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Full Length Research Paper

Reaction of selected rice genotypes with monogenic resistance to the isolate of *Magnaporthe Oryzae* collected at Namulonge, Uganda

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The fungus *Magnaporthe oryzae* causes rice blast disease leading to substantial yield losses. This study aims at understanding the effectiveness of *R-genes* to the isolate of *M. oryzae* from Namulonge in Central Uganda to decide the incorporation of their carrier lines in local gene pyramiding programs. Eighty-three genotypes (73 monogenic resistance lines and 10 local varieties) were evaluated in two different experiments along with two susceptible and one resistant check in the screen house in a 10 × 8 alpha lattice design with two replications, inoculated by spraying, phenotyped at 7, 14 and 21 days after inoculation (DAI), and data on for disease severity, severity percentage, disease incidence, and relative area under disease progression were analyzed using the restricted maximum likelihood (ReML) in Genstat. The tested genotypes were significantly (P<0.001) different for the first, second and across experiments at 7, 14 and 21 DAI for all the measured traits indicating genetic variability among the tested germplasm for rice resistance to blast disease caused by the Namulonge isolate of *M. oryzae*. Twenty-five out of eighty-three (22.9%) genotypes had consistently low disease severity scores during the two experiments with a range of 0-3. The *R* genes *Pi3, Pi5 (t), Pi7 (t), Pi-b, Pik, Pi54, Pik-m, Pit, Pita, Pita-2, Piz, Piz-4, Piz-5* were considered effective to this particular isolate. The results of this study set the basis of a breeding program for rice resistance to blast disease caused by the Namulonge isolate of *M. oryzae* through gene pyramiding.

Key words: Rice, blast disease resistance, monogenic differential lines, reaction profiles, *R-genes.*

INTRODUCTION

Rice blast disease caused by the fungus *Magnaporthe oryzae* is one of the most devastating diseases to rice production in Uganda (Onaga and Asea, 2016). Presently, the rice blast pathogen has recently been reported to exhibit a high rate of production of new virulent races. Host plant resistance is the most cost effective and environmentally safe method of controlling rice blast disease. However, the pathogen has the ability to change into several different strains and overcome the resistance of the available resistant varieties making it

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difficult to breed for durable resistance due to the high variability among the strains of the pathogen as well as the favorable environmental conditions that favor the multiplication of the pathogen in the tropics (Bevitori and Raquel*,* 2014; Rajput et al*.,* 2017; Syakira et al*.,* 2016). Therefore, deployment of varieties with several R-genes that possess overlapped resistance is the most efficient method of controlling rice blast disease (Kumar et al., 2017). Currently there are over 100 R- genes that have been identified and mapped using DNA based markers (Devi et al., 2015) out of which 23 have been fully characterized, cloned and are now being deployed in varieties to confer resistance to the blast pathogen. In order to develop rice breeding lines with durable resistance, there is need to study the effectiveness of different R-genes to the strain of *M. oryzae* in a given geographical area. Lines with monogenic resistance to the pathogen (monogenic differential lines) have been identified as important genetic materials for determining the effectiveness of different R-genes. These were developed by the International Rice Research Institute (IRRI) using two susceptible genetic backgrounds namely the Japonica type Lijiangxin Tuan Heigu (LTH) and the indica type (CO39). Although several commercial varieties with broad spectrum resistance have been used as sources of resistance in Uganda, the *R-genes* have not been characterized. Uganda is known to have various different isolates of *M. oryzae* but the effectiveness of the R-genes against the different isolates has not been established. Niyongabo (2012) conducted an experiment using monogenic lines to determine the pathogenicity of the different isolates of *M. oryzae* collected from different parts of Uganda. In this test only five monogenic differential lines were used. In the same way the local resistant sources have been tested severally and showed resistance to a wide range of races. For instance a study by Mutiga et al. (2016) indicated that varieties Nerica 4 and Nerica 15 were resistant to >91% and >95% respectively of the isolates in Uganda. This is an indication that these locally released varieties could be carrying either a broad-spectrum resistance *R*-gene or maybe the resistance is quantitative in nature with several minor genes contributing. In addition, there is also a possibility that these local varieties could carry several major genes occurring on different chromosomes and interacting to cause resistance. Overall, in Uganda, the reaction profile of the different R-genes and their distribution in the local resistant rice varieties is not well known. Therefore, the objective of the study was to determine the reaction profiles of selected rice genotypes with monogenic resistance to the isolate of *M. oryzae* collected at Namulonge.

MATERIALS AND METHODS

The study was conducted to determine the effectiveness of resistance of 83 different rice genotypes to the Namulonge isolate of *M. oryzae.*

Site of study

The studies were carried out at the National Crops Resources Research Institute (NaCRRI) located at Namulonge, Wakiso District, Uganda. The station is about 30 km away from Kampala along latitudes 0°32'N and longitudes 32°53E'. The soils are ferralitic (red sandy and clay loams) and have a pH range of 4.9 to 5.0. The average annual rainfall is 1300 mm and maximum and minimum temperature of 28.5 and 13.0°C, respectively (Mugume et al., 2016).

Genetic plant materials

Eighty-three genotypes; 73 monogenic lines from IRRI and 10 local varieties were evaluated in the screen house. Among these, the genotype IR64 was used as a resistant check and Supa Soroti and WH13-3198(CO39) were used as the susceptible checks (Table 1).

Experimental design

The design was an alpha lattice with two replications and ten genotypes per block. Seed was planted in trays measuring 50 cm x 30 cm and filled with forest soil. Genotypes were planted in the trays at five plants per plot closely spaced with each row being a genotype at a spacing of 10 cm between rows. The three check varieties were planted after every four blocks in a replication. This experiment was carried out twice in the screen house at different times. The first experiment was run between June and July 2017 and the second experiment was run between September and October 2018 because of the differences in the outer environmental conditions during the two times of the season.

Isolation and preparation of *M. oryzae* isolate and Inoculation of test materials

This study involved the use of a virulent form of *M. oryzae* from Namulonge. Infected leaves were collected from the old rice field at Namulonge. The leaf samples were first washed with sodium hypochlorite and then in distilled water before being incubated in Petri dishes for about 30 minutes to enhance sporulation. After incubation, single spores were picked under a microscope with a pin-loop and placed on V8 media with rice bran. The fungal growth started within 7 days after spore placement on media (Mishra et al., 2015; Kulmitra et al., 2017). The fungus was then sub-cultured to obtain pure cultures that were stored at 4°C for preparation of inoculum for later studies (Figure 1). The isolate was multiplied on potato dextrose broth where the fungus grows a layer of conidia on top which turns black after 2-3 weeks. The suspension was made by blending the isolate and diluting it with distilled water at a concentration of 1.5×10^5 conidia/ml using a Neubauer haemacytometer under a compound microscope *(*Akagi et al., 2015). Two drops of 0.05% tween 20 were added to the inoculum to facilitate adhesion of the pathogen to rice leaves.

Pathogenicity test on the collected isolate of *M. oryzae* from Namulonge

The inoculum formed was first tested on a known susceptible genotype (Supa Soroti) to observe the true symptoms of rice blast disease. This was done in accordance with Koch's postulates for disease identification (Cohen, 1890). The inoculated plants showed the typical symptoms of rice blast disease after 7 days. The leaf samples from the diseased plants were then collected and then the strain of *M. oryzae* was re-isolated a second time which confirmed the first identification.

Table 1. Pedigrees of the 83 genotypes and the resistance genes (R-genes) in lines with known resistance and the origin of the lines with known resistance.

Entry no.	Genotype name	IRBL (Background)	*R-genes*	Origin
1	WH13-3198	CO39		IRRI
2	WH13-3199	IRBLb-IT13 [CO]	*Pib*	IRRI
3	WH13-3200	IRBLks-CO [CO]	*Piks*	IRRI
4	WH13-3201	IRBLk-Ku [CO]	*Pik*	IRRI
5	WH13-3202	IRBLk-Ka [CO]	*Pik*	IRRI
6	WH13-3203	IRBLKh-K3 [CO]	*Pi54*	IRRI
7	WH13-3204	IRBLkm-Ts [CO]	*Pik-m*	IRRI
8	WH13-3205	IRBLkp-K60 [CO]	*Pik-p*	IRRI
9	WH13-3206	IRBL1-LA [CO]	*Pi1*	IRRI
10	WH13-3207	IRBL7-M [CO]	*Pi7(t)*	IRRI
11	WH13-3208	IRBLsh-Ku [CO]	*Pish*	IRRI
12	WH13-3209	IRBLsh-S [CO]	*Pish*	IRRI
13	WH13-3210	IRBLsh-B [CO]	*Pish*	IRRI
14	WH13-3211	IRBLta-Ya [CO]	*Pita*	IRRI
15	WH13-3212	IRBLta-Me [CO]	*Pita*	IRRI
16	WH13-3213	IRBLta2-Pi [CO]	*Pita-2*	IRRI
17	WH13-3214	IRBLta2-Re [CO]	*Pita-2*	IRRI
18	WH13-3215	IRBLta2-IR64 [CO]	*Pita-2*	IRRI
19	WH13-3216	IRBLz5-CA [CO]	*Piz-5*	IRRI
20	WH13-3217	IRBLzt-IR56 [CO]	*Piz-t*	IRRI
21	WH13-3218	IRBL5-M [CO]	*Pi5(t)*	IRRI
22	WH13-3219	IRBLb-B [LT]	*Pib*	IRRI
23	WH13-3220	IRBLz5-CA [LT]	*Piz-5*	IRRI
24	WH13-3221	IRBL9-W[LT]	*Pi9*	IRRI
25	WH13-3222	IRBL3-CP4 [LT]	*Pi3*	IRRI
26	WH13-3223	IRBLa-Ze [LT]	*Pia*	IRRI
27	WH13-3224	IRBLk-Ka [LT]	*Pik*	IRRI
28	WH13-3225	IRBLkh-K3[LT]	*Pi54*	IRRI
29	WH13-3226	IRBLks-S [LT]	*Piks*	IRRI
30	WH13-3228	IRBLKs-zh [LT]	*Piks*	IRRI
31	WH13-3229	IRBL7-M [LT]	*Pi7(t)*	IRRI
32	WH13-3230	IRBLk*-NP [LT]	*Pik*	IRRI
33	WH13-3231	IRBLk*-DU [LT]	*Pik*	IRRI
34	WH13-3232	IRBLk*-F14 [LT]	*Pik*	IRRI
35	WH13-3233	IRBLk*-F25[LT]	*Pik*	IRRI
36	WH13-3234	IRBLk*-F66[LT]	*Pik*	IRRI
37	WH13-3235	IRBLta-CT2[LT]	*Pia*	IRRI
38	WH13-3236	IRBLta-K1[LT]	*Pita*	IRRI
39	WH13-3237	IRBLta-Zh [LT]	*Pita*	IRRI
40	WH13-3238	IRBLta2-P1 [LT]	*Pita*	IRRI
41	WH13-3239	LTH	*No R gene*	IRRI
42	WH13-3240	IRBLa-A	*Pia*	IRRI
43	WH13-3241	IRBLa-C	*Pia*	IRRI
44	WH13-3242	IRBLi-F5	*Piz-4*	IRRI
45	WH13-3243	IRBLks-F5	*Piz-4*	IRRI
46	WH13-3244	IRBLks-S	*Piks*	IRRI
47	WH13-3245	IRBLk-Ka	*Pik*	IRRI
48	WH13-3246	IRBLkp-K60	*Piz-4*	IRRI
49	WH13-3247	IRBLkh-K3	*Piz-4*	IRRI
50	WH13-3248	IRBLz-Fu	*Piz*	IRRI

Table 1. Contd.

51	WH13-3249	IRBLz5-CA	*Piz-5*	IRRI
52	WH13-3250	IRBLzt-T	*Pizt*	IRRI
53	WH13-3251	IRBLta-k1	*Pita*	IRRI
54	WH13-3252	IRBLta-CT2	*Pita*	IRRI
55	WH13-3253	IRBLb-B	*Pib*	IRRI
56	WH13-3254	IRBLt-K59	*Pit*	IRRI
57	WH13-3255	IRBLsh-S	*Pish*	IRRI
58	WH13-3256	IRBLsh-B	*Pish*	IRRI
59	WH13-3257	IRBL1-CL	*Pi1*	IRRI
60	WH13-3258	IRBL3-CP4	*Pi3*	IRRI
61	WH13-3259	IRBL5-M	*Pi5(t)*	IRRI
62	WH13-3260	IRBL7-M	*Pi7(t)*	IRRI
63	WH13-3261	IRBL9-W	*Pi9*	IRRI
64	WH13-3262	IRBL12-M	*Pi12(t)*	IRRI
65	WH13-3263	IRBL19-A	*Pi19*	IRRI
66	WH13-3264	IRBLkm-Ts	*Pikm*	IRRI
67	WH13-3265	IRBL20-IR24	*Pi20*	IRRI
68	WH13-3266	IRBLta2-Pi	*Pita2*	IRRI
69	WH13-3267	IRBLta2-Re	*Pita2*	IRRI
70	WH13-3268	IRBLta-CPI	*Pita*	IRRI
71	WH13-3269	IRBL11-Zh	*Pi11(t)*	IRRI
72	WH13-3270	IRBlz5-CA (R)	*Piz-5*	IRRI
73	IR65482-4-136-2-2	Indica	*Pi40*	IRRI
74	Nerica 4	CG14/WAB56-104	*Not known*	JICA/NaCRRI
75	Nerica 15	CG14/WAB56-104	*Not known*	JICA/NaCRRI
76	Nerica 14	CG14/WAB56-104	*Not known*	JICA/NaCRRI
77	NamChe 2	NM7-8-2-B-P-11-6	*Not known*	JICA/NaCRRI
78	K-38	Local lowland cultivar	*No R gene*	JICA/NaCRRI
79	IR64	(IR5657-33-2-1/IR2061-465-1-5-5)	*Not known*	JICA/NaCRRI
80	Supa Soroti	Local lowland cultivar	*No R gene*	JICA/NaCRRI
81	K85-8	Local lowland cultivar	*No R gene*	JICA/NaCRRI
82	Basmati 370	Unknown	*Not known*	JICA/NaCRRI
83	Nerica 6	CG14/WAB56-104	*Not known*	JICA/NaCRRI

IRRI stands for International Rice Research Institute and IRBL stands for IRRI breeding line.

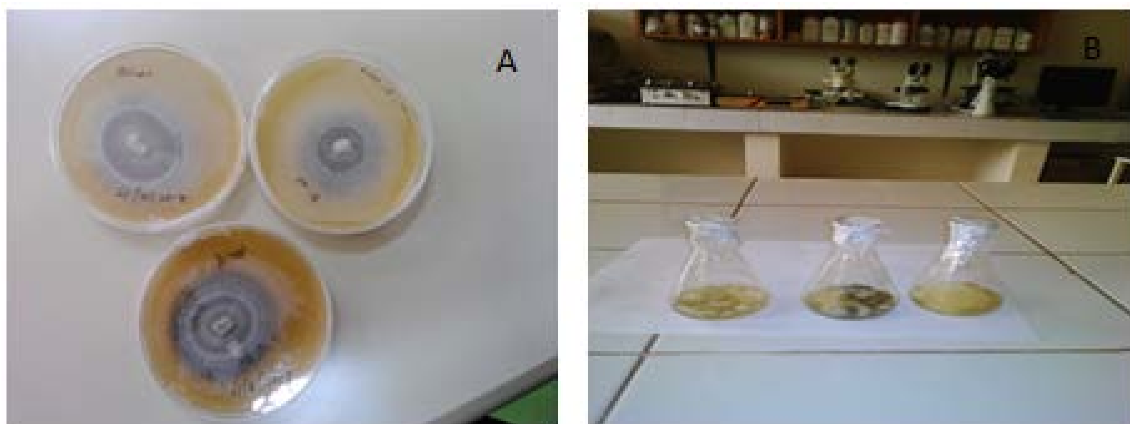

Figure 1. Pictures A and B showing the appearance of the *M. oryzae* isolate from Namulonge on Rice bran agar with V8 juice media and potato dextrose broth respectively.

Molecular characterization of the collected isolate of *M. oryzae* from Namulonge

The purpose of this activity was to confirm the pathogen species. The fungus was grown on Potato Dextrose Broth for two weeks at a temperature of 24°C. The DNA from the fungus was extracted using the CTAB (Cetyl Tri Methyl Ammonium Bromide) method (Thompson and Murray, 1980) with modifications by Naoto (unpublished data). The fungal DNA was subjected to a PCR reaction using forward and Reverse primers for internally transcribed spacers (ITS4) and (ITS5) respectively. The PCR products were first separated by a 2% agarose gel to observe whether the gene was amplified and the remaining PCR products were taken for sequencing to Bioneer Inc. Korea. The sequence results were BLAST searched in NCBI for the purpose of checking for similarity of the isolated pathogen with *M. oryzae* strain.

Inoculation of test materials

Inoculation of leaves on each genotype was done at 21 days after planting by spraying them with the suspension of density 31.5×10^5/ml until the upper most leaves were soaked (Akagi et al., 2015). The plants were then covered with a black polythene for 24 h to create darkness for the spores to attach and colonize the leaves (Mishra et al., 2015). Subsequently, the black polythene was replaced with a white polythene to act as a dew chamber at about 24-35°C and humidity of 80- 90% for about 48 h. Then, the white polythene was removed to let the fungus grow for 1 week before data collection.

Data collection

Each plant was scored for rice blast disease severity on the 0 to 9 scale (IRRI, 2013) where 0 signifies no lesions observed and 9 means 75% of the leaf area has lesion and also most of the leaves on the plant are infected. This was used to derive the variables; disease severity expressed as a percentage of the inoculated leaf area, percent disease incidence and, area under disease pressure stair (AUDPS) based on the standard Evaluation system (IRRI, 2013). Numerical ratings were converted to letter symbols where genotypes that scored 0 to 3 were recorded as R (resistant), 3.1 to 3.4 as MR (moderately resistant), 3.5 to 3.9 as MS (moderately susceptible) and 4 to 9 as S (susceptible) (Hayashi, 2011). Below are the formulae used to calculate derived parameters.

$$\textbf{Disease severity}\ \% = \frac{\textbf{sum of all the numerical ratings}}{\textbf{total number of ratings} \times \textbf{maximum rating}} \times \mathbf{100}$$

(Sabin et al., 2016).

The percent Incidence was also calculated using the formula below;

$$\textbf{Incidence} = \frac{\textbf{Number of infected plants}}{\textbf{Total number of plants observed}} \mathbf{x\ 100}$$

(Nishant et al., 2017)

To compare the level of resistance amongst the different monogenic differential lines, data were collected at 7, 14, and 21 days after inoculation giving three data readings and the Area Under Disease pressure stairs (AUDPS) was calculated from the formula described by Simko and Piepho (2012):

$$AUDPS = \overline{Y} \times \frac{D \times n}{n-1}$$

Where, $\overline{Y}$ is the average of the numerical ratings, n is the number of scores that were taken at equal intervals, however $D = n-1$, which makes the formula $AUDPS = \overline{Y} \times n$ The standardized and relative forms of AUDPS were also calculated by;

$$sAUDPS = \frac{AUDPS \times n-1}{Dn};$$

$$rAUDPS = \frac{sAUDPS}{Y\max}$$

where; $Y\max$ is the maximum possible observation of the disease.

Data analysis

The collected data were analyzed using Restricted Maximum Likelihood (ReML) in Genstat 18th edition (VSN International LTD, 2016). Genotypes were considered as a fixed factor while replications and blocks were random factors. However, where the lattice blocking was not effective for particular traits (where the block mean squares were less or equal to the residual mean square), data were analyzed using the unbalanced analysis of variance due to some missing genotypes that resulted from germination failure in some replications. The linear model used was follows;

$$\mathrm{Y_{ijk}} = \overline{\overline{\mathrm{Y}}} + \mathrm{G_i} + \mathrm{R_j} + \mathrm{B/R_{jk}} + \mathrm{e_{ijk}}$$

Where**;** $\overline{\overline{Y}}$ Is the Grand mean, G_i is the effect of genotype i, R_j the effect of replication j, B/R_{jk} is the effect of block k within replication j and e_{ijk} is the residual error.

Pearson's correlation was computed in order to test for consistency in the severity scores among the three days of evaluation after inoculation that is 7, 14 and 21 days after inoculation.

RESULTS

The result from the study showed that all the tested genotypes reacted differently to the isolate of *M. oryzae* collected from Namulonge which indicates genetic variation among the tested genotypes. R-genes in different backgrounds reacted quite differently to this isolate which shows some of these R-genes could be introgressed in the susceptible varieties to improve their resistance to blast.

Molecular characterization of the isolated pathogen from Namulonge

Results from BLAST (http://www.ncbi.nlm.nih.gov/blast) of the fungal sequence that was obtained from Bioneer Inc. showed that the pathogen that was sampled from Namulonge was 99% similar to *Magnaporthe oryzae.* The sequence ID was KT693184.1 and the query cover of 96% with an e-value of 0.0 from the NCBI. This confirmed the isolated blast pathogen from Namulonge based on the symptom caused as *M. oryzae*.

Table 2. Mean squares for the 83 genotypes for the first experiment for the disease ratings, leaf blast severity, severity percentage and incidence taken at 7, 14 and21 days after inoculation in the screen house at NaCRRI[1](June-July 2017A).

		Severity score				Disease severity percentage (%)				Disease incidence (%)			
SOV	DF	7 DAI	14 DAI	21 DAI	rAUDPS	7 DAI	14DAI	21 DAI	rAUDPS	7 DAI	14 DAI	21 DAI	rAUDPS
REP	1	16.06**	4.61*	2.41^{ns}	0.028^{ns}	538.26^{ns}	391.96^{ns}	333.3^{ns}	0.033^{ns}	803.6^{ns}	12223.9**	94.6^{ns}	0.201^{ns}
REP/block	14	1.12^{ns}	N/A	1.66*	0.0083^{ns}	117.82^{ns}	97.67^{ns}	228.7*	0.007^{ns}	459.3^{ns}	852.9*	N/A	0.043***
GENOTYPE	80-81	2.36***	5.51***	9.68***	0.059***	314.59***	603.81***	983.58***	0.0585***	1439.4***	974.8***	522.6***	0.065***
RESIDUAL	46.6-80	0.6	1.0	0.9	0.0071	74.9	85.7	123.4	0.0064	459.3	428.0	188.2	0.013
LEE	38.7-71.3	0.7	N/A	1.0	0.0074	83.7	89.6	138.5	0.0067	525.3	477.9		0.015
GM		1.9	3.5	3.7	0.309	18.8	30.8	42.4	0.3	79.6	84.6	91.7	0.8
CV (%)		43.4	27.8	27.3	27.8	48.6	30.7	27.7	26.5	28.8	25.9	15.0	14.4
SD		0.8	1.0	1.0	0.1	9.1	9.5	11.8	0.1	22.9	21.9	13.7	0.1

***, ** and * denote significance at P= 0.001, 0.01 and 0.05 respectively, ns=not significant, SOV-source of variation, LEE- lattice effective error, GM-grand mean, rAUDPS- relative area under disease pressure stairs, REP is replication and DF- degree of freedom, [1]National Crops Resources Research Institute.

Analysis of variance for both the combined and single experimental analyses for blast resistance parameters

The analysis of variance for the data of the first experiment (Environment 1) showed a highly significant difference in the reaction of genotypes to the isolate of *M. oryzae* from Namulonge in terms of disease severity, severity percentage and disease incidence (Table 2) (P<0.001). This reveals genetic variability among the tested genotypes. In addition, 41 genotypes (50.6%) were resistant with a score range of 0-3, 4 genotypes (4.8%) were moderately resistant with a range of 3.1-3.4, 4 genotypes (4.8%) were moderately susceptible with a range of 3.5-3.9 and 34 genotypes (40.9%) were susceptible with a range of 4-9 (Figure 2). In environment 1, genotype performance in terms of severity scores at 21 DAI ranged from 0.3 to 8.6. This shows genetic variation among the genotypes and therefore selection of resistant genotypes can easily be effective in breeding for resistance to blast. The genotypes that showed the highest in resistance include; WH13-3250(0.3), WH13-3220 (0.3), WH13-3248(0.5), WH13-3265(0.6), IR64 (0.6), WH13-3224(0.7), WH13-3245(0.7), WH13-3236(0.7), WH13-3212(0.8), WH13-3261(0.9) and Nerica 15(1.0) (Table 6). The genotypes that showed the highest susceptibility to the disease include; WH13-3205(6.8), WH13-3217(6.8), WH13-3252(7.1), K85-8(7.1), Supa Soroti (7.2), WH13-3256(7.3), WH13-3201(7.7), WH13-3223(7.8), WH13-3225(8.4) and WH13-3218(8.6) (Table 6).

Analysis of variance for the second experiment (environment 2) data also showed significance differences (P<0.001) among the genotypes in terms of disease severity, severity percentage (Table 3) and incidence. They also confirmed the presence of genetic variation among the genotypes for resistance to blast. In addition, 32 genotypes (38.8%) were resistant with a score range of 0-3, 9 genotypes (10.8%) were moderately resistant with a range of 3.1-3.4, 8 genotypes (9.6%) were moderately susceptible with a range of 3.5-3.9 and 31 genotypes (37.4%) were susceptible with a range of 4-9 (Figure 2). Disease severity scores at 21DAI ranged from 0.2 to 7.7. Genotypes that had the lowest mean score include; WH13-3270(0.2), WH13-3260(0.2), IR64 (0.1), Nerica 6 (0.0), NamChe 2(0.5), WH13-3247(0.5), WH13-3264(0.6), WH13-3259(0.7), WH13-3213(0.7) and WH13-3212(0.8). In the second experiment, genotypes that showed the highest susceptibility to rice blast include; WH13-3230(5.5), WH13-3262(5.7), WH13-3206(5.7), WH13-3217(5.8), WH13-3201(5.8), WH13-3205(6.0), WH13-3200(6.0), WH13-3218(6.5), WH13-3202(6.9) and Supa Soroti (7.7) (Table 6). Nineteen genotypes (22.9%) had consistently low disease severity scores ranging from 0-3 during the two experiments and hence their genes were considered to be effective against the isolate of *M. oryzae* from Namulonge. These include; WH13-3222, WH13-3259, WH13-3260, WH13-3219, WH13-3234, WH13-3245, WH13-3203, WH13-3264, WH13-3254, WH13-3212, WH13-3236, WH13-3213, WH13-3214, WH13-3248, WH13-3247, WH13-3216, WH13-3220, WH13-3249 and WH13-3270 (Table 6 and Figure 3). Their respective *R*-genes include; *Pi3, Pi5 (t), Pi7 (t),*

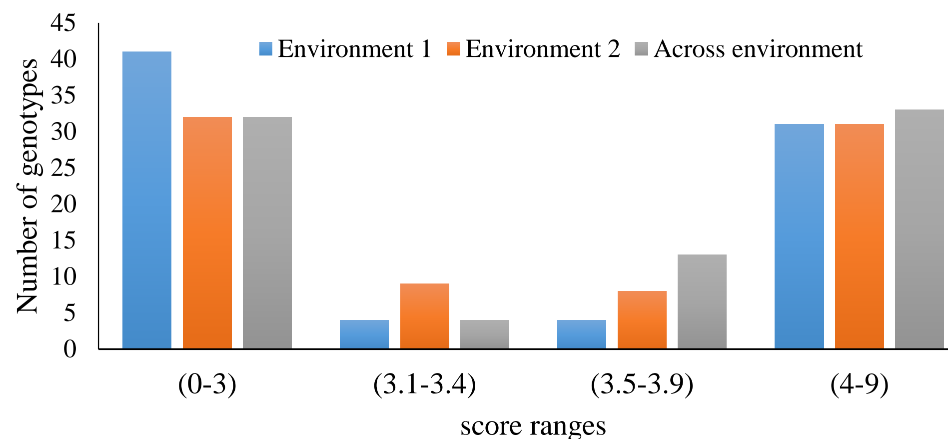

Figure 2. Frequency of genotypes for disease severity score in the two experimental conditions and across experiments in terms of severity scores.

Table 3. Mean squares for the second experiment on rice genotypes for leaf blast severity, severity percentage and incidence taken at 7, 14 and 21 days after inoculation in the screen house at NaCRRI[1] (September-October 2017B).

		Severity score				Disease severity %				Disease incidence %			
SOV	DF	7dai	14dai	21dai	rAUDPS	7dai	14dai	21dai	rAUDPS	7dai	14dai	21dai	rAUDPS
REP	1	13.9^{ns}	4.9*	1.71^{ns}	0.023^{ns}	242.3^{ns}	580.13*	1327.4^{ns}	0.033^{ns}	1953.3^{ns}	680.7^{ns}	136^{ns}	0.1897*
REP/block	14	1.23*	N/A	1.32^{ns}	1.50^{ns}	100.61^{ns}	N/A	839.1**	0.022**	897.1^{ns}	534.8^{ns}	123.8^{ns}	0.294^{ns}
GENOTYPE	82	2.087***	4.81***	10.22***	0.128***	217.64***	304.82***	530.46***	0.025***	1710.71***	900.38***	604.88***	0.087***
RESIDUAL	60.1-77	0.621	1.135	0.199	0.0114	85.42	91.94	283.1	0.01005	685.6	353.7	108.8	0.02764
LEE	38.7-71	0.72	N/A	1.00	0.0124	97.59	N/A	370.95	0.01	803.16	422.71	121.22	0.03
GM		2.05	2.77	3.55	0.39	16.72	25.83	40.79	0.26	80.13	91.49	95.83	0.86
CV (%)		41.39	38.46	28.17	28.55	59.10	37.12	47.22	40.59	35.37	22.47	11.49	19.93
SD		0.77	0.87	1.63	0.20	9.15	9.47	11.77	0.08	22.92	21.86	13.72	0.12

***, ** and * denote significance at P= 0.001, 0.01 and 0.05 respectively, ns=not significant, SOV-source of variation, LEE- lattice effective error, GM-grand mean, rAUDPS- relative area under disease pressure stairs, REP is replication and DF- degree of freedom, [1]National Crops Resources Research Institute.

Pi- b, Pik, Pik,Pi54, Pik-m, Pit, Pita, Pita, Pita-2, Pita-2, Piz, Piz-4, Piz-5, Piz-5, Piz-5 and *Piz-5* respectively. This shows that these particular genes were effective against this isolate. Some of the local varieties that consistently had low scores across experiments include; Nerica 6, NamChe 2, Nerica 15, Basmati 370 and IR64 (resistant check) while K85-8 and Supa Soroti were *oryzae* consistently susceptible. The combined analysis of variance revealed that, there was a significant difference (P<0.001) in the mean disease ratings for the isolate at the 7th, 14th and 21st day after

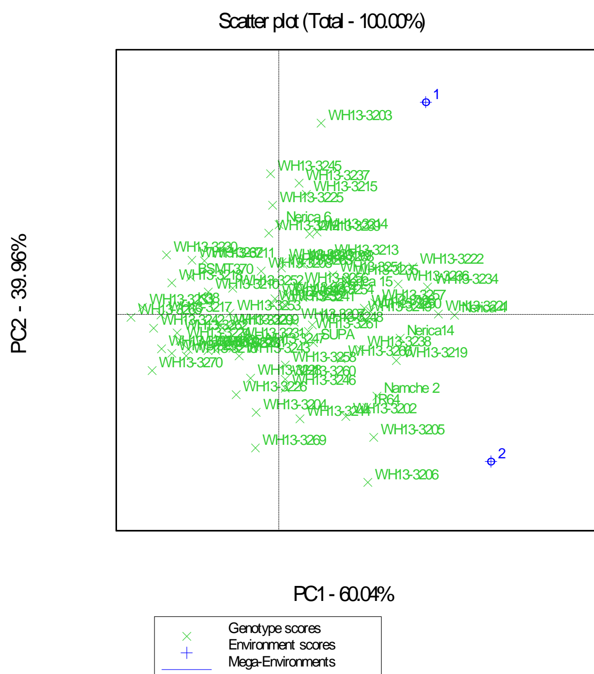

Figure 1. Scatter plot showing the rice blast scores for the 83 genotypes for experiment 1 and 2.

inoculation (DAI) for blast disease severity scores, severity percentage and the relative area under disease progress stairs (rAUDPS) (Table 4). This indicates genetic variability among the genotypes. However, genotypes at 21 days after inoculation of disease incidence did not show significant variation. In addition, there was a significant interaction between genotypes and experiment (Environment) at P<0.001 for all the variables at different intervals (Table 5 and Figure 4). This shows that there was a difference in the ranking of genotypes for disease ratings between the two environments. This indicates that the reaction of the genotypes to the disease is dependent on environmental conditions. The mean performance of the tested genotypes indicates that at 21DAI, 32 genotypes had scores between 0-3 (resistant), 4 genotypes had severity score means between 3.1-3.4 (moderately resistant), 13 genotypes scored between 3.5-3.9 (Moderately susceptible) and 33 genotypes had disease severity scores between 4-9 (susceptible) (Figure 2).

From Figure 2 under environments one and two, two peaks were observed at the extreme ends of the graphs where by most genotypes were observed at the end with scores of 0-3 considering them resistant and another peak was observed at the end with scores of 4-9 considering them susceptible. There were few genotypes that reacted as intermediate.

The graph illustrates that the environments in the two experiments were quite different. However, most of the varieties were located at the center of the two axes (Figure 3) showing that their performance across experiments was quite uniform. In addition, the two principle components adequately explain with a total of 100% the variation observed in the performance of the genotypes across the two experiments. Genotype WH13-3203 and WH13-3206 were highly unstable across the two experiments (environments).

The mean performance of genotypes was not different at 21 days after inoculation according to the boxplot where the mean disease score of the first experiment was 3.4 and the mean disease score of the second experiment was 3.6. This also shows that there was no difference in the results obtained from the two experiments, that is Experiment 1 and 2 which confirms uniformity of the conditions provided for the experiment.

DISCUSSION

This study establishes that understanding the reaction profiles of different R-genes under different backgrounds provides information that is useful for selection of R-genes that could be introgressed into susceptible varieties. From the analysis, the reaction profiles of monogenic differential lines were quite significantly different at a probability level of <0.001 which indicates genetic variability among tested genotypes. Similar results were obtained by Zelalem et al. (2017) who tested the reaction of Korean genotypes along with local rice genotypes and their reactions were quite different. In addition, most of the lines were made of single major R-genes but in susceptible backgrounds for example in the first experiment, monogenic differential line WH13-3203(score 2.4) was resistant; while WH13-3255 (score 8.4) was susceptible yet they both carry the same gene Pi54. However, these have different susceptible genetic backgrounds where the former has a background of CO39 while the latter has a background of LTH-Lijiangxin Tuan Heigu. This concurs with Quenouille et al. (2013) who suggested that the genetic background of a genotype determines its resistance or susceptibility.

Effectiveness of R-genes to the isolate of *M. oryzae* collected at Namulonge

In this study, the monogenic lines were screened in order to test their reaction to the Namulonge isolate of *M. oryzae* such that they could be used in the identification of resistance genes in the local resistant sources through allelism tests. At 21 days after inoculation, 25 genotypes (30.1%, 19 monogenic lines and 6 local resistant commercial varieties) were consistently resistant across experiments and 13 genes were considered effective against the isolate of *M. oryzae* from Namulonge. These genes include; *Pi3, Pi5 (t), Pi7 (t), Pi-b, Pik, Pi54, Pik-m, Pit, Pita, Pita-2, Piz, Piz-4, Piz-5.* Similar results were observed by Thuy Thi Thu Nguyen et al. (2015) where 26 monogenic lines were tested with 15 isolates. All the

Table 4. Mean squares for across experiments data of disease severity, severity percentage and incidence of 83 genotypes planted in the screen house at NaCRRI[1].

		Disease severity					Disease severity %					Disease incidence				
SOV	DF	7dai	14dai	21dai	rAUDPS	AUDPS	7dai	14dai	21dai	rAUDPS	AUDPS	7dai	14dai	21dai	rAUDPS	AUDPS
Experiment	1	0.48^{ns}	5.9^{ns}	6.2^{ns}	0.02^{ns}	48.7^{ns}	0.44^{ns}	2026.2^{ns}	217.2^{ns}	0.06^{ns}	17602^{ns}	17912^{ns}	3836^{ns}	1411.4^{ns}	0.003^{ns}	1990^{ns}
REP/Expt	2	8.6***	2.3**	14.7***	0.01***	18.9^{ns}	390.3*	290.1^{ns}	830*	0.03*	1866.5^{ns}	976.6^{ns}	6111.95***	8056.5***	0.2***	16528^{ns}
Genotype	82	2.9***	5.9***	12.2***	0.07***	48.7***	286.8***	620.8***	934.6***	0.17***	5086.5***	5084***	1103.2***	595^{ns}	0.1***	8642***
Genotype x Expt	78-79	1.2***	2.4***	3.8***	0.02***	18.97***	111.9***	284.4***	530.2***	0.03***	2486***	2486***	540^{ns}	467.5^{ns}	0.04***	4286^{ns}
Pooled error	62.7-152	0.78	0.44	1.8	0.01	22.7	91.67	91.1	328.3	0.02	1405.2	403.0	405.1	14359.2	0.02	116135.2
SD		0.8	1.0	1.1	0.2	2.7	9.6	9.5	16.0	0.1	28.4	28.3	21.3	119.8	0.2	341.7
GM		2.0	2.6	3.5	0.3	7.8	16.7	28.6	41.6	0.29	85.3	88.2	88.4	93.9	0.85	254.6
CV%		55.4	59.2	55.3	46.9	55.5	63.5	59.6	55.4	56.5	58.5	62.6	18.6	23.0	24.34	25.7
SED		0.8	1.1	1.0	0.1	2.2	7.5	11.9	16.3	0.1	24.9	24.9	11.6	10.8	0.10	32.7
VC genotypes		4.97	8.06	11.38	25.38	8.48	3.35	8.94	4.71	22.5	8.42	8.43	2.63	-0.93	5.13	-0.89
VC genotype x Expr		0.95	1.80	2.32	1.10	1.70	0.22	2.12	1.07	2.18	2.09	2.1	0.19	-0.97	0.91	-0.96

***, ** and * denote significance at P= 0.001, 0.01 and 0.05 respectively, ns=not significant, SOV-source of variation, LEE- lattice effective error, GM-grand mean, rAUDPS- relative area under disease pressure stairs, REP is replication and DF- degree of freedom, SED- standard error of the difference, SD- standard deviation, CV- coefficient of variation, expt- experiment [1]National Crops Resources Research Institute.

Table 5. Means for severity scores, severity percentage, incidence at 7, 14 and 21 days after inoculation with their area under disease pressure stair and relative area under disease pressure stair across experiments under screen house experiments planted at NaCRRI[1].

Genotype	**R-gene**	**Severity scores**					**Severity percentage**					**Incidence (%)**				
		7dai	**14dai**	**21dai**	**AUDPS**	**raudps**	**7dai**	**14dai**	**21dai**	**AUDPS**	**raudps**	**7dai**	**14dai**	**21dai**	**raudps**	**AUDPS**
WH13-3198	NO R gene	3.2	3.5	5.1	11.7	0.6	12.1	37.6	51.2	109.7	0.4	96.6	78.6	93.7	1.0	293.5
WH13-3199	Pib	1.7	3.4	4.4	9.3	0.5	26.0	39.8	56.1	100.4	0.4	77.0	100.8	100.3	0.9	279.2
WH13-3200	piks	3.4	4.5	6.1	13.9	0.7	33.8	45.4	62.4	167.5	0.5	102.2	101.2	99.9	1.0	300.5
WH13-3201	pik	3.9	4.1	6.8	14.8	0.8	30.2	48.9	75.5	151.2	0.5	99.5	98.2	100.1	0.9	273.6
WH13-3202	pik	3.1	4.4	6.1	12.7	0.7	34.8	49.2	55.4	130.4	0.4	100.6	100.2	99.8	0.9	271.1
WH13-3203	**Pi54**	**1.8**	**2.1**	**2.4**	6.1	0.2	19.0	24.3	28.9	61.9	0.2	83.1	87.4	99.8	0.9	272.0
WH13-3204	Pik-m	3.4	4.0	4.3	11.8	0.5	38.0	44.6	39.1	132.0	0.4	100.6	98.1	99.9	1.0	268.8
WH13-3205	Pik-p	3.3	4.6	6.4	14.3	0.7	27.5	58.1	64.0	151.9	0.5	93.5	97.6	99.8	1.0	292.4
WH13-3206	Pi1	1.5	4.4	4.5	11.0	0.5	35.7	39.3	58.9	115.8	0.4	48.6	71.7	100.0	0.8	218.6
WH13-3207	Pi7(t)	2.4	3.1	3.8	9.5	0.4	21.8	34.1	37.2	99.0	0.3	102.3	98.7	99.9	1.0	301.1
WH13-3208	Pish	2.3	2.9	4.2	8.0	0.4	25.5	32.7	53.5	108.4	0.4	99.4	99.4	99.9	0.9	275.0
WH13-3209	Pish	2.1	2.5	4.2	8.7	0.5	15.0	29.0	53.6	101.3	0.3	92.7	99.0	100.1	1.0	291.1
WH13-3210	Pish	2.3	3.2	4.2	9.7	0.5	16.1	33.0	52.7	109.5	0.4	98.0	100.9	99.7	1.0	306.5
WH13-3211	Pita	0.5	1.2	3.3	4.9	0.4	8.4	13.5	42.6	59.1	0.2	41.3	89.3	100.0	0.8	236.3
WH13-3212	**Pita**	**1.5**	**1.3**	**0.9**	**3.8**	**0.1**	**4.8**	**15.2**	**21.8**	**41.3**	**0.1**	**64.3**	**60.0**	**93.8**	**0.7**	**222.3**
WH13-3213	**Pita-2**	**2.1**	**1.9**	**1.2**	**5.4**	**0.1**	**14.9**	**20.0**	**42.7**	**74.1**	**0.2**	**44.5**	**73.5**	**99.9**	**0.8**	**229.7**

Table 5. Contd.

WH13-3214	**Pita-2**	**1.1**	**1.2**	**1.5**	**3.7**	**0.2**	**8.0**	**12.4**	**22.4**	**41.7**	**0.1**	**68.7**	**100.4**	**75.3**	**0.8**	**236.5**
WH13-3215	**Pita-2**	**1.7**	**2.2**	**2.9**	**6.8**	**0.3**	**18.1**	**22.8**	**37.6**	**74.2**	**0.2**	**72.6**	**85.6**	**96.4**	**0.8**	**254.4**
WH13-3216	**Piz-5**	**1.8**	**1.4**	**1.5**	**4.6**	**0.2**	**7.9**	**15.0**	**39.3**	**71.7**	**0.2**	**74.3**	**73.9**	**84.8**	**0.8**	**230.6**
WH13-3217	Piz-t	3.6	4.6	6.2	14.4	0.7	23.3	51.0	59.1	141.2	0.5	104.1	101.0	99.8	1.0	315.2
WH13-3218	Pi5(t)	3.0	4.2	7.6	15.1	0.8	36.5	48.8	78.8	161.4	0.5	97.2	96.7	100.0	1.0	293.8
WH13-3219	**Pib**	**2.0**	1.3	1.1	4.2	0.1	10.1	14.7	28.8	49.8	0.2	79.8	95.8	96.8	0.8	237.3
WH13-3220	**Piz-5**	**1.0**	**0.6**	**1.1**	**2.4**	**0.1**	**7.8**	**8.4**	**31.3**	**42.0**	**0.1**	**54.6**	**77.0**	**64.0**	**0.6**	**167.1**
WH13-3221	Pi9	2.4	4.4	4.7	11.5	0.5	16.2	41.2	53.8	111.1	0.4	89.0	82.4	93.6	1.0	294.1
WH13-3222	**Pi3**	1.7	2.2	2.0	5.9	0.2	14.2	23.3	35.6	70.9	0.2	85.9	87.9	83.6	0.9	260.1
WH13-3223	Pia	3.2	2.3	5.6	10.0	0.6	8.0	33.1	66.6	113.0	0.4	90.1	86.9	99.8	0.8	232.4
WH13-3224	**Pik**	**1.3**	**1.9**	**2.2**	**5.3**	**0.2**	**15.0**	**20.5**	**26.5**	**64.0**	**0.2**	**96.1**	**58.8**	**71.5**	**0.8**	**223.9**
WH13-3225	Pi54	3.5	3.6	6.4	13.6	0.7	34.7	42.6	72.5	152.5	0.5	97.3	96.0	99.8	1.0	293.2
WH13-3226	Piks	1.4	4.0	3.4	8.6	0.4	0.0	36.7	47.0	94.7	0.3	55.6	105.2	100.0	0.9	267.3
WH13-3228	Piks	1.9	3.3	4.3	9.6	0.5	27.1	23.8	43.0	71.2	0.3	75.0	100.9	99.7	0.7	205.9
WH13-3229	Pi7(t)	2.7	4.1	4.7	11.6	0.5	30.4	41.1	39.6	97.8	0.3	73.7	96.4	99.7	0.9	269.5
WH13-3230	Pik	2.5	3.3	3.6	8.4	0.4	31.4	33.2	36.8	71.2	0.2	69.5	101.8	92.6	0.8	238.5
WH13-3231	Pik	2.6	2.5	3.9	9.2	0.4	24.8	28.8	38.0	85.0	0.3	90.5	92.8	100.1	1.0	289.4
WH13-3232	Pik	2.1	2.8	3.8	7.2	0.3	17.7	28.3	36.0	68.6	0.2	82.5	89.4	85.3	0.8	246.7
WH13-3233	Pik	1.5	2.9	3.4	7.8	0.4	18.9	33.7	42.1	94.1	0.3	93.9	100.3	100.2	1.0	286.6
WH13-3234	**Pik**	**2.4**	**2.0**	**2.3**	**6.6**	**0.2**	**18.7**	**21.1**	**37.5**	**78.0**	**0.3**	**96.7**	**95.3**	**96.2**	**0.9**	**288.0**
WH13-3235	Pia	1.9	2.2	2.6	6.7	0.3	15.7	24.4	34.5	77.4	0.3	99.8	102.0	100.3	1.0	301.2
WH13-3236	Pita	2.1	2.0	1.6	5.6	0.2	10.9	21.4	35.9	64.6	0.2	71.9	85.3	100.0	0.8	247.7
WH13-3237	Pita	2.2	2.1	3.6	8.1	0.4	10.8	24.3	30.4	78.1	0.3	64.7	64.9	99.9	0.8	229.4
WH13-3238	Pita	1.4	4.9	3.0	9.4	0.3	11.9	27.2	59.4	101.4	0.3	88.3	97.8	96.8	0.9	279.3
WH13-3239	No R gene	1.4	3.7	4.6	11.4	0.5	9.8	41.0	60.7	95.7	0.4	66.2	95.9	100.2	0.8	226.8
WH13-3240	Pia	1.7	3.5	3.6	7.8	0.3	12.6	30.4	36.3	70.7	0.2	96.4	83.5	91.8	0.9	269.5
WH13-3241	Pia	2.1	3.1	4.5	9.6	0.5	18.4	27.9	48.1	98.9	0.3	97.3	100.4	100.2	1.0	291.5
WH13-3242	Piz-4	2.4	2.9	3.7	8.4	0.4	18.9	38.9	44.6	93.1	0.3	93.9	103.2	100.3	0.9	275.5
WH13-3243	Piz-4	2.0	3.0	5.0	10.4	0.6	14.8	38.7	54.6	117.4	0.4	96.9	79.6	93.7	1.0	290.1
WH13-3244	Piks	1.6	1.2	3.7	6.2	0.4	26.0	16.3	41.6	69.6	0.2	95.8	94.5	99.8	1.0	298.5
WH13-3245	**Pik**	**2.1**	**2.4**	**3.2**	**7.5**	**0.4**	**23.3**	**26.5**	**37.2**	**83.1**	**0.3**	**71.7**	**94.9**	**71.3**	**0.8**	**221.7**
WH13-3246	Piz-4	1.9	1.3	2.0	5.2	0.2	13.9	19.7	37.6	64.4	0.2	81.1	76.2	87.6	0.8	254.6
WH13-3247	Piz-4	2.0	1.6	2.9	6.4	0.3	10.6	17.2	36.4	60.2	0.2	71.4	75.3	100.0	0.8	234.9
WH13-3248	Piz	1.0	0.3	0.6	1.9	0.1	7.7	8.6	21.7	33.9	0.1	31.6	48.2	79.6	0.7	210.4
WH13-3249	Piz-5	1.1	1.5	1.9	4.3	0.2	10.0	16.7	29.4	54.2	0.2	72.8	89.8	96.6	0.9	262.4
WH13-3250	Pizt	0.9	1.8	1.1	3.8	0.1	18.5	10.8	23.5	35.6	0.1	40.6	73.6	87.3	0.7	227.3
WH13-3251	pita	2.9	3.2	4.4	10.4	0.5	21.5	35.4	40.4	100.2	0.3	90.8	98.7	99.8	1.0	283.9

Table 5. Contd.

WH13-3252	Pita	1.4	2.5	5.2	11.6	0.6	18.7	25.6	61.5	130.0	0.4	89.7	95.3	100.1	1.0	235.8
WH13-3253	Pib	2.2	2.9	4.5	9.5	0.5	23.2	32.1	52.5	106.4	0.4	100.9	99.8	100.0	1.0	291.7
WH13-3254	Pit	1.9	2.5	3.2	6.6	0.2	14.3	33.8	41.9	89.5	0.3	96.3	98.6	100.1	0.8	240.9
WH13-3255	Pish	2.1	3.6	3.6	9.4	0.4	21.1	40.1	49.9	113.3	0.4	77.4	86.1	94.7	0.9	257.2
WH13-3256	Pish	2.4	4.6	5.9	11.4	0.7	19.8	51.4	62.8	123.5	0.4	89.2	100.3	99.8	0.8	236.7
WH13-3257	Pi1	4.3	4.5	4.7	11.7	0.5	20.3	50.1	50.8	121.3	0.4	103.1	102.8	100.1	1.0	313.3
WH13-3258	Pi3	2.6	3.4	4.9	10.9	0.5	19.5	37.6	62.0	128.4	0.4	98.0	99.6	100.1	1.0	294.7
WH13-3259	**Pi5(t)**	1.9	1.6	2.3	5.8	0.3	10.1	18.4	30.3	57.2	0.2	73.9	87.3	92.7	0.9	262.6
WH13-3260	**Pi7(t)**	1.8	1.6	1.5	4.7	0.2	8.8	20.0	30.9	64.4	0.2	87.5	101.2	100.1	1.0	293.9
WH13-3261	**Pi9**	**0.5**	**1.0**	**1.3**	**2.9**	**0.1**	**5.6**	**16.4**	**31.1**	**40.3**	**0.1**	**85.3**	**48.1**	**86.6**	**0.6**	**194.4**
WH13-3262	Pi12(t)	1.4	1.3	2.4	5.1	0.3	18.1	15.6	22.9	44.9	0.1	63.0	60.7	64.3	0.6	188.3
WH13-3263	Pi19	2.8	3.4	5.8	12.1	0.6	21.7	39.7	66.8	141.4	0.5	97.6	97.2	99.9	1.0	294.2
WH13-3264	**Pikm**	**1.1**	**1.3**	**2.3**	4.6	0.3	6.7	11.4	21.2	41.3	0.1	74.2	82.3	100.1	0.8	252.5
WH13-3265	Pi20	1.2	0.7	0.5	2.5	0.1	14.1	6.8	14.0	27.4	0.1	43.1	54.6	76.2	0.6	169.6
WH13-3266	Pita2	2.4	3.1	4.3	9.9	0.4	16.6	34.1	36.2	98.0	0.3	98.7	98.3	99.8	1.0	303.1
WH13-3267	Pita2	0.5	3.2	3.2	3.6	0.2	3.7	34.2	49.2	31.7	0.1	46.7	102.1	49.8	0.4	103.5
WH13-3268	**Pita**	**1.4**	**1.8**	**1.7**	**5.0**	**0.2**	**14.7**	**20.5**	**21.5**	**51.9**	**0.2**	**90.7**	**96.8**	**92.9**	**0.6**	**177.7**
WH13-3269	Pi11(t)	2.6	4.1	5.1	11.9	0.6	29.1	36.4	26.5	95.8	0.3	99.1	103.3	100.3	1.0	300.0
WH13-3270	**Piz-5**	**0.6**	**0.9**	**1.6**	**2.5**	**0.0**	**2.3**	**13.8**	**23.1**	**40.2**	**0.1**	**55.2**	**81.9**	**99.8**	**0.8**	**222.3**
WH13-3273	**Pi40**	**1.3**	**0.6**	**1.3**	**3.2**	**0.2**	**7.5**	**12.0**	**44.0**	**62.7**	**0.2**	**68.1**	**90.0**	**100.0**	**0.9**	**256.1**
Basmati 370		1.7	2.4	2.8	7.1	0.3	12.5	27.0	28.1	73.2	0.3	100.5	103.0	100.2	1.0	302.1
IR64		0.3	0.0	0.2	0.6	0.0	8.8	1.7	1.1	3.4	0.0	4.8	5.9	29.4	0.2	47.0
K38		2.3	2.7	2.2	7.3	0.2	6.6	29.6	27.4	78.7	0.3	78.6	102.9	99.8	0.9	286.1
K85-8		2.5	4.3	5.0	12.4	0.6	13.6	46.1	61.9	129.3	0.4	97.9	97.1	99.9	1.0	288.8
Namche 2		0.4	1.4	1.2	3.0	0.1	10.1	12.9	34.1	50.8	0.2	4.0	59.4	95.3	0.5	153.8
Nerica 15		1.0	1.0	1.2	3.6	0.1	6.5	12.7	19.5	40.8	0.2	57.5	66.0	80.8	0.8	237.2
Nerica 4		1.2	1.1	1.4	3.6	0.2	9.2	13.8	26.3	47.8	0.2	91.2	90.0	100.1	1.0	261.5
Nerica 6		1.1	1.7	1.5	4.2	0.2	5.7	20.8	26.1	52.9	0.2	27.0	94.9	95.4	0.7	217.1
Nerica14		2.4	2.3	2.7	7.3	0.3	10.5	25.3	33.4	80.3	0.3	98.8	93.4	100.0	1.0	297.2
Supa Soroti		2.7	4.2	5.0	11.7	0.5	15.7	39.0	54.7	143.9	0.4	93.5	99.8	100.3	1.0	284.5
GM		2.0	2.6	3.3		0.4	16.7	28.3	41.6	85.3	0.3	79.7	88.5	93.9	0.9	254.6
Minimum		0.3	0.0	0.2	0.6	0.0	0.0	1.7	1.1	3.4	0.0	4.0	5.9	29.4	0.2	47.0
Maximum		4.3	4.9	7.6	15.1	0.8	38.0	58.1	78.8	167.5	0.5	104.1	105.2	100.3	1.0	315.2
$LSD_{0.05}$		1.1	1.6	2.1	4.3	0.2	10.5	16.8	22.9	49.6	0.2	24.9	23.1	21.5	0.2	64.8

rAUDPS is the relative area under disease pressure stairs, $LSD_{0.05}$ is the least significant difference at P=0.05and dai is days after inoculation, GM is the grand mean, Rxn- reaction LTH-Lijiangxin Tuan Heigu, R-resistant, MR-moderately resistant, MS-moderately susceptible and S- susceptible, *National Crops Resources Research Institute.

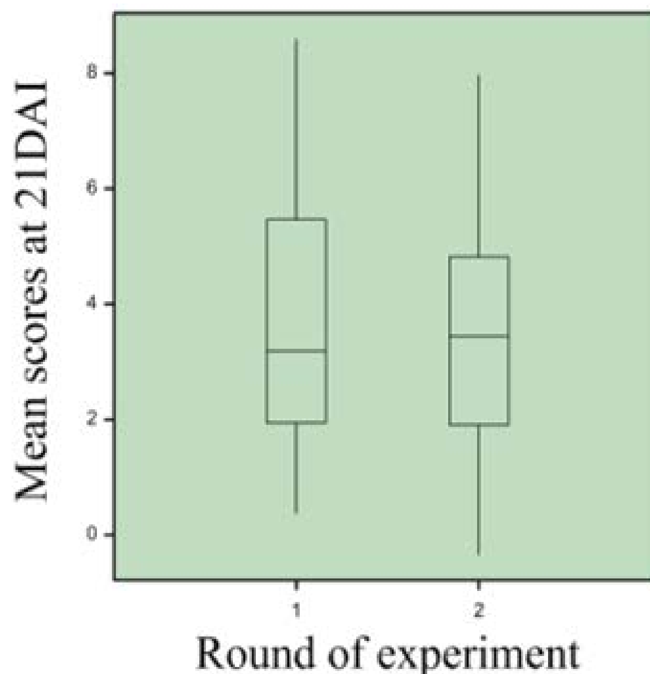

Figure 4. Box plot comparing mean performance of the genotypes for severity scores at 21 days after inoculation during the two rounds of the experiment.

26 monogenic lines were resistant to the 3 out of the 15 isolates. *R -genes* that are carried by these monogenic lines evaluated in the current study can be used to identify the different *R-genes* within the local resistant sources through allelism study such that they could be used as sources of resistance. In addition, Wang *et al.*, (2013) observed similar results where *Pi9*, *Pi19*, *Piz*, *Piz-5*, *Piz-t*, *Pi12*(t), *Pi5*(t), and *Pi54* were observed to be resistant to 4 isolates of *M. oryzae* making these R-genes broad spectrum resistant in nature.

Results in this study showed some of the monogenic lines to have the same level of resistance based on the scores because they basically carry the same R-genes though they have different backgrounds. However, only two monogenic lines were selected based on their mean performance at 21 days after inoculation. In addition, the two monogenic lines carry broad spectrum resistant genes (*Pi54* and *Pi7t*) that were already characterized. In the same way the local resistant sources show a wide range of resistance; for instance a study by Mutiga et al. (2016) indicates that Nerica 4 and Nerica 15 were resistant to >91 and >95% respectively of the isolates in Uganda. This is an indication that these local genotypes could either be carrying a broad-spectrum resistance *R*-gene or maybe the resistance is quantitative in nature with several minor genes contributing. In addition, there is also a possibility that these local varieties could be having several major genes occurring on different chromosomes and interacting to cause resistance.

Disease incidence

Disease incidence was high even for some of the resistant genotypes because disease symptoms were observed in these genotypes as only five plants were evaluated; and the genes they carry can permit formation of sporulating lesions but have the ability to stop proliferation of the disease probably because they could have some components of partial resistance. For example, a study by Yasuda et al., (2015) showed that some genes like *Pi21*, *Pi35* and *Pi36* have the ability to stop hyphae growth of the fungus and therefore reduce the elongation of the lesion because they have smaller lesion sizes. However, none of the resistance genes had an effect on the penetration frequency of the rice blast fungus. In addition, according to recent studies, these R-genes produce proteins that recognize pathogen effectors differently and their immunity is associated with the PAMP triggered immunity which mainly causes hypersensitive reactions hence the observed small lesions (Schweizer, 2007; Schulze-Lefert and Panstruga, 2011; Strugala et al., 2015). This could be an explanation for the high incidence observed even amongst the resistant monogenic lines

Variation among the tested genotypes

The variation observed amongst the genotypes

Table 6. Means of severity scores at 7, 14 and 21 days after inoculation with their and relative area under disease pressure stair across experiments for experiment 1 and experiment 2 at NaCRRI[1].

Genotype	Back ground	R-genes	Environment 1				Rxn	Environment 2				Rxn
			7DAI	14DAI	21_DAI	Raudps		7DAI	14DAI	21_DAI	Raudps	
WH13-3198	CO39	no R gene	3.3	3.6	4.8	0.5	S	3.2	3.3	5.3	0.6	S
WH13-3199	IRBLb-IT13 [CO]	*Pib*	1.2	3.6	4.4	0.5	S	2.3	3.1	4.5	0.5	S
WH13-3200	IRBLks-CO [CO]	*piks*	3.2	4.0	7.0	0.7	S	2.7	4.2	6.0	0.7	S
WH13-3201	IRBLk-Ku [CO]	*pik*	4.5	5.5	7.7	0.9	S	3.3	2.8	5.8	0.7	S
WH13-3202	IRBLk-Ka [CO]	*pik*	3.1	4.4	5.5	0.6	S	3.1	4.4	6.9	0.8	S
WH13-3203	IRBLKh-K3 [CO]	*Pi54*	1.2	1.5	2.0	0.2	R	1.8	3.0	3.1	0.2	R
WH13-3204	IRBLkm-Ts [CO]	*Pik-m*	3.5	4.2	4.7	0.5	S	3.4	3.8	3.8	0.4	MS
WH13-3205	IRBLkp-K60 [CO]	*Pik-p*	0.6	3.0	6.8	0.8	S	3.6	3.8	6.0	0.7	S
WH13-3206	IRBL1-LA [CO]	*Pi1*	-0.1	1.9	4.8	0.4	MR	3.0	3.9	5.7	0.6	S
WH13-3207	IRBL7-M [CO]	*Pi7(t)*	2.8	3.5	4.8	0.5	S	2.0	2.8	2.8	0.3	R
WH13-3208	IRBLsh-Ku [CO]	*Pish*	2.2	3.9	4.9	0.5	S	2.3	2.0	3.6	0.2	MS
WH13-3209	IRBLsh-S [CO]	*Pish*	2.2	3.2	5.4	0.6	S	2.0	1.8	3.1	0.3	MR
WH13-3210	IRBLsh-B [CO]	*Pish*	1.2	2.7	4.9	0.5	S	1.8	2.5	3.6	0.4	MS
WH13-3211	IRBLta-Ya [CO]	*Pita*	0.3	1.6	3.1	0.3	MR	0.7	0.8	3.4	0.4	MR
WH13-3212	IRBLta-Me [CO]	*Pita*	1.0	1.2	0.9	0.1	R	0.9	0.8	1.5	0.1	R
WH13-3213	IRBLta2-Pi [CO]	*Pita-2*	0.9	1.2	1.8	0.2	R	0.7	2.6	2.9	0.1	R
WH13-3214	IRBLta2-Re [CO]	*Pita-2*	1.2	1.0	1.4	0.1	R	1.1	1.4	1.6	0.2	R
WH13-3215	IRBLta2-IR64 [CO]	*Pita-2*	1.1	1.9	2.5	0.3	R	2.3	2.5	3.2	0.4	MR
WH13-3216	IRBLz5-CA [CO]	*Piz-5*	2.2	2.3	2.0	0.2	R	0.9	0.7	1.2	0.1	R
WH13-3217	IRBLzt-IR56 [CO]	*Piz-t*	3.3	4.1	6.8	0.8	S	3.2	3.2	5.8	0.6	S
WH13-3218	IRBL5-M [CO]	*Pi5(t)*	2.4	3.7	8.6	1.0	S	3.7	3.3	6.5	0.7	S
WH13-3219	IRBLb-B [LT]	*Pib*	1.1	2.5	5.1	0.1	R	0.9	1.4	1.5	0.1	R
WH13-3220	IRBLz5-CA [LT]	*Piz-5*	1.0	0.5	0.5	0.1	R	1.0	0.6	1.7	0.2	R
WH13-3221	IRBL9-W[LT]	*Pi9*	1.2	2.2	4.5	0.5	S	2.7	3.6	4.8	0.5	S
WH13-3222	IRBL3-CP4 [LT]	*Pi3*	1.4	1.9	5.0	0.2	R	2.0	2.1	2.5	0.2	R
WH13-3223	IRBLa-Ze [LT]	*Pia*	2.8	4.0	7.8	0.9	S	2.4	1.8	3.4	0.4	MR
WH13-3224	IRBLk-Ka [LT]	*Pik*	0.4	1.1	3.3	0.1	R	1.4	3.2	3.7	0.4	MS
WH13-3225	IRBLkh-K3[LT]	*Pi54*	0.7	3.8	8.4	0.9	S	3.1	3.9	4.4	0.5	S
WH13-3226	IRBLks-S [LT]	*Piks*	1.4	2.3	4.4	0.4	MR	2.3	3.7	4.6	0.5	S
WH13-3228	IRBLKs-zh [LT]	*Piks*	1.5	2.1	3.9	0.4	MS	2.9	4.2	5.4	0.6	S
WH13-3229	IRBL7-M [LT]	*Pi7(t)*	2.5	2.4	4.0	0.4	S	3.1	4.4	4.9	0.6	S
WH13-3230	IRBLk*-NP [LT]	*Pik*	1.9	2.2	4.0	0.3	R	3.2	3.5	5.5	0.6	S
WH13-3231	IRBLk*-DU [LT]	*Pik*	2.1	1.6	2.9	0.2	R	3.0	4.2	5.1	0.4	S
WH13-3232	IRBLk*-F14 [LT]	*Pik*	1.2	1.3	4.0	0.3	R	1.2	3.4	4.0	0.4	S

Table 6. Contd.

WH13-3233	IRBLk*-F25[LT]	*Pik*	1.9	2.4	2.7	0.3	R	2.4	2.1	3.1	0.3	MR
WH13-3234	IRBLk*-F66[LT]	*Pik*	2.3	1.9	1.4	0.2	R	2.0	1.8	2.0	0.2	R
WH13-3235	IRBLta-CT2[LT]	*Pia*	1.9	1.5	6.0	0.4	MR	2.4	3.2	3.2	0.3	R
WH13-3236	IRBLta-K1[LT]	*Pita*	0.9	0.7	0.8	0.1	R	1.0	0.6	2.4	0.3	R
WH13-3237	IRBLta-Zh [LT]	*Pita*	3.4	3.7	5.1	0.6	S	1.6	2.9	4.1	0.5	S
WH13-3238	IRBLta2-P1 [LT]	*Pita*	1.2	6.2	1.9	0.2	R	0.5	3.0	3.6	0.4	MS
WH13-3239	LTH	*no R gene*	2.3	3.4	5.5	0.6	S	2.0	3.5	3.8	0.2	MS
WH13-3240	IRBLa-A	*Pia*	1.4	3.5	5.2	0.4	MS	1.2	2.4	3.0	0.3	R
WH13-3241	IRBLa-C	*Pia*	2.1	3.0	5.9	0.7	S	2.9	3.1	4.4	0.5	S
WH13-3242	IRBLi-F5	*Piz-4*	1.6	2.9	3.0	0.3	R	1.2	2.2	3.6	0.4	MS
WH13-3243	IRBLks-F5	*Piz-4*	1.9	4.9	6.7	0.7	S	1.5	1.5	5.2	0.6	S
WH13-3244	IRBLks-S	*Piks*	1.6	0.8	3.9	0.2	R	3.4	4.0	5.5	0.6	S
WH13-3245	IRBLk-Ka	*Pik*	0.8	0.8	0.7	0.1	R	1.3	1.8	2.2	0.2	R
WH13-3246	IRBLkp-K60	*Piz-4*	2.5	0.9	1.9	0.2	R	2.6	2.8	4.5	0.5	S
WH13-3247	IRBLkh-K3	*Piz-4*	1.3	0.3	1.3	0.1	R	1.0	0.5	0.5	0.1	R
WH13-3248	IRBLz-Fu	*Piz*	1.0	0.0	0.6	0.1	R	1.0	1.3	1.5	0.2	R
WH13-3249	IRBLz5-CA	*Piz-5*	1.3	1.7	2.3	0.3	R	1.4	1.8	3.1	0.2	R
WH13-3250	IRBLzt-T	*Pizt*	0.5	0.5	0.3	0.0	R	3.5	3.0	4.6	0.5	S
WH13-3251	IRBLta-k1	*pita*	2.3	3.4	4.1	0.5	S	1.8	2.0	3.4	0.4	MR
WH13-3252	IRBLta-CT2	*Pita*	0.9	3.0	7.1	0.8	S	2.0	2.6	4.5	0.5	S
WH13-3253	IRBLb-B	*Pib*	2.4	3.1	4.6	0.5	S	3.0	4.0	1.1	0.1	R
WH13-3254	IRBLt-K59	*Pit*	1.9	2.3	2.7	0.3	R	1.3	1.8	1.8	0.1	R
WH13-3255	IRBLsh-S	*Pish*	2.4	5.4	6.0	0.7	S	2.5	3.8	4.5	0.5	S
WH13-3256	IRBLsh-B	*Pish*	2.3	5.5	7.3	0.8	S	2.6	4.0	4.0	0.3	R
WH13-3257	IRBL1-CL	*Pi1*	4.5	5.0	6.7	0.7	S	2.6	2.7	3.8	0.4	MS
WH13-3258	IRBL3-CP4	*Pi3*	2.7	4.0	5.9	0.7	S	2.3	2.3	3.3	0.4	MR
WH13-3259	IRBL5-M	*Pi5(t)*	1.6	0.9	4.0	0.1	R	1.0	1.3	0.7	0.1	R
WH13-3260	IRBL7-M	*Pi7(t)*	2.5	1.8	2.3	0.3	R	0.7	1.0	-0.1	0.0	R
WH13-3261	IRBL9-W	*Pi9*	1.9	1.0	1.0	0.1	R	0.9	0.4	3.2	0.4	MR
WH13-3262	IRBL12-M	*Pi12(t)*	1.9	2.1	1.7	0.2	R	2.9	3.4	5.7	0.6	S
WH13-3263	IRBL19-A	*Pi19*	2.7	3.5	5.8	0.6	S	1.3	1.4	3.0	0.3	R
WH13-3264	IRBLkm-Ts	*Pikm*	0.8	1.3	2.5	0.2	R	1.5	0.9	0.6	0.0	R
WH13-3265	IRBL20-IR24	*Pi20*	0.9	0.5	5.5	0.1	R	2.5	2.6	4.9	0.4	S
WH13-3266	IRBLta2-Pi	*Pita2*	2.2	3.6	3.8	0.4	MS	0.5	3.2	4.5	0.3	S
WH13-3267	IRBLta2-Re	*Pita2*	1.7	3.7	-	-	S	1.0	0.0	3.4	0.4	MR
WH13-3268	IRBLta-CPI	*Pita*	1.4	0.9	-	-	S	2.6	4.1	-	-	S
WH13-3269	IRBL11-Zh	*Pi11(t)*	-	-	-	-	-	0.9	0.4	5.2	0.6	S

Table 6. Contd.

WH13-3270	IRBlz5-CA (R)	*Piz-5*	1.7		2.0	0.1	R			-0.2	0.0	R
WH13-3273	Indica	Pi40	-	-	-	-	-			1.3	0.2	R
BASMTI370	Local		1.7	2.3	2.7	0.3	R	1.6	2.6	3.0	0.3	R
IR64			0.3	0.1	0.7	0.1	R	0.2	0.0	-0.1	0.0	R
K38	Local cultivar		3.4	3.9	2.5	0.3	R	1.3	1.6	1.9	0.2	R
K85-8	Local cultivar		3.7	5.3	7.1	0.8	S	1.3	3.4	4.2	0.3	S
Namche 2			0.0	2.6	2.0	0.2	R	0.9	0.3	0.5	0.0	R
Nerica 15	CG14/WAB54-104		1.5	1.5	1.2	0.1	R	0.5	0.6	1.4	0.1	R
Nerica 4	CG14/WAB54-104		1.5	1.1	1.7	0.2	R	1.0	1.1	1.2	0.1	R
Nerica 6	CG14/WAB54-104		1.3	3.0	2.9	0.3	R	0.9	0.4	0.0	0.0	R
Nerica14	CG14/WAB54-104		2.6	2.2	3.0	0.3	R	2.1	2.4	2.4	0.3	R
Supa Soroti	Local cuktivar		3.7	5.3	7.2	0.8	S	1.7	3.2	7.7	0.9	S
Minimum			-0.1	0.0	0.3	0.0		0.5	0.0	-0.2	0.0	
maximum			4.5	6.2	8.6	1.0		3.7	4.4	7.7	0.8	
LSD0.05			0.8	0.9	1.6	0.2		1.7	2.1	3.2	0.2	

rAUDPS is the relative area under disease pressure stairs, LSD is the least significant difference at a significant level of 5% and Dai is days after inoculation, GM is the grand mean, -denotes missing due no germination, Rxn- reaction LTH-Lijiangxin Tuan Heigu, R-resistant, MR-moderately resistant, MS-moderately susceptible and S- susceptible, [1]National Crops Resources Research Institute.

(monogenic differential lines) for leaf blast during the two experiments in the analysis is due to the presence of genetic variation among these genotypes which is brought about by the difference in the response of the different R-genes to this particular strain of *M. oryzae* from Namulonge. This response also depends on the genetic background from which they were made (Odjo et al., 2017). This shows that genetic improvement can be done on the genotypes especially the susceptible ones using the resistant ones (Zewdu et al., 2017). Similar results were observed by Haggag and Tawfik (2014) Sabin et al. (2016) and Zewdu et al. (2017). Ghazanfar (2009) reported screening of about 113 rice lines and he obtained only one resistant line. Nine of the lines tested were moderately resistant and 77 lines were recorded as susceptible.

Genotype by environment (experiment) significantly varied at (P<0.001) significance level. A probable explanation to this could have been variation in the growth factors in the screen house that affects sporulation that are responsible for the growth of blast lesions including temperature and relative humidity (Faivre *et al.,* 2013; Zhang *et al.,* 2014). However, according to analysis of variance across environments most of the variation observed was due to genotype effect looking at the fact that the variance component of genotype at 21 days after inoculation (11.38) was higher than the variance component across experiment (2.32) (Table 5). This is an indication that most the variations observed among the tested genotypes were primarily due to genetic variability.

Genotype by environment (experiment) significantly varied at (P<0.001) significance level. A probable explanation to this could have been variation in the environmental factors that are responsible for the growth of blast lesions including temperature and relative humidity (Faivre-Rampant., 2013; Zhang et al., 2014). However, according to analysis of variance across environments most of the variation observed was due to genotype effect looking at the fact that the mean square of genotype at 21 days after inoculation (12.6) was higher than the error mean square across experiment (1.8) and the mean square of genotype by round of experiment (3.9) (Table 5). This is an indication that most of the variations observed were primarily due to genetic variability among the genotypes. The CVs were moderately high for some evaluations because of the differences in readings in the two replications. This is brought about by variations in the screening conditions during the two screenings that influence the growth of the pathogen. These conditions have severally been documented to be

important in the growth of the conidia (Zhang et al., 2014).

The nested factor of replication within experiment (Table 5) had significant effects which came about probably due to the variation in the growth conditions for the conidia within the different replications in terms of temperature and relative humidity within the different replications. These are factors that influence sporulation and lesion formation (Castejon'-Munoz, 2008).

CONCLUSION AND RECOMMENDATIONS

In this present study, monogenic lines with R-genes Pi3, Pi5 (t), Pi7 (t), Pi-b, Pi54, Pik-m, Pit, Pita, Pita-2, Piz, Piz-4, and Piz-5 emerged as highly effective to the isolate of *M. oryzae* from Namulonge. In comparison with the local genotypes, NamChe 2, Nerica 15, Nerica 6 and IR64 (resistant check) were equally highly resistant. On the other hand, R-gene *Pi5(t)* from monogenic line WH13-3218 emerged as the most susceptible. Supa Soroti and K85-8 were equally very susceptible to this particular isolate. Therefore, assessment of the reaction of monogenic differential lines to isolates *M. oryzae* is important in that it assists one to understand the reaction profiles of different R-genes to a particular strain of the fungus such that appropriate breeding strategies for resistance to this devastating rice disease can be carried out.

The strain from Namulonge has been identified as one that has the highest virulence as compared to other strains in Uganda. Therefore, studying the reaction of the newly introduced monogenic differential lines is important such that incase of presence of the resistant genes, they can be introgressed into local susceptible varieties in Uganda to improve them for resistance to blast disease or better still increase the durability of the local resistant varieties as single broad spectrum resistant genes or by gene pyramiding. However, there is a need to challenge the monogenic differential lines with various isolates from different parts of the country in order to select out those that truly have broad spectrum resistance against the different isolates. In addition, the resistance genes found in the local varieties need to be further characterized.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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