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Inheritance of resistance to brown spot disease in upland rice in Uganda

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Brown spot disease caused by *Bipolaris oryzae* [Breda de Haan (Shoem.)] is one of the most important diseases affecting rice (*Oryza sativa* L.) worldwide. Host plant resistance is considered an effective, cheap and environment friendly means of managing this disease. Nine rice genotypes with varying resistance levels were crossed in a full diallel mating design including reciprocals and parents. Parents, reciprocals and F_2 progenies were evaluated in an alpha lattice design in the screen house and field trials at the National Crops Resources Research Institute in Uganda in 2013-2014. The objectives of the study were to determine the mode of inheritance for resistance to brown spot disease and characterize segregation patterns of specific F_2 progenies. Significant (P ≤0.001) variation for brown spot resistance occurred among the tested genotypes. The general combining ability (GCA) and specific combining ability (SCA) effects of brown spot disease scores were both significantly different (P≤0.001), indicating that both additive and non-additive genetic effects were present. There was, however, a predominance of non-additive genetic effects in the genetic control of brown spot resistance as shown by low estimates of baker's ratio (0.29) and narrow sense coefficient of genetic determination (0.24), implying that progeny performance could not be predicted from parents GCA effects as it was better only in specific crossing combinations. Segregation patterns also indicated that resistance to brown spot was controlled by one or two dominant genes. The reciprocal effects for the crosses were significantly different (P ≤ 0.05), suggesting that cytoplasmic genetic effects modified the expression of resistance. Care should, therefore, be taken when selecting female parents during hybridization. Family-based breeding programs would also be effective for improving resistance to brown spot in rice varieties adapted to Uganda.

Key words: Diallel analysis, gene action, non-additive effects, *Oryza sativa*, segregation patterns

INTRODUCTION

Rice is an important economic and food security crop in Uganda (MAAIF, 2008, 2009). Demand for the crop has

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Table 1. Rice parental genotypes used in full diallel crosses for brown spot resistance in Uganda

Entry code	Origin	Resistance designation
NERICA 4	Africa Rice	Highly resistant
NERICA 10	Africa Rice	Resistant
[1]E 20	NaCRRI- Namulonge	Resistant
[2]E 22	NaCRRI- Namulonge	Resistant
K5	Local - Uganda	Moderately resistant
P4R1	NaCRRI- Namulonge	Susceptible
NERICA 1	Africa Rice	Susceptible
TXD 306	Tanzania	Susceptible
PAKISTAN (UP)	Pakistan (Jica)	Susceptible

[1]E20 Pedigree:NM7-20-4- B-P-1-1, crosses (IRAT 325/WAB 365-B-1H1-HB); [2]E22 Pedigree: NM7-22-11- B-P-1-1, crosses (WAB 450-1-BL1-136-HB /WAB 450-B-136-HB).

increased in the past decade due to a rapid growth in population, urbanization and shifts in consumption patterns. This trend has been further stimulated by several economic and political initiatives, within Uganda and the East African region, which have transformed the rice value chain (MAAIF, 2009; Kilimo Trust, 2014). In the year 2002, the area under production was 80,000 hectares, with yield of 120,000 MT milled rice and average yield of 1.5 MT/ha for milled rice (FAOSTAT, 2016). By 2014, the area under production had risen to 95,000 hectares, with yield of 249,470 MT and average yield of 2.5 MT/ha for milled rice (FAOSTAT, 2016). This implies that the area under production increased by up to 15.8%, while yield increased by 51.9%. Currently, production is estimated at 260,000 MT, leaving a gap of 40,000 tonnes (Lamo, 2016). At a sufficiency level of 86.7%, Uganda is thus making great strides in meeting both local and regional demand (Kilimo Trust, 2014; Lamo, 2016). Sadly, however, these gains have been made mainly by increasing the area under production since productivity still falls far below the yield potential for developed nations at 8 t/ha (5 t/ha for upland production). This shortfall has been attributed to a number of factors, including pests and diseases, drought and water shortage and declining soil fertility (Kilimo Trust, 2014).

Brown spot is one of the major diseases limiting rice production in Uganda (Awio et al., 2015). The local varieties grown by farmers in Uganda are susceptible to the disease (Kawube et al., 2005; Odogola, 2006). In 2011, brown spot was ranked as the third most important disease after *Rice yellow mottle virus* (RYMV) disease and leaf blast (Adur et al., 2011). The disease affects both rain-fed and upland rice production, causing losses in both yield and grain quality (Singh and Singh, 2000; Savary et al., 2005). Heavily infected grains are rendered unsuitable for human consumption (Barnwal et al., 2013) and yield reductions as high as 45% occur with severe infection and 12% with moderate infection (IRRI, 1983). Disease management is possible through use of appropriate agronomic practices, pesticides, biological control and resistant varieties (Shabana et al., 2008). Sources of resistance to brown spot are available in Asia and Africa. These sources can be used for the development of resistant varieties for release to farmers (Yaqoob et al., 2011; Nneke, 2012). Differences in varietal susceptibility to brown spot (Datnoff and Lentini, 2003) and diversity within *Bipolaris oryzae* species (Kamal and Mia, 2009), however, pose a challenge to breeding for resistance. In order to overcome this problem, the use of local germplasm and pathogen isolates is required.

While varieties preferred by farmers in Uganda are NERICA 1, K5 and TXD 306 exhibit desirable attributes that include aroma and high yielding ability; these varieties are mostly susceptible to brown spot. This study was therefore done to determine the mode of gene action conditioning the inheritance of resistance to brown spot and characterize the segregation patterns of specific F_2 progenies. Knowledge of the mode of gene action from this study will help in the introgression of genes for disease resistance to local farmer preferred genotypes.

MATERIALS AND METHODS

Study area

The study location was the National Crops Resources Research Institute (NaCRRI) in Central Uganda. The Institute is located at 0° 32" N and 32° 37" E and stands at an elevation of 1150 m above sea level within the Lake Victoria crescent agro-ecological zone. It receives average annual precipitation of 1200 mm, with peaks from April to May and September to October. Two cropping seasons are experienced, namely, season A covering the period from March to July and season B covering August to December. The study reported was conducted during season 2013 A, 2013 B and 2014 A.

Development of breeding population

Nine rice genotypes with varying levels of resistance to brown spot (Table 1) were grown and crossed in a full diallel mating design with

Table 2. F2 rice populations used in studying segregation patterns for brown spot resistance in Uganda.

Crossed parents	Resistance status of parents
TXD 306 × NERICA4	S × R
NER 1 × NERICA4	S × R
E22 × PAKISTAN	R × S
E20 × NERICA1	R × S
NER 4 × TXD 306	R × S
NER 4 × NERICA1	R × S
E20 × PAKISTAN	R × S

S = Susceptible; R = Resistant.

parents and reciprocals in a screen house. The diallel mating design was used because the genotypes under study showed reaction to brown spot disease at varying levels, from highly resistant, resistant, moderately resistant to susceptible scores. Forty (40) F_1 progenies were advanced to F_2 in the screen house. The parents, reciprocals and F_2 populations were evaluated for brown spot resistance in the field.

Experimental design and management

The F_2 plants, including the reciprocals and their parents, were planted in the field at NaCRRI using an alpha-lattice design with two replications at a spacing of 5 × 10 cm (one plant per hill). About 20 to 60 F_2 plants from crosses between resistant and susceptible families were selected to be used in studying segregation patterns (Table 2). The plants were supplied with 25 kg/ha of nitrogen two weeks after transplanting. At two weeks, the plants were also inoculated mechanically with a *Bipolaris oryzae* isolate prepared in the laboratory (Motlagh et al., 2006) using a conidia suspension (1×10^5 conidia ml^{-1}) (Sato et al., 2008). To increase surface absorption, 1% Tween-20 was incorporated into the conidia suspension (Motlagh et al., 2006). Standard cultural practices like watering and hand weeding were carried out regularly.

Data collection

Disease severity was scored on five plants per plot at full panicle stage for every genotype following the standard evaluation system (SES) for rice (IRRI, 2002). The rating scale varies from 1 (highly resistant) to 9 (highly susceptible).

Statistical analysis

The data were analyzed in GENSTAT 14, using model 1, method 1 of Grifffings (1956) to determine the effects of general combining ability (GCA) and specific combining ability (SCA). Parents were considered as fixed since they were chosen considering their levels of resistance to brown spot. The Diallel analysis model 1 and method 1 were adjusted to reduce the error effect due to missing crosses following Bernado (2006). Combining ability analysis was therefore performed on 9 parental genotypes and 40 crosses (28 parental combinations and 12 reciprocals).

The statistical linear model for this analysis was:

$$Yijk = \mu + gi + gj + sij + rij + eijk$$

where μ = overall mean, gi = GCA effect of the i^{th} parent, gj = GCA effect of the j^{th} parent, sij = SCA effect of the ij^{th} genotype, rij = reciprocal effect of the ij^{th} genotype, and eijk = the environmental effect of the ijk^{th} observation.

The ratio of GCA variance to SCA variance was estimated according to Baker (1978) as:

$$X = 2\sigma^2gca / (2\sigma^2gca + \sigma^2sca)$$

where σ^2gca = GCA variance components and σ^2sca = SCA variance components.

The estimates of broad and narrow sense coefficient of genetic determination were calculated on family mean basis using the following formulas as outlined by Dabholkar (1992).

$$BSCGD = (2 \times \sigma2GCA + \sigma2SCA)/ (2 \times \sigma2GCA + \sigma2SCA + \sigma2e/r)$$

$$NSCGD = (2 \times \sigma^2_{GCA})/ (2 \times \sigma^2_{GCA} + \sigma^2_{SCA} + \sigma^2_e/r)$$

where σ^2_{GCA} and σ^2_{SCA} are variance components estimates of GCA and SCA, respectively, σ^2_e is the variance due to experimental error and r is the number of replications.

The combining ability effects of parents (GCA) and crosses (SCA) were tested for deviation from zero by using two tailed t-tests as described by Singh and Chaudhary (2004) and Dabholkar (1992). The GCA effect of each individual parent was divided by the standard error of GCA, while the SCA effect of each cross combination was divided by the standard error of SCA.

Data collected on disease severity were interpreted using frequency distribution of trait measurements (histogram) to study the segregating F_2 populations in order to understand the nature of inheritance and number of genes influencing brown spot resistance (Fehr, 1987). The distinct phenotypic classes and segregation ratios were compared with theoretical ratios using the Chi-square goodness-of-fit test. For analysis, highly resistant, resistant and moderately resistant genotypes were grouped as resistant, and all genotypes with higher scores were grouped as susceptible (Ongom et al., 2012) to best fit the reduced phenotypic classes due to epistasis effects exhibited and enable determination of the departure of observed frequencies from hypothesized frequencies. A chi-square (χ^2) probability was used, where χ^2 was significant at $P < 0.05$, the fitted model was rejected.

RESULTS

Genetic variability, combining abilities and heritability

Results of analysis of variance for resistance to brown

Table 3. Analysis of variance for combining ability for brown spot disease scores in F_2 populations and their parents.

Source	df	MS	F_{calc}	Variance component
Crosses	39	0.94***	4.94	
GCA	8	1.37***	7.21	0.14
SCA	19	1.11***	5.84	0.66
Reciprocal	12	0.39*	2.05	0.10
Error	39	0.19		
Baker's Ratio			0.29	
NS-CGD			0.24	
BS-CGD			0.83	

*, ***Statistically significant at α = 0.05, 0.001 respectively; the calculation for coefficient of genetic determination are based on entry means.

Table 4. General combining ability effects for brown spot resistance for parents.

Parents	Parental mean	GCA effects	SE_{gca}
K5	5.7	0.53***	0.066
PAKISTAN	7.0	0.35***	0.044
TXD306	7.0	0.16^{ns}	0.056
E20	3.0	-0.09^{ns}	0.036
E22	3.0	-0.23**	0.044
NER 1	5.7	0.38 ***	0.033
NER 4	4.3	-0.63***	0.030
NER 10	3.7	-0.42***	0.056
P4R1	7.0	0.31 **	0.056

**, ***Highly significant at α = 0.01, 0.001 respectively; ^{ns}Not significant at α = 0.05.

spot revealed highly significant differences (P ≤ 0.001) among parents and F_2 progenies tested (Table 3). General and specific combining ability mean squares were very significant (P ≤0.001); reciprocal mean squares were also highly significant (P ≤ 0.001). The Baker's ratio was low (0.29) while the estimate of broad sense coefficient of genetic determination was high (0.83). The transmissibility of brown spot resistance from parents to progenies, as shown by the estimate of narrow sense coefficient of genetic determination, was low (0.24).

Estimates of general combining ability effects

Parental lines K5, PAKISTAN, P4R1 and NER 1 had significant positive GCA effects (Table 4). In contrast, the lines E22, NER 4 and NER 10 had significant negative GCA effects (P ≤ 0.01, 0.001, 0.001, respectively). The line E20 had negative non-significant GCA effects, while TXD 306 had non-significant positive GCA effects.

Estimates of specific combining ability effects

The crosses K5 × NER 1, TXD 306 × NER 4, NER 4 × P4R1, PAKISTAN × E20, E 22 × E 20 and NER 1 × NER 10 had significant negative SCA effects (P ≤ 0.05, 0.01, 0.01, 0.001 respectively) (Table 5). The crosses TXD 306 × NER 1, E20 ×x K5, NER 10 × E20, NER 1 × P4R1 and E22 × NER 4 displayed significant positive SCA effects.

Reciprocal effects

Significant (P<0.05) negative reciprocal effects were realized with the NER 10 × E22 cross (Table 6). The cross NER 4 × E20 and NER 4 × NER 1 showed significant positive reciprocal effects at P<0.05.

Segregation pattern of brown spot reaction in F_2 progeny of selected crosses

F_2 progenies from the crosses showed distinct phenotypic classes for brown spot scores (Table 7). Analysis of segregation ratios revealed that crosses TXD 306 × NER4, NER 1 × NER 4, NER 4 × NER 1 and E22 × PAK conformed to the 3:1 ratio. Crosses E20 × NER 1 and NER 4 × 306 conformed to the 9:7 ratio, while cross E20

Table 5. Specific combining ability effects for brown spot resistance in F_2 rice population.

Parents	K5	PAK	TXD306	E20	E22	NER 1	NER 4	NER10	P4R1
					Female				
K5			-0.19^{ns}						
PAK					- 0.12^{ns}				0.00^{ns}
TXD306				- 0.23 ^{ns}			-1.03**		
E20	0.73*	-1.76***				0.38^{ns}	0.22 ^{ns}		
E22	-0.13^{ns}		0.23^{ns}	-1.51***		0.01^{ns}	1.35***		
NER 1	-0.74*	-0.06^{ns}	0.63^{ns}				-0.59^{ns}		
NER 4	0.27^{ns}	2.11***						0.05^{ns}	
NER10	0.06^{ns}			0.68*	0.15^{ns}	-1.13**			
P4R1				0.45^{ns}		1.14**	-1.02**		

*, **, ***Significant at α = 0.05, 0.01, 0.001 respectively; ^{ns}Not significant at α = 0.05; PAK: Pakistan upland; NER: NERICA.

Table 6. Reciprocal effects for brown spot resistance in F_2 populations.

Parents	K5	PAKS	306	E20	E22	NER1	NER 4	NER10	P4R1
K5	-	-	-	-	-	-	-	-	-
PAKS	-	-	-	-	-	-	-	-	-
306	-	-	-	-	-	-	-	-	-
E20	-	-	-	-	-	-	-	-	-
E22	-	0.17^{ns}	-	-	-	-	-	-	-
NER 1	-	- 0.50^{ns}	- 0.33^{ns}	- 0.50^{ns}	-	-	-	-	-
NER 4	-	-	0.50^{ns}	0.67*	-	0.67*	-	-	-
NER10	-	-	-	-	- 0.67*	-	0.17^{ns}	-	-
P4R1	-	- 0.17^{ns}	-	- 0.17^{ns}	-	-	- 0.17^{ns}	-	-

*Significant at α = 0.05; ^{ns}Not significant at α = 0.05; PAKS: Pakistan upland; 306: TXD 306; NER: NERICA.

Table 7. Phenotypic segregation ratios for resistance to brown spot in F_2 population.

F_2 populations			Observed		Expected		Goodness-of-fit	
Cross	No.P	Type	R	S	R	S	χ^2	Prob.
Best fit ratio 3:1								
TXD 306 × NER 4	60	S × R	50	10	45	15	2.222^{ns}	0.136
NER 1 × NER 4	60	S × R	50	10	45	15	2.222^{ns}	0.136
NER 4 × NER 1	30	R × S	27	3	28	2	3.60^{ns}	0.058
E22 × PAK	60	R × S	50	10	45	15	2.222^{ns}	0.136
E20 × NER 1	18	R × S	11	7	14	4	1.852^{ns}	0.174
NER 4 × 306	21	R × S	12	9	16	5	3.571^{ns}	0.058
E20 × PAK	18	R × S	16	3	18	6	3.555^{ns}	0.136
Best fit ratio 9:7								
E20 × NER 1	18	R × S	11	7	10	8	0.172^{ns}	0.678
NER 4 × 306	21	R × S	12	9	12	9	0.006^{ns}	0.934
Best fit ratio 15:1								
E20 × PAK	18	R × S	16	2	17	1	0.725^{ns}	0.394

No. P = No of plants: χ^2 = Chi- square test; R, S resistant and susceptible parents respectively; PAK: Pakistan; NER: NERICA; ns: non-significant at p≤ 0.05 probability level

× PAK conformed to the 15:1 ratio.

DISCUSSION

Genetic variability

Results of analysis of variance for resistance to brown spot revealed significant differences among parents, reciprocals and F_2 progenies. This shows there is adequate genetic diversity among the parents and their respective crosses that could be used in population development. According to Bertan et al. (2007) superior recombinant genotypes are generated when there is significant variability in the parental genotypes.

Heritability and combining ability

The general and specific combining ability mean squares of brown spot disease scores were highly significant (P ≤0.001) indicating that both additive and non-additive genetic effects were important in the genetic control of brown spot resistance. The relative importance of additive over non-additive genetic effects as shown by Baker's ratio was low (0.29), indicating the predominance of non-additive genetic effects over additive genetic effects; hence, a low predictability of progenies performance from parents GCA effects. The progeny performance in this set of crosses was only better in specific crossing combinations and therefore could not be predicted for a wide range of crosses. The estimates of broad sense coefficient of genetic determination, which measures the proportion of phenotypic variance that is due to genetic causes, were high (0.83). This indicates that the environment did not play a key role in the expression of resistance to brown spot. The estimates of narrow sense coefficient of genetic determination, which measures the proportion of phenotypic variance that is due to transmitted genetic effects, were low (0.24) suggesting that the contribution of non-additive variance to the total genetic variance was key in controlling resistance to brown spot in this set of crosses.

Combining ability effects

Dabholkar (1992) and Singh and Chaudhary (2004) reported that parents with significant GCA effects in the desired direction for a character of interest are the best for hybridization. Parents E22, NER 4 and NER 10 had desirable significant negative GCA effects indicating they contributed to brown spot resistance in F_2 progeny. The parent K5, which was moderately resistance, had significant positive GCA effect indicating it contributed towards susceptibility to brown spot disease. The susceptible parent TXD 306 had a positive non-significant GCA effect indicating that it contributed average effects towards susceptibility that were not meaningful. The susceptible parents PAKISTAN and P4R1 had significant positive GCA effect indicating that these parents contributed susceptibility in F_2 progenies as expected. The parent NERICA 1 had non-significant positive GCA effects indicating it contributed average effects towards resistance that were not meaningful. The parent E20 had non-significant negative GCA effects indicating it did not contribute to resistance. Therefore, NER 4, E22, and NERICA 10 were the best combiners for resistance to brown spot. These parents can be used in the breeding programme to introduce resistance genes to locally adapted rice germplasm.

Crosses TXD 306 × NER 4, NER 1 × K5, E20 × PAKISTAN, NER 10 × NER 1, E 22 × E 20 and NER 4 × P4R1 had significant negative SCA effects indicating they contributed to resistance. The crosses between TXD 306 × NER 1, E20 × K5, NER 10 × E20, NER 1 × P4R1, and E22 × NER 4 displayed significantly positive SCA effects indicating they have little value as they will contribute to susceptible progenies. These crosses are undesirable in a hybridization program since they would produce high frequencies of susceptible progeny (Dabholkar, 1992). Significant SCA effects suggest that resistance levels in progeny of certain parental combinations were significantly higher or lower than the predictions based on the parents' GCA effects. Improvement of resistance to brown spot could, thus, be accomplished by selection of crosses having high significant negative SCA effects and advancing progenies to later generations. Also, highly significant reciprocal effects found in the populations generated suggest presence of cytoplasmic or maternal effects. Further studies involving the parents with suspected cytoplasmic or maternal effects is required in order to guide breeding for improved resistance to brown spot. Parents of these crosses can be used for bi-parental mating or reciprocal recurrent selection for developing varieties with resistance to brown spot disease. The differences between reciprocal crosses indicated maternal contribution towards moderating resistance (Crusio, 1987). The study revealed significant reciprocal effects for NER 10 (P ≤0.05) and NER 4, suggesting the presence of cytoplasmic or maternal effects contributing to brown spot resistance. Thus, care should be taken to use the more resistant parent as female when making crosses for resistance to brown spot as it has been observed that the maternal effects plays a role in conditioning resistance.

Segregation patterns of selected F_2 progenies

The F_2 progenies from the crosses showed distinct phenotypic classes for brown spot scores. Analysis of segregation ratios revealed that crosses TXD 306 × NER4, NER 1 × NER 4, NER 4 × NER 1 and E22 × PAK conformed to the 3:1 ratio, suggesting the presence of at least one gene showing dominance (Allard, 1999).

Crosses E20 × NER 1 and NER 4 × 306 agreed with the 9:7 ratio, indicating presence of complementary dominant alleles (duplicate recessive epistasis). The cross E20 × PAK conformed to the 15:1 ratio, highlighting the presence of dominant alleles at either of the two loci that masked the expression of recessive alleles (duplicate dominant epistasis) (Fehr, 1987).

The separation of allelic pairs and their distribution to different cells during meiosis influences phenotypic expression of an individual (Fehr, 1987). In this study, F_2 progenies for selected crosses between resistant and susceptible rice genotypes displayed phenotypically-distinct classes based on brown spot scores, indicating that qualitative inheritance is primarily controlled by one or few genes. This suggests that individual alleles of a major gene can be predicted and readily identified on the basis of the genotype (Fehr, 1987). Goel et al. (2006) reported inheritance of brown spot resistance to involve additive and dominant effects as well as interaction between loci for the inheritance of resistance from crosses involving *Oryza nivara* germplasm. Harap (1979) and Balal et al. (1979) suggested two dominant genes were associated with resistance, while one gene was associated with susceptibility. Nagai and Hara (1930) suggested that resistance to brown spot disease is dominant while Adair (1941) suggested the involvement of several recessive genes.

Conclusions

This study revealed the influence of both additive and non-additive genes effects in the genetic control of brown spot disease resistance. The genes for resistance can, therefore, be transferred from one genotype to another through family-based breeding programs such as pedigree selection, single seed descent and back-crossing. The role of cytoplasmic gene effects in modifying resistance was also elucidated, suggesting careful selection of desirable female parents during hybridization. Segregating patterns for crosses between resistant and susceptible parents showed dominance of resistance, indicating resistance is controlled by one or a few genes.

CONFLICT OF INTEREST

The authors have not declared any conflict of interest.

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