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Research Article

Inheritance of Resistance to Fusarium Root Rot in Three Common Bean Genotypes

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Abstract
A complete diallel set of F_1 and F_2 crosses was generated from three resistant and two susceptible parents to study nature of inheritance and gene action governing resistance to *Fusarium solani* f.sp *phaseoli*. General combining ability and specific combining ability effects for root rot score in the screenhouse were significant. High Baker's ratio ($2\sigma^2gca/(2\sigma^2gca + \sigma^2sca)$) = 85% and 90% in F_1 and F_2, respectively, indicated that additive genetic effects were predominant. Narrow-sense coefficient of genetic determination based on an entry means was 0.76 and 0.86 in F_1 and F_2 respectively. Segregation for F_2 progenies indicated that resistance in each cross was conditioned by one to three partially dominant loci, modified by epistasis. We concluded that using the resistant parent as a donor, with backcrossing to the adapted recurrent parent and agronomic testing would be the best breeding procedure for improving resistance in the popular large-seeded bean varieties in Uganda.
Key words: Dry beans, *Fusarium* root rot, Gene action, Diallel crosses, epistasis

Introduction

The common bean (*Phaseolus vulgaris* L.*)* is an important grain legume in Uganda, providing up to 25% of total caloric intake and 45% of total dietary protein (Pachico, 1993). Uganda is among the top world producers of dry beans (FAOSTST, 2010), but it lags behind in production per unit area. The low yield has been partly attributed to diseases, with one of the most important being *Fusarium* root rot (FRR) (Wortmann *et al*., 1998). In 1994, farmers from South Western Uganda lost all the bean crops to FRR (Spence, 2003). The problem of FRR is increasing in many parts of Uganda, making it a high priority disease (Spence, 2003). Integrated Pest Management (IPM) is the best control strategy for FRR, with the use of resistant varieties being its essential component especially for small-scale farmers (Abawi, *et al*., 2006). Consequently, some resistant sources have been identified (Mukankusi *et al*., 2010). In a sample of these sources, Mukankusi *et al*. (2011) found resistance to be controlled by two to four additive genes, modified by dominant epistasis. In different resistant genotypes, Romàn-Avilès and Kelly, (2005) indicated complex inheritance mainly influenced by environment. They identified nine FRR resistance QTLs, with a combined effect of 5–53% on the phenotype. Schneider *et al.* (2001) reported heritability (based on entry means within environments) of 0.48 to 0.71. The present study involved NABE 13 and NABE 14 in addition to RWR719, since the inheritance of FRR resistance in these two locally adapted sources has not been studied. NABE 13 and NABE 14 are introductions from Rwanda, released in Uganda in 2006 because of their high yields, resistance to root rots and good performance in conditions of low soil fertility (Namayanja *et al*., 2003). Despite these desirable attributes, there is limited adoption of these two sources because they are mostly suited for the highlands, and the highlands represent only a small part of the total bean production in Uganda (Van Mele *et al*., 2011). These genotypes could therefore contribute genes for resistance to FRR, but the mode of inheritance of FRR in these cultivars is not adequately understood which limits their usage in bean breeding programmes.

Material and Methods

Plant material: Three bean lines, NABE 14, NABE 13 and RWR 719, resistant to FRR, were crossed with two susceptible, Ugandan popular varieties, K132 and NABE 4 (Table 1). A five-parent full-diallel mating scheme was used at the National Crops Resources Research Institute (NaCRRI) Namulonge, to produce 10 F_1 and F_2 families and their reciprocals.

The isolate**:** The pathogenic isolate of *Fusarium solani* f.sp. *phaseoli* (*FSP*-3) identified by Mukankusi *et al*.

(2010) and preserved at the National Laboratories Research Institute at Kawanda (NALRI) was used. Rejuvenation of the *FSP*-3 isolate and multiplication of inoculum was done following the method described by Mukankusi *et al*. (2010).

Screening populations: The five parents, F_1, F_2 and reciprocal diallel populations were planted in wooden trays containing mature *FSP*-3 inoculum. Each tray consisted of one row each of a particular F_1 and its reciprocal cross, two rows each of the F_2 of that same cross, and its reciprocal, two rows of each of the 2 parents involved in each cross, and one row of each of the susceptible (K132) and resistant (RWR719) checks for a total of 12 rows per tray. Each of the 12 rows contained 10 plants and the experiment contained 10 trays in total, replicated twice. This grouping according to parental combinations was done to minimize tray-to-tray variability. To obtain more plants for segregation analysis, a second non replicated set with F_2s' only was laid in the same fashion. The total number of F_2 plants varied from one population to the other in the range of 233 to 336 plants. Watering was done 1-3 times daily depending on sunshine intensity and amount of rainfall (Mukankusi *et al*, 2010). Reactions to disease were assessed 28 days after planting, and scoring the reaction according to the C1AT 1-9 scale (Abawi and Pastor-Corrales, 1990).

Data analysis**:** All statistical analyses were done using GenStat (version 12) statistical program. Diallel model 1, method 1 of Grifffing (1956) was used to detrmine the GCA, SCA, and reciprocal effects. The ratio of GCA to SCA variance components was estimated according to Baker (1978), and standard errors were calculated according to Dabholkar (1992). The fixed-effects equivalent to heritability was obtained as the narrow-sense coefficient of genetic determination (NSCGD) and broad-sense coefficient of genetic determination (BSCGD) on an entry-mean basis. $NSCGD = 2\sigma^2g/(2\sigma^2g + \sigma^2s + \sigma^2e)$, $BSCGD = (2\sigma^2g+ \sigma^2s)/(2\sigma^2g + \sigma^2s + \sigma^2e)$. Segregation ratios of the F_2 populations were tested against possible oligogenic ratios using χ^2 (Fehr, 1987). Plants or genotypes with disease scores of 1-4.9 were considered resistant, and those with higher ratings were considered susceptible.

Results and Discussion

Mean Fusarium root rot sc**ores:** The mean of the resistant (R) x susceptible (S) F_1s were significantly more resistant than the mid-parents (MP), while the mean of most F_2s with the exception of NABE 14 x K132 were more susceptible than MP (Table 2). Conversely, the mean of RxR F_1s were equal to MP, but the RxR F_2s were significantly more susceptible than the MP (Table 2). These results clearly suggest presence of a dominant form of epistasis (Fehr, 1987). Similar results were observed in the F_3 data by Mukankusi *et al*. (2011).

F_2 segregation**:** The F_2 distribution for FRR severity in all crosses was discontinuous (Fig.1). Two of the R x S crosses tended towards susceptibility in the F_2 and yet were moderately resistant in the F_1 (Fig. 1 (D), (G)). Four other R x S crosses had a skewed distribution towards resistance depicting transgressive segregation for resistance (Fig. 1 (B), (C), (E), (F)). Segregation for susceptibility was observed among all the three R x R crosses (Fig. 1 (I), (J), (H)). These results suggested presence of few genes, modified by dominant epistasis. Similar segregation patterns were reported by Mukankusi *et al*. (2011). Wijngaarden and Brakefield, (2000) noted that deviation from the additive plus dominance expectation is often indicative of epistatic effect. Additionally, the depicted transgressive segregation suggested a difference in loci controlling resistance in the three resistant parents (Wijngaarden and Brakefield, 2000).
Six of nine crosses (excluding the SxS cross, which did not segregate) significantly deviated from a 3:1 single dominant gene model (Table 3). One cross fit a 9:7 phenotypic ratio and two crosses fit 13:3 ratio, while NABE13 x NABE14 matched both a 3:1 and a 13:3 ratios, making both a single gene and a two gene explanation plausible (Table 3). Four crosses fit a 27:37 ratio (Table 3). Consistent with F_2 distribution, most crosses fit epistatic ratios of 9:7, 13:3 and 27:37. Although Romàn-Avilès and Kelly (2005) detected 9 QTLs for FRR resistance, Kamfwa, (2010), reported a single significant QTL which is suggestive of a few major genes influencing FRR resistance.

Combining ability and heritability**:** There were highly significant differences among the progenies due to the effects of both GCA and SCA (Table 4). This suggested possible improvement using these sources of resistance (Alghamdi, 2009). Similar results were reported by Mukankusi, *et al* (2011). High Baker's ratios (0.85 and 0.90 in F_1 and F_2, respectively), NSCGD (0.76 in F_1 and 0.86 in F_2) BSCGD (0.91 in the F_1 and 0.96 in the F_2) were observed (Table 4). These results implied that the performance of progeny could be predicted fairly accurately based on the GCA of its parents (Baker, 1978). A moderately resistant parent, NABE14, showed the largest negative GCA effect even though RWR719 was itself more resistant (Table 5). Since NABE14 also possesses farmer preferred attributes; large seed size, seed color and adaptability to low soil fertility (Namayanja *et al*., 2003), it will be recommended as an additional resistance source. It is evident that crosses K132 x NABE 14 (F_1 and F_2), NABE 4 x NABE 14 (F_1), and NABE 4 x RWR 719 (F_1) had a unique combination

(Table 5). They displayed significant negative SCA effects and at least one of the parents involved in these crosses was a good combiner for FRR resistance, in addition, they are well adapted and possess desirable market traits (Namayanja *et al.*, 2003).

The present study concluded that high performing parents of diverse genetic backgrounds be selected for crossing in order to increase chances of obtaining pure lines that are superior to the best parent. Also, selection should be effective for families in early generations and the value of a cross is probably predictable from the parents. A pedigree and/or backcross selection program would be appropriate to improve FRR resistance, as additive variance is predominant for this trait. Given the complex nature of FRR resistance and that field phenotyping can be laborious and requires destructive sampling, use of molecular markers would facilitate improvement of this trait.

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References

Abawi, G.S. and Pastor-Corrales, M.A. 1990. Root rots of beans in Latin America and Africa: Diagnosis, research methodologies, and management strategies. Pub No. 35, CIAT, Cali, Colombia. 114p. ISBN 958-9183-14-X

Abawi, G.S., Ludwig, J. W. and Gugino, B. K. 2006. Bean root rot evaluation protocols currently used in New York, Ann. Rept. Bean Improv. Coop 49:83-84. Available from: www.css.msu.edu/bic/PDF/Bean_Root_Rots.pdf [Accessed October 2011].

Alghamdi, S.S. 2009. Heterosis and combining ability in a diallel cross of eight faba bean (*Vicia faba L.*) genotypes. *Asi. J. Crop Sci.* **12**: 66-76.

Baker, R.J. 1978. Issues in diallel analysis. *Crop Sci.*, **18**:533-536.

CIAT, 2001. Released varieties: Uganda. Available from: http://webapp.ciat.cgiar.org/beans/released_uganda.htm [Accessed 5 March 2012]

Dabholkar, A.R. 1992. Elements of Biometrical Genetics. Revised and Enlarged Edn., New Delhi, India; Concept Publishing House. ISBN 81-7022-667-8

FAOSTAT, 2010. Food and Agriculture Organization of the United Nations. Available from: http://faostat.fao.org/site/567/default.aspx#ancor [Accessed 17 May 2010].

Fehr,W.R. 1987. Principles of cultivar development. Theory and technique. New York, USA; Macmillan publishing company. ISBN 0-9635989-0-2

Griffing, B. 1956. Concept of general and specific combining ability in relation to diallel crossing systems. *Australian J. B. Sci*. **9**:4163-4193.

Mukankusi, C., Derera, J., Melis, R., Gibson, P.T. and Buruchara, R. 2011. Genetic analysis of resistance to Fusarium root rot in common bean. *Euphytica*, **182**:11-23.

Mukankusi, M. C., Melis, R., Derera, J., Laing M. and Buruchara, R. A. 2010. Identification of sources of resistance to Fusarium root rot among selected common bean lines in Uganda. *J. Animal and Plant Sci.*, **7**: 876- 891.

Namayanja, A., Tukamuhabwa, P., Opio, F., Ugen, M.A., et al. 2003. Selection for low soil fertility bean tolerant to root rot. In: BIC. Ann. report. East Lansing, MI, USA. v. 43: 95-96. Available from: http://www.css.msu.edu/bic/PDF/Reports/BIC_2003_volume_46.pdf [Accessed 5 March 2012].

Pachico, D. 1993. The demand for bean technology. CIAT, Cali, Colombia.

Román-Avilés, B. and Kelly, J.D. 2005. Identification of Quantitative Trait Loci Conditioning resistance to Fusarium root rot in common bean. *Crop Sci*., 45:1881-1890.

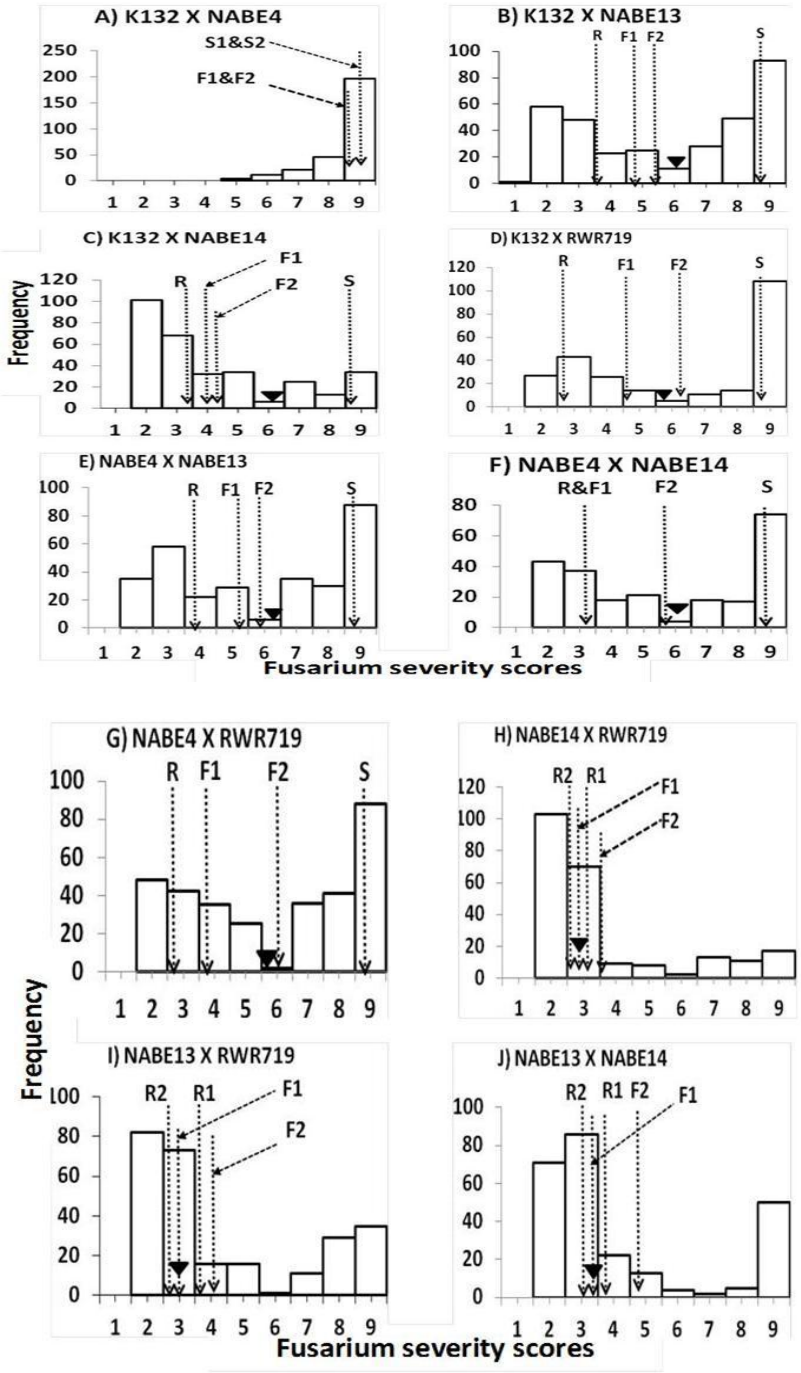
Schneider, K.A, Kenneth, F., Grafton and Kelly, J. D. 2001. QTL Analysis of Resistance to Fusarium Root Rot in Bean. *Crop Sci.*, **41**:535-542.

Spence, N., 2003. Investigation of indigenous technical knowledge of root rots. Final technical report CIAT, Cali, Colombia.

Van Mele, P., Ugen, M.A., Wanyama, D., Anyang, R., Claude, J., Rubyogo and Sperling, L. 2011. Uganda: Dreams of Starting a Company. FAO and Africa Rice. African Seed Enterprises. Avaliable from: http://www.agroinsight.com/downloads/african-seed-enterprises/Chapter10-Uganda.pdf [Accessed 20 November 2011]

Wijngaarden, P. J. and Brakefield, M. P. 2000. The genetic basis of eyespot size in the butterfly *Bicyclus anynana*: an analysis of line crosses. *Heredity*, **85**: 471-479.

Wortmann, C.S., Kirkby, R.A., Aledu C.A. and Allen, D.J. 1998. Atlas of common bean (*Phaseolus vulgaris* L) production in Africa. Centro Internacional de Agricultura Tropica,Cali, Colombia.



Key; ▼: Mid-parent, R: Resistant parent, S: Susceptible patent, R1: Resistant parent 1, R2: Resistant parent 2, S1: Susceptible parent 1, S2: Susceptible parent 2

Figure 1.Distribution frequency of FRR ratings F_2 crosses at National Laboratories Research Institute (NALRI)-Kawanda, Uganda in 2010

Table 1.Characteristics of parents used in the study

Varieties	Pedigree	Gene pool	Origin	Growth habit	Seed size	Seed color	Rxn to RR
K132	Calima-2 x Argentino1	Andean	CIAT	Bush	Large	Red mottled	S
NABE4	SUG 47 x CAL 103	Andean	CIAT	Bush	Large	Red mottled	S
NABE13	Unknown	Andean	Rwanda	Bush	Large	Dark red	MR
NABE14	Unknown	Andean	Rwanda	Bush	Large	Kidney red	MR
RWR719	Cyunyu x Kermes	Meso-American	Rwanda	Bush	Small	Red	R

RR = root rots, R, = resistant, MR = moderately resistant to root rot, S = susceptible to root rot, CIAT = International Centre for Tropical Agriculture
Source: CIAT, 2001, 2008.

Table 2. Mean severity scores of *Fusarium solani* for F_1, F_2 diallel progenies, and parents

Parents		K132(S)	NABE4(S)	NABE13(R)	NABE14(R)	RWR719(R)	Means
K132(S)	**F_1**		8.3^{ns}	5.2^{ns}	4.0*	5.2^{ns}	6.2
	F_2		8.6^{ns}	5.6^{ns}	4.1***	6.7**	6.7
	MP	**8.7**	8.7	6.2	5.9	5.4	
NABE4(S)	**F_1**	8.7^{ns}		4.9^{ns}	3.0***	3.4**	6.0
	F_2	8.6^{ns}		5.9^{ns}	5.8^{ns}	5.5^{ns}	7.0
	MP	8.7	**8.7**	6.2	5.9	5.4	
NABE13(R)	**F_1**	4.6*	4.8*		3.7^{ns}	3.1^{ns}	4.0
	F_2	5.9^{ns}	5.4^{ns}		4.8**	4.2**	4.8
	MP	6.2	6.2	**3.7**	3.4	2.9	
NABE14(R)	**F_1**	4.1*	3.6**	3.1^{ns}		2.7^{ns}	3.3
	F_2	4.1***	6.0^{ns}	4.3*		3.6*	4.2
	MP	5.9	5.9	3.4	**3.2**	2.7	
RWR719(R)	**F_1**	3.7*	4.1^{ns}	3.3^{ns}	3.0^{ns}		3.4
	F_2	5.9^{ns}	6.5*	3.9*	3.9**		4.6
	MP	5.4	5.4	2.9	2.7	**2.7**	
					SEM	LSD	CV%
	F_1				0.7	2.0	10.5
	F_2				0.4	1.2	10.5

*, **, ***: Significant deviation from mid-parent at ($p<0.05$), ($p<0.01$), and ($p<0.001$), respectively. ns: Non significant deviation from mid-parent, R: Resistant parent, S: Susceptible parent, MP: Mid-parent, SEM: Standard error of the mean, LSD: Least significant difference, CV%: Coefficient of variability. All scores are based on a 1 – 9 scale

Table 3. Segregation pattern for FRR in F_2 crosses

Crosses	R:S ratio (Obs)	$\chi 2$ under different model ratios			
		3:1	9:7	13:3	27:37
K132 (S)xNABE13(R)	155:181	149***	14**	272***	2^{ns}
K132(S)xNABE14(R)	235:78	0.001^{ns}	45***	7**	138***
K132(S)xRWR719(R)	110:138	124***	14**	221***	0.48^{ns}
NABE4(S)xNABE13(R)	144:159	121***	9**	226***	3.5^{ns}
NABE4(S)xNABE14(R)	119:113	69***	2^{ns}	136***	8**
NABE4(S)xRWR719(R)	150:167	129***	10**	239***	3^{ns}
NABE13(R)xNABE14(R)	196:57	0.82^{ns}	46***	2^{ns}	129***
NABE13(R)xRWR719(R)	187:76	2^{ns}	23***	17***	90***
NABE14(R)xRWR719(R)	190:43	5*	60***	0.01^{ns}	147***

*, **, ***: Significant deviation from model ratio at p<0.05), (p<0.01), and (p<0.001), ns: Non significant deviation from model ratio; Obs: observed ratio.

Table 4. Anova for F_1 and F_2 of 5 x 5 diallel for FRR

Sources of variation	d.f	m.s.		Variance components	
		F_1	F_2	F_1	F_2
Replications	1	0.37^{ns}	1.22*		
Genotypes	24	3.79***	3.19***		
GCA	4	17.92***	16.43***	1.74	1.63
SCA	10	1.72**	0.92***	0.63	0.38
Reciprocal difference	10	0.20^{ns}	0.16^{ns}	-0.14	0.00
Error	24	0.47	0.16	0.47	0.16
[a]Bakers' ratio $(2\delta^2g)/(2\delta^2g + \delta^2s)$				0.85	0.90
[b]BSCGD $(2\delta^2g + \delta^2s)/(2\delta^2g + \delta^2s + \delta'^2e)$				0.90	0.96
[c]NSCGD $(2\delta^2g)/(2\delta^2g + \delta^2s + \delta'^2e)$				0.76	0.86

*: Significant (p<0.05), **: Significant (p<0.01), ***: Highly significant (p<0.001), ns: Not significant, a: Relative importance of GCA and SCA according to Baker (1978), b: Broad sense coefficient of genetic determination for a fixed model (analogous to H), c: Narrow sense coefficient of genetic determination for a fixed model (analagous to h^2), δ^2g: GCA variance component, δ^2s: SCA variance component, δ^2r: Reciprocal variance component, δ^2e: Error variance averaged over two replications. All mean squares and coefficient of genetic determination (CGD) values are on the basis of the mean of two replications.

Table 5. Combining ability effects for Fusarium root rot score in F_1 (above diagonals) and F_2 (below diagonals)

Parents	K132	NABE4	NABE13	NABE14	RWR719	GCA(F_1)
K132		1.10*	-0.68^{ns}	-0.88^{*}	-0.49^{ns}	1.59***
NABE4	0.36^{ns}		-0.42^{ns}	-1.33**	-0.91^{*}	1.28***
NABE13	-0.25^{ns}	-0.43^{ns}		0.54^{ns}	0.36^{ns}	-0.54**
NABE14	-1.42***	0.30^{ns}	0.94***		0.63^{ns}	-1.18***
RWR719	0.54^{*}	-0.08^{ns}	0.15^{ns}	0.33^{ns}		-1.15***
GCA(F_2)	1.22***	1.55***	-0.69***	-1.17***	0.91***	
					F_1	F_2
S.Egca = $[((p-1)/2p^2)\delta^2e]^{1/2}$					0.19	0.11
S.Esca = $[((p^2-2p+2)/2p^2)\delta^2e]^{1/2}$					0.40	0.23

*: Significant (p<0.05), **: Significant (p<0.01), ***: Highly significant (p<0.001), ns: Not significant, S.Egca: Standard error for GCA effects, S.Esca: Standard error for SCA effects.