^{a}$ |
| Ug10 | 18.1$^{f}$ | 11.8$^{bcde}$ | 10.6$^{bcde}$ | 15.2$^{def}$ | 0.7$^{ab}$ | -0.5$^{a}$ | 14$^{def}$ | 12.8$^{cde}$ | 18.5$^{f}$ | 20.1$^{fg}$ | 8.8$^{abcd}$ | 8$^{abcde}$ | 9.6$^{abcde}$ | -5.6$^{a}$ | 6.2$^{abcd}$ | 32.4$^{g}$ | 11.3$^{a}$ |
| V-Mean | 16.3$^{ef}$ | 14.7$^{def}$ | 13.2$^{cde}$ | 18.2$^{f}$ | 0.9$^{ab}$ | 2.3$^{abc}$ | 8.5$^{abcde}$ | 14.4$^{def}$ | 17.6$^{f}$ | 10.6$^{bcde}$ | 4.0$^{abcd}$ | 12.0$^{cde}$ | 10.9$^{bcde}$ | -1.1$^{a}$ | 5.6$^{abcd}$ | 32.4$^{g}$ | 11.3$^{a}$ |

In a column, means followed by a common letter are not significant at the 5% level by Tukey's HSD test. Data illustrated was collected at 32 days post inoculation. The genotypes: $V_1$: NERICA 1, $V_2$: NERICA 4, $V_3$: NERICA 6, $V_4$: NERICA 10, $V_5$: ITA 257 (NARIC 1), $V_6$: ITA 325 (NARIC 2), $V_7$: TOG 5682, $V_8$: CG 14, $V_9$: WITA 9, $V_{10}$: PNA647F4-56, $V_{11}$: IR47686-15-1-1, $V_{12}$: GIGANTE, $V_{13}$: WAC 116, $V_{14}$: WAC 117, $V_{15}$: K 85, $V_{16}$: IR 64

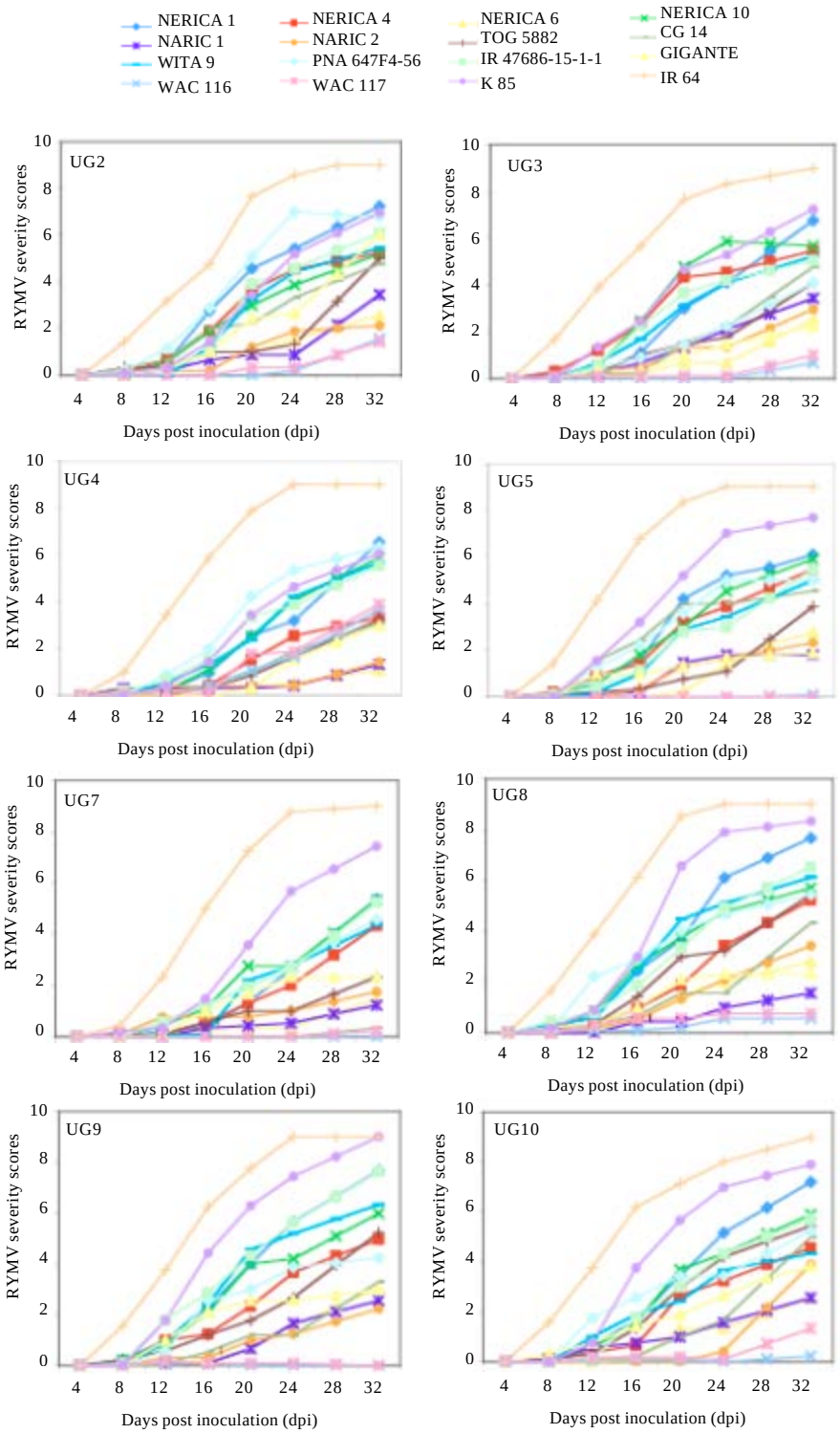


Fig. 1: The pattern of the reaction of 16 rice cultivars challenged by isolates Ug2, Ug3, Ug4, Ug5, Ug7, Ug8, Ug9 and Ug10 from Eastern Uganda

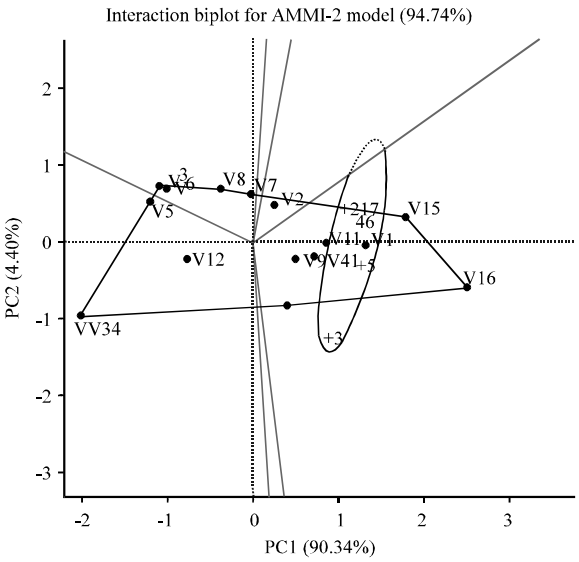


Fig. 2: Genotype (cultivar) by environment (isolate) interaction effects on disease severity using the additive main effects and multiplicative interaction (AMMI) anlaysis. Genotypes: $V_1$: NERICA 1, $V_2$: NERICA 4, $V_3$: NERICA 6, $V_4$: NERICA 10, $V_5$: ITA 257 (NARIC 1), $V_6$: ITA 325 (NARIC 2), $V_7$: TOG 5682, $V_8$: CG 14, $V_9$: WITA 9, $V_{10}$: PNA647F4-56, $V_{11}$: IR47686-15-1-1, $V_{12}$: GIGANTE, $V_{13}$: WAC 116, $V_{14}$: WAC 117, $V_{15}$: K 85, $V_{16}$: IR 64. Isolates: 1: Ug 2, 2: Ug 3, 3: Ug 4, 4: Ug 5, 5: Ug 7, 6: Ug 8, 7: Ug 9, 8: Ug 10

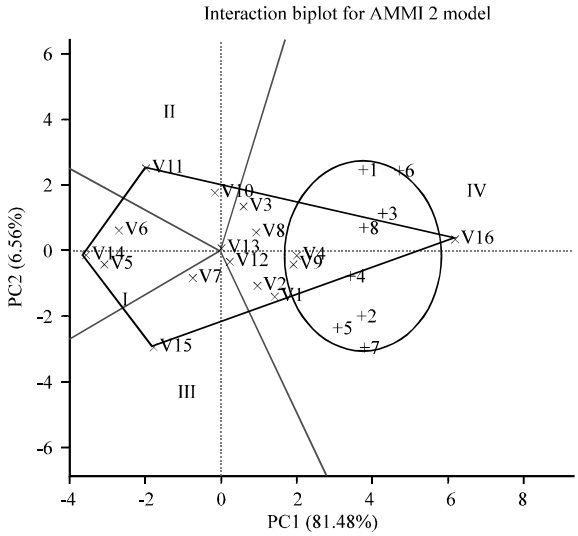


Fig. 3: Genotype (cultivar) by environment (isolate) interaction effects on percentage stunting using the additive main effects and multiplicative interaction (AMMI) anlaysis. Genotypes: $V_1$: NERICA 1, $V_2$: NERICA 4, $V_3$: NERICA 6, $V_4$: NERICA 10, $V_5$: ITA 257 (NARIC 1), $V_6$: ITA 325 (NARIC 2), $V_7$: TOG 5 682, $V_8$: CG 14, $V_9$: WITA 9, $V_{10}$: PNA647F4-56, $V_{11}$: IR47686-15-1-1, $V_{12}$: GIGANTE, $V_{13}$: WAC 116, $V_{14}$: WAC 117, $V_{15}$: K 85, $V_{16}$: IR 64. Isolates: 1: Ug 2, 2: Ug 3, 3: Ug 4, 4: Ug 5, 5: Ug 7, 6: Ug 8, 7: Ug 9, 8: Ug 10

with WAC117, IR47686-15-1-1, K85 and IR64 as the vertex cultivars. The cultivars that were tolerant or most susceptible to some or all of the environments fell in the sector in which IR64 was the vertex cultivar. The cultivars IR 64 and K85 possessed relatively long vectors suggesting that they were highly responsive to infection by all RYMV isolates. Conversely, the sector in which WAC117 was the vertex cultivar represented the most resistant cultivars in any environment. The cultivars WAC116 and WAC117 responded to infection more oppositely from all other cultivars, portraying a presence of resistance against many of the RYMV isolates. However, GGE biplots revealed lack of pathotype variability in Eastern Uganda because isolates were strongly associated with each other to be distinguished and classified according to their pathogenicity on the 16 evaluated cultivars.

**Classification of cultivar viral resistance levels:** The relative resistance levels of 16 rice cultivars were classified using cluster-dendrograms on the precedent of symptom severity and percentage stunting. In Fig. 4, rice cultivars were successfully discriminated according to their severities into four distinct classes: two highly resistant cultivars WAC116 and WAC117; six resistant cultivars NERICA6, ITA257, ITA325, GIGANTE, TOG5682 and CG 14; five moderately resistant cultivars PNA647F4-56, NERICA4, NERICA10, WITA9 and IR47686-15-1-1; and three susceptible cultivars NERICA1, K85 and IR64. However, when resistance level was determined on the basis of percentage stunting at different times after inoculation, classification and rank changes between some cultivars occurred simply because partial resistance may develop at different times of the infection process to sufficiently eliminate further pathogenic challenge (Fig. 5). All the

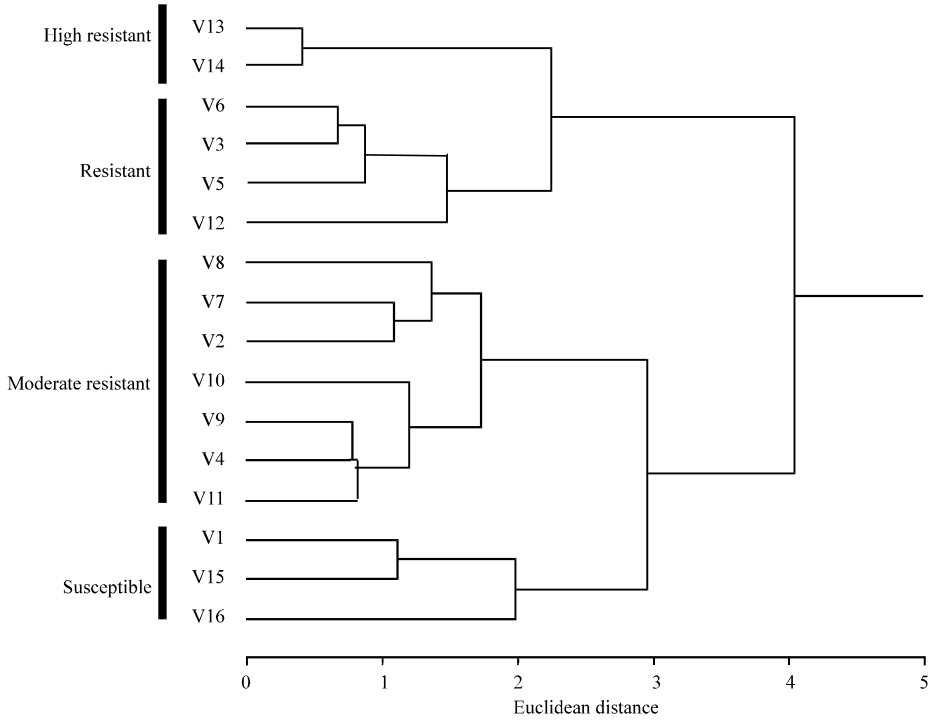


Fig. 4: Classification of varietal resistance based on disease severity data. Genotypes: $V_1$: NERICA 1, $V_2$: NERICA 4, $V_3$: NERICA 6, $V_4$: NERICA 10, $V_5$: ITA 257 (NARIC 1), $V_6$: ITA 325 (NARIC 2), $V_7$: TOG 5682, $V_8$: CG 14, $V_9$: WITA 9, $V_{10}$: PNA647F4-56, $V_{11}$: IR47686-15-1-1, $V_{12}$: GIGANTE, $V_{13}$: WAC 116, $V_{14}$: WAC 117, $V_{15}$: K 85, $V_{16}$: IR 64

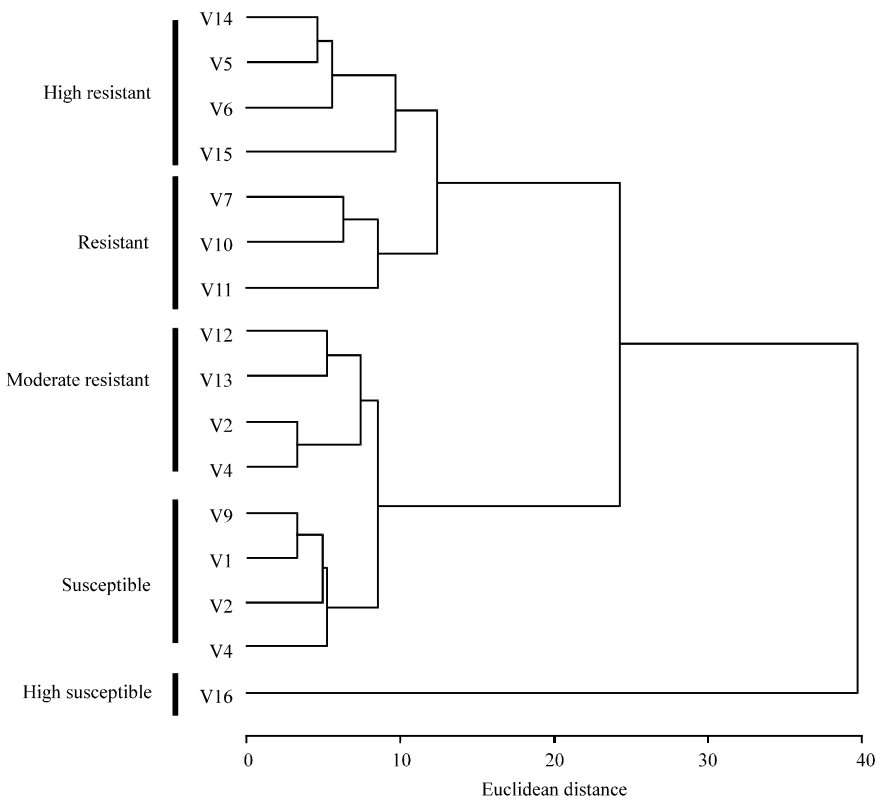


Fig. 5: Classification of varietal resistance based on percentage stunting data. Genotypes: $V_1$: NERICA 1, $V_2$: NERICA 4, $V_3$: NERICA 6, $V_4$: NERICA 10, $V_5$: ITA 257 (NARIC 1), $V_6$: ITA 325 (NARIC 2), $V_7$: TOG 5682, $V_8$: CG 14, $V_9$: WITA 9, $V_{10}$: PNA647F4-56, $V_{11}$: IR47686-15-1-1, $V_{12}$: GIGANTE, $V_{13}$: WAC 116, $V_{14}$: WAC 117, $V_{15}$: K 85, $V_{16}$: IR 64

isolates used were capable of inducing disease and would not be differentiated by looking at their levels of aggressiveness on rice plants. This finding supports the absence of pathotype variability of RYMV strains from Eastern Uganda.

## DISCUSSION

Studies of rice yellow mottle virus pathogenicity conducted with eight isolates and 16 rice cultivars revealed highly significant differences in disease response among cultivars and significant effect of isolate aggressiveness on diseases severity but not on percentage stunting. The current study could not identify cultivar by isolate interaction. Nevertheless, all isolates induced various degrees of plant invasion relative to the systemically damaging infection in the susceptible cultivar IR64. The severe effect of disease on the local variety K85 emphasizes the probable consequence of an outbreak of the disease in rice-growing areas of Eastern Uganda where susceptible lowland rice varieties are prevalent. As just noted, the absence of significant cultivar by isolate interaction was not a lack of virulence of the isolates per se but evidence that a great deal of variation in resistance of the evaluated cultivars was due to race-non-specific mechanisms (Pagán *et al*., 2007). Sequential deployment of race-non-specific resistance against infection was notable in five moderately resistant cultivars, PNA647F4-56, NERICA4, NERICA10, WITA9 and IR47686-15-1-1. Apart from delaying the already anticipated risk of these cultivars succumbing at early stages of

infection, this mechanism of resistance had little effect towards eliminating further pathogenic challenge on some cultivars since it was transient and only effective against few isolates (Ioannidou *et al*., 2000, 2003; MacKenzie *et al*., 2006). Conversely, majority of field resistant plants characterized by lack of visual symptoms and limited with-in host viral multiplication tend to possess a race-specific resistance mechanism that is effective against all isolates of a pathogen. This study identified two highly resistant cultivars (WAC116 and WAC117) and six resistant cultivars (ITA257, ITA325, NERICA6, GIGANTE, TOG5682 and CG14) that were able to reduce the effect of infection on their fitness (Jeger *et al*., 2006). To induce a disease, the virus must be able to rapidly replicate coupled by full systemic spread to distal parts of the plant (Hull, 2009). In response to infection, plant resistance genes are elicited by the loss or alteration of host factors that participated in replication, translation, movement or pathogenicity of the virus. In particular, host populations possessing recessive resistance genes are able to select for a higher level of virulence in pathogens compared to those lacking recessive resistance genes (Gandon and Michalakis, 2000). Over the past decade, scientists have discovered a single recessive gene *Rymv1* responsible for RYMV resistance in a few *O. glaberimma* accessions and in the *O. sativa* cultivars GIGANTE and BEKAROSAKA (Ndjiondjop *et al*., 1999; N'Guessan *et al*., 2001; Albar *et al*., 2007). Although it was efficient against isolates of major strains of the virus, the durability of *Rymv1-2* mediated-resistance was questionable during this study. This was supported by the fact that isolate Ug 2 readily circumvented the resistance of GIGANTE (Traorè *et al*., 2006; Fargette *et al*., 2002; Pinel-Galzi *et al*., 2007). This illustrates a major challenge that rice breeders are bound to face during the introgression of the *Rymv1-2* allele into the susceptible preferred local varieties currently grown in Eastern Uganda (Rakotomalala *et al*., 2008). The great majority of resistance breakdown is attributed to the appearance of novel pathotypes, generated during the co-evolutionary competition between pathogens with their hosts in an attempt to develop and accumulate invasive mechanisms that counter host resistance (Kaltz and Shykoff, 1998). Most reports consider virulence to be an inevitable consequence of within-host multiplication and virus accumulation in infected plant tissues, despite its negative effects on growth, life span and fecundity of the host, (Lenski and May, 1994; Ebert, 1998; Mackinnon and Read, 1999). For this reason, it is widely supposed that viral genes evolve in a highly specific way with an amino acid rate comparable to that of host genes in order to restrict significant damage of the host (Koonin and Gorbalenya, 1989). However, in this ever-continuing arms race for survival, it is quite possible that natural selection will favor virus strains with less virulence to have more time to exploit their hosts to attain virulence (Regoes *et al*., 2000). Therefore, virulence is more of a driving force in host-pathogen co-evolution modulating the important role of pathogens in shaping ecosystem composition and dynamics of their hosts (Ebert and Hamilton, 1996; Sacristan and Garcia-Arenal, 2008). In itself, RYMV is a highly variable plant virus that can rapidly explore the adaptive landscape by fixing favourable mutations (Opalka *et al*., 2000) so that different isolates within a given RYMV population may vary in their ability to overcome plant resistance at different times during and after infection (MacKenzie *et al*., 2006). Results from a separate study examining genetic diversity of RYMV isolates in Eastern Uganda, showed that the isolate Ug 2 possessed thirteen amino acid alterations in its coat protein compared to isolates Ug 3 and Ug 4 that were highly conserved though from the same locality (D. Ochola, Unpublished data). Therefore, it is conceivable that a resistance-breaking strain with a distinct virulence pattern exists at low frequencies among the prevalent strains in Eastern Uganda (MacKenzie *et al*., 2006). We suppose that a strict conservative selection pressure limited amino acid alterations in the coat protein of prevalent

strains for purposes of meeting specific functional requirements of the virus (Pinel-Galzi *et al.*, 2007). However, it must have restricted the rate of evolution of novel pathotypes in the region. Putting into consideration that we used only eight isolates in this study, it is likely that a more extensive sample would have revealed pathotypes that react with cultivars differently. Working on this assumption, we cannot overemphasize the magnitude of devastation that a pending RYMV epidemic could cause in Eastern Uganda where susceptible lowland rice varieties are still being cultivated. Plant breeders and virologists have a challenge of providing agronomically significant protection against adverse effects of a broad range of RYMV isolates throughout the commercial life of the cultivar. However, in view of the narrow genetic base of the available rice germplasm in Uganda (Mogga *et al.*, 2010) there is an urgent need for pyramiding different sources of resistance into the farmer preferred lowland varieties that possess wonderful culinary traits. Three *O. sativa japonica* cultivars (NERICA6, ITA257 and ITA325) and two *O. sativa indica* cultivars (WAC116 and WAC117) have been identified as potential sources of resistance for improving susceptible local rice varieties through backcross breeding.

## CONCLUSION

This current study has obtained information that could improve the efficiency of germplasm screening and deployment of durable resistant varieties for effective RYMV control. Not neglecting the inability of japonicas to sufficiently restrict virus diffusion and multiplication in susceptible indica varieties, there is a need for breeding strategies to use genetically different host populations that will increase divergence in selection pressure exerted by individual host genotypes on virus infection and within-host multiplication. Similarly, employing a combination of isolates with different levels of aggressiveness will exert sufficient prerelease challenge to avoid the future breakdown of then deployed resistant cultivars. In this way, the less aggressive isolates could differentiate between genotypes with low or moderate resistance while the aggressive isolates could differentiate resistant cultivars.

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