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DOI: <http://dx.doi.org/10.4314/acsj.v24i2.8>



HERITABILITY FOR RESISTANCE TO ROSETTE DISEASE IN EXOTIC VALENCIA GROUNDNUTS

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(Received 25 March, 2015; accepted 4 April, 2016)

ABSTRACT

Groundnut rosette disease (GRD) is the most destructive virus disease of Valencia groundnuts (*Arachis hypogaea* L.) in sub-Saharan Africa. Cultural, biological and chemical control measures have received limited success due to small scale farmers' inability to use them. Use of host plant resistance provides the most effective and economically viable management option for the resource poor farmers. This study was conducted to determine heritability for resistance to GRD in Valencia groundnuts. Six crosses; Valencia C (P₁) × ICGV-SM 90704 (P₂), Valencia C (P₁) × ICGV-SM 96801 (P₂), Valencia C (P₁) × ICGV-SM 99566 (P₂), NuMex-M₃ (P₁) × ICGV-SM 90704 (P₂), NuMex-M₃ × ICGV-SM 96801 (P₂), and NuMex-M₃ (P₁) × ICGV-SM 99566 (P₂), were made to generate F₁, F₂, BC₁P₁ and BC₁P₂ populations. Data on GRD severity were collected on a 1-9 score scale. Genetic Advance as a percentage of the mean (GAM) and heritability were estimated using variance components. Phenotypic Coefficient of Variation (PCV) and Genotypic Coefficient of Variation (GCV) estimates were high (20.04-70.1%) in the six crosses, except for Valencia C × ICGV-SM 96801 (18.1%) and NuMex-M₃ × ICGV-SM 96801 (17.1%), which exhibited moderate GCV values. Broad and narrow sense heritability estimates for GRD disease score ranged from 64.1 to 73.7% and 31 to 41.9%, respectively, in all the crosses. GAM was high in all the crosses (21-50.7%), except for Valencia C × ICGV-SM 96801 (14.67), M₃ × ICGV-SM 99566 (18%) and NuMex-M₃ × ICGV-SM 96801 (13.5%) crosses that exhibited moderate GAM. The study revealed the presence of variability of GRD resistance, implying that genetic improvement of these exotic materials is possible.

Key Words: *Arachis hypogaea*, coefficients of variation

RÉSUMÉ

La maladie de rosette chez l'arachide (*Arachis hypogaea* L.) est la plus destructive des maladies virales chez les variétés Valencia en Afrique au sud du Sahara. Les moyens de lutte culturale, biologique et chimique présentent des limitations surtout liées à l'inhabilité des petits producteurs à les mettre en pratique. L'utilisation de variétés résistantes représente le moyen le plus efficace et économiquement viable pour les petits producteurs. La présente étude a été conduite afin de déterminer l'héritabilité de la résistance à la maladie de rosette chez les variétés d'arachide Valencia. Six croisements; Valencia C (P₁) × ICGV-SM 90704 (P₂), Valencia C (P₁) × ICGV-

SM 96801(P₂), Valencia C (P₁) × ICGV-SM 99566 (P₂), NuMex-M₃ (P₁) × ICGV-SM 90704 (P₂), NuMex-M₃ × ICGV-SM 96801 (P₂), et NuMex-M₃ (P₁) × ICGV-SM 99566 (P₂), ont été effectués afin de générer F₁, F₂, BC₁P₁ et BC₁P₂ populations. Des données ont été collectées sur le degré de sévérité de GRD en se servant d'une échelle de 1-9. Les paramètres d'avancée génétique exprimée en pourcentage de la moyenne (GAM) et héritabilité ont été estimés à partir de composantes de variance. Le coefficient de variation phénotypique (PCV) et génotypique (GCV) estimés étaient élevés (20,04-70,1%) dans les six croisements, sauf pour Valencia C × ICGV-SM 96801(18,1%) et NuMex-M₃ × ICGV-SM 96801(17,1%), où les valeurs de GCV étaient modérées. Les valeurs de l'héritabilité au sens large et au sens strict pour la résistance à GRD variaient respectivement de 64,1 à 73,7% et de 31 à 41,9%, au niveau de tous les croisements. Les valeurs de GAM étaient élevées au niveau de tous les croisements (21-50,7%), sauf pour les croisements Valencia C × ICGV-SM 96801 (14,67), M₃ × ICGV-SM 99566 (18%) et NuMex-M₃ × ICGV-SM 96801 (13,5%) où les valeurs de GAM étaient modérées. L'étude a révélé l'existence dans la résistance au GRD, ceci implique qu'il est possible d'entreprendre l'amélioration génétique de ces matériels.

Mots Clés: *Arachis hypogea*, coefficients de variation

INTRODUCTION

Valencia groundnuts belong to one of the botanical varieties of cultivated groundnuts (*Arachis hypogea* L.) (Krapovickas and Gregory, 1994) known for their quality attributes like good and distinctive flavour with a soft skin (Patte *et al.*, 2001; Mark *et al.*, 2009), early maturity, high number of seeds per pod and relatively bigger seeds that make it key for commercial purposes. Also, they are the most preferred for high oil content (Kaaya and Warren, 2005) compared with other groundnut botanical varieties. Also, they are the most preferred for high oil content (Kaaya and Warren, 2005) compared with other groundnut sub-species. Despite their importance, production is still constrained by the groundnut rosette disease (GRD) in Uganda. The disease is sporadic and unpredictable, and can result in yield losses of up to 100% (Waliyar, 1999; Subrahmanyam *et al.*, 2001; Adu Dapaah *et al.*, 2004).

There have been efforts to control GRD using a combination of cultural, biological and chemical measures (Waliyar *et al.*, 2007; Okello *et al.*, 2014); however, little success has been achieved because small scale farmers seldom use them. In addition, chemical control of aphids (*Aphis craccivora*) which transmit the disease is not economically viable because of their persistent nature in the disease transmission (Waliyar *et al.*, 2007). Use of host plant resistance is the most effective and economically viable management

options for the resource poor farmers, especially in Uganda. However, it is limited by lack of resistant varieties and information on heritability of GRD resistance on the available Valencia breeding materials.

Estimation of genetic variability with the help of suitable parameters such as genetic coefficients of variation, heritability estimates and genetic advance is absolutely necessary to start an efficient breeding programme (Atta *et al.*, 2008; Janila *et al.*, 2013; Wambi *et al.*, 2014). Kayondo *et al.* (2014) reported a high (93%) heritability estimate for GRD rosette disease in Uganda. However, heritability estimates depend on the genetic background of the materials used and the environment from which the populations are evaluated (Kearsey and Pooni, 1996; Wambi, 2014; Wambi *et al.*, 2014). Therefore, this study was conducted to estimate genotypic and phenotypic coefficients of variations, and heritability for GRD resistance, in Valencia groundnut genotypes.

MATERIALS AND METHODS

Study area. The study was conducted at the National Semi-Arid Resources Research Institute (NaSARRI), of the National Agricultural Research Organisation (NARO), located 01°- 30' 00"N and 33° 33' 00"E in Serere district in Uganda. This is a known hotspot for GRD in the country (Okello *et al.*, 2010). It receives an annual rainfall of 1,000-1,200 mm.

The plant materials for this study were developed by crossing two exotic susceptible Valencia lines, Valencia C and NuMex-M₃, provided by the Plant Breeding Department New Mexico State University, USA; and Rosette resistant lines, namely Serenut 6T (ICGV SM 99566), Serenut 2 (ICGV-SM 90704) and Mali (ICGV-SM 96801), provided by the Groundnut Improvement Programme at the NaSARRI, Uganda.

Six crosses, namely Valencia C × ICGV-SM 90704, Valencia C × ICGV-SM 96801, Valencia C × 99566, NuMex-M₃ × ICGV-SM 90704, NuMex-M₃ × ICGV-SM 96801 and NuMex-M₃ × ICGV-SM 99566) were made to generate F₁s. The F₁s of each cross were further crossed to their parents P₁ (female parent) and P₂ (male parent) to derive BC₁P₁ and BC₁P₂ generations, respectively. On the same F₁ plants, F₂ seed was generated by allowing some flowers to self-pollinate.

The six generations, namely P₁, P₂, F₁, F₂, BC₁P₁ and BC₁P₂ of each cross were evaluated in a randomised complete block design (RCBD), with three replications. The materials were planted in six row plots of 3 m length, at a spacing of 45 cm x 15 cm. Acholi white, a highly susceptible local variety, was used as the infector line to increase disease pressure, and was planted in a single row between every two rows of test materials. The infector rows were planted 14 days before the test materials.

Data collection. Disease severity data were recorded at 115 days after planting, on ten randomly selected plants in each replicate. Each plant was scored for a rating scale of 1-9 adopted from the Groundnut Improvement Programme at NaSARRI, Serere in Uganda (Okello *et al.*, 2014).

Where:

1-3 represented highly resistant, HR, (where 1= resistant with no symptom, 2 = very slight leaf symptoms and 3 = slight leaf symptoms but still negligible), 4-5 resistant, R, with leaf symptoms and no stunting (where 4 = showed 50% symptoms on leaves, and 5 = all leaves showed symptoms of chlorosis), 6-7 moderately susceptible (MS) with leaf symptoms and stunting (where 6 was 25% stunted and 7 = 50%

stunted), 8-9 highly susceptible (HS) with severe leaf symptoms with >50% stunt (where 8 = has few pods; while 9 = no pod at all is expected).

Data analysis. Data on disease severity on individual plants of each generation for each cross were subjected to one way ANOVA, using GenStat Version 13 computer program. ANOVA was based on the linear mathematical model:

$$Y_{ij} = \mu + r_i + g_j + e_{ij};$$

Where:

Y_{ij} = observed effect for ith replication and jth genotype,

μ = grand mean of the experiment,

r_i = effect of the ith replication,

g_j = effect of the jth genotype,

e_{ij} = residual effect

Where the ANOVA showed significant differences, the treatment means were separated using by Fisher's protected Least Significant Difference at 5% probability level (Payne *et al.*, 2010).

Estimation of variance components. Variance components that included, environmental, genotypic, additive and dominance, were obtained following the procedure of Kearsy and Pooni (1996).

$$\sigma^2_e = (\sigma^2_{P_1} + \sigma^2_{P_2} + 2\sigma^2_{F_1})/4 \dots\dots\dots \text{Equation 1}$$

Where: σ²e = Environmental variance or error, σ²P₁, σ²P₂ and σ²F₁=Variance of susceptible parents, resistant parents and first filial generations, respectively.

$$\text{Phenotypic variance} = [\sigma^2_{F_2}] = \text{variance of } F_2 \text{ generation} \dots\dots\dots \text{Equation 2}$$

$$\text{Genotypic variance in } F_2 [\sigma^2_G (F_2)] = \sigma^2_{F_2} - \sigma^2_e \dots\dots\dots \text{Equation 3}$$

Where:

σ²F₂ = variance of F₂ generation, and σ²e = Environmental variance

Additive variance in F_2 [$\sigma^2A (F_2)$] = $(2 \sigma^2F_2) - [\sigma^2BC_1 + \sigma^2BC_2]$ Equation 4

Where:

σ^2F_2 = variance of F_2 generation, and σ^2BC_1 and σ^2BC_2 = variance of backcross to female and male parents, respectively.

Dominance variance in F_2 [$\sigma^2D (F_2)$] = $\sigma^2G (F_2) - \sigma^2A (F_2)$ Equation 5

Where:

[$\sigma^2G (F_2)$] = Genotypic variance in F_2 , and $\sigma^2A (F_2)$ = Additive variance in F_2 .

Coefficient of variability. Both the genotypic coefficient of variation (GCV) and phenotypic coefficient of variation (PCV) were estimated following the method suggested by Singh and Chaudhury (1985), where the GCV and PCV values were classified as low (0-10), medium (11-20) and high (20 and above) (Sivasubramanian and Menon, 1973).

PCV = $(\sqrt{V_p})/X * 100$ Equation 6

GCV = $(\sqrt{V_G})/X * 100$ Equation 7

Where:

V_p = Phenotypic variance, V_G = Genotypic variance, and X = Grand mean of the character.

Estimation of heritability. The variance components described above were used to determine broad sense heritability (h^2_b) and narrow sense heritability (h^2_n), following Keasey and Pooni (1996) in all the six crosses as detailed below:

$h^2_b = 100[\sigma^2G (F_2)/V_{F_2}]$ Equation 8

Where: h^2_b = Broad-sense heritability, $\sigma^2G (F_2)$ = genotypic variance in F_2 and V_{F_2} = variance of F_2 generation

$h^2_n = 100[\sigma^2A (F_2)/V_{F_2}]$ Equation 9

Where: h^2_n = Narrow-sense heritability, $\sigma^2A (F_2)$ = additive variance in F_2 , and V_{F_2} = variance of F_2 generation.

Estimation of genetic advance (GA). Genetic advance was estimated following Singh and Chaudhury (1985) method.

Genetic advance (GA) = $h^2_n \times k \times \sigma^2_p$ Equation 10

Where:

h^2_n = Narrow sense heritability estimate, σ^2_p = Phenotypic standard deviation, and K = Selection intensity at 5% is equal to 2.06.

Genetic advance as percent of mean (GAM%) = $(GA/X) * 100$

Where:

X = Grand mean of the trait, and GA = Genetic advance

The Genetic Advance as percent of mean (GAM%) was categorised as described by Johnson *et al.* (1955), as low (0-10), medium (10-20) and high (21 and above).

RESULTS

There were significant variations in the generations for disease severity (Table 1). The donor parents, ICGV - SM 99566 and ICGV-SM 90704, were highly resistant with mean scores ranging from 1.33-2.83; while ICGV -SM 96801 was slightly resistant with the score that ranged 4 to 6. All the susceptible genotypes, Valencia C and NuMex-M₃ exhibited higher disease score that ranged from 7.5 to 8.

All the six F_1 s showed high resistance to GRD (mean score range 1.67 to 2.0), and the mean disease score in F_2 were moderately resistant (Table 1). The segregants of F_2 from ICGV-SM 90704 donor line were highly resistant, with mean scores in the range of 2.3-2.73; whereas those from ICGV-SM 96801 were susceptible with mean scores of 5 to 6.

Coefficients of variability and heritability. Table 2 shows phenotypic and genotypic coefficients of variation, heritability and GAM estimates for GRD resistance in six crosses. In all crosses, the dominance variance component (V_D) exhibited relatively higher magnitudes (0.65-0.99) compared with the additive component (V_A), except for NuMex-M₃ × ICGM-SM 90704 which had a relatively lower (0.37) magnitude. The PCV and GCV estimates were high (20.04-70.1%) in all the six crosses, except for Valencia C × ICGV-SM 96801 (18.1%) and NuMex-M₃ × ICGV-SM

TABLE 1. Groundnut Rosette Disease mean score and standard error for the six generations of the 6 crosses of groundnuts in Eastern Uganda

Generation	Valencia C × ICGV-SM 99566	Valencia C × ICGV-SM 90704	Valencia C × ICGV-SM 96801	NuMex-M ₃ × ICGV-SM 99566	NuMex-M ₃ × ICGV-SM 90704	NuMex-M ₃ × ICGV-SM 96801
P ₁ (S)	7.5	7.83	8.167	8.67	7.67	7.83
P ₂ (R)	2.17	1.35	5.05	2.83	1.33	4.67
F ₁	1.67	1.83	1.83	2.0	1.67	1.67
F ₂	4.67	2.3	6.0	4.3	2.73	5.0
BC ₁ P ₁	6.33	6.33	7.5	8.67	7.0	6.0
BC ₁ P ₂	2.83	3.0	5.0	1.83	2.33	1.67
F cal	12.16**	8.27**	5.47**	48.1**	34.0**	59.4**
MS	20.2	23.2	21.1	16.1	13.3	11.8
CV (%)	29.2	26.8	27.1	24.9	30.2	20.4

P₁(S) = Parent 1 elite parents, (Valencia C and NuMex-M₃) P₂ = P₂(R) - the donor parents. F₁ = 1st Filial generation, F₂ = 2nd Filial generation, BC₁P₁ = Backcross to susceptible parent (P₁) and BC₁P₂ = Backcross to resistant parent (P₂), ** = significant at 95%, CV = Coefficient of variation, Fcal = F-value, MS = Mean sum of square. The Resistance rating scale: 1-3 (highly resistant), 4-5 slight (Slightly resistant), 6-7 (moderately susceptibility), 8-9 (highly susceptible)

TABLE 2. Genetic variance components and parameters for groundnut rosette resistance

Generation	Valencia C × ICGV-SM 99566	Valencia C × ICGV-SM 90704	Valencia C × ICGV-SM 96801	NuMex-M ₃ × ICGV-SM 99566	NuMex-M ₃ × ICGV-SM 90704	NuMex-M ₃ × ICGV-SM 96801
V _E	0.83	0.90	0.90	0.70	0.60	0.50
V _G	1.50	1.93	1.63	1.93	1.07	1.20
V _A	0.83	0.99	0.79	0.97	0.70	0.55
V _D	0.67	0.94	0.82	0.99	0.37	0.65
V _P	2.33	2.83	2.51	2.66	1.67	1.70
X	5.30	2.40	7.00	6.80	2.20	6.40
PCV	28.80	70.10	22.60	23.60	58.00	20.40
GCV	23.10	57.80	18.10	20.50	47.10	17.10
h ² _b	64.40	68.20	64.00	73.70	64.10	70.60
h ² _n	35.60	34.90	31.00	36.40	41.90	32.40
GA	1.12	1.20	1.02	1.23	1.10	0.86
GAM (%)	21	50.5	14.67	18	50.7	13.5

V_E = Environmental variance, V_G = Genotypic variance, V_A = Additive variance, V_D = Dominance variance, PCV and GCV = Phenotypic and Genotypic Coefficient of Variation respectively, h²_b and h²_n = Broad and narrow sense heritability, respectively, X = Grand mean of the generations, GAM% = Genetic Advance as percent of mean.

96801(17.1%) crosses, which exhibited moderate GCV values. Broad and narrow sense heritability estimates for GRD disease score ranged from 64.1 to 73.7% and 31 to 41.9%, respectively, in all the crosses. The GAM was high in all the crosses (21-50.7%), except for Valencia C x ICGV-SM 96801 (14.67), M₃ x ICGV-SM 99566 (18%) and NuMex-M₃ x ICGV-SM 96801 (13.5%) which exhibited moderate GAM.

DISCUSSION

The six crosses showed highly significant differences ($P < 0.01$) among generations for GRD resistance (Table 1), suggesting presence of variability for GRD disease score in the generations. The significant variation implies that genetic improvement of groundnut for GRD resistance is possible, and can be exploited by breeders for groundnut improvement. The presence of such variability could be a result of wide genetic distances between the parental backgrounds that were used in the study.

Results of the current study are comparable to those Monyo *et al.* (2007), who reported variability for GRD resistance among 143 accessions that were evaluated. Naidu *et al.* (1999) also reported GRD variations to be due to diversity among the causal agents (sat RNA variants), differences in genotype response, variable climatic conditions and mixed infections with other viruses. High resistance was exhibited by donor parents, ICGV-SM 99566 and ICGV-SM 90704. Waliyar *et al.* (2007) and Okello *et al.* (2010) also reported the two lines as universal donor for GRD resistance.

The mean of F₁ generations tended towards the mean of resistant parents (Table 1), indicating that resistance to GRD could be controlled by dominant genes or epistatic gene action. These results support earlier reports that resistance to GRD is controlled by dominant genes (Olurunju *et al.*, 1992; Akpan and Olurunju, 2009). Tolin (2012) approximated that over 80% of known virus resistance was 50% completely dominant, with the remaining being polygenic, which is comparable to the finds of the current study. In contrast, Harkness (1977) and Nigam and Bock (1990) reported recessive genes controlling resistance to GRD. The presence of dominant

genes, implies that the F₁s could be utilised due to relative heterosis where hybrid vigour could be exploited. However, in groundnuts, commercial production of F₁ seed can not be achieved since it is a self-fertilising crop and a tetraploid nature makes the F₁s unstable. Wambi (2014), suggested that in such situations, selection should take place at later generations when the dominance effects of the genes are decreased. To exploit such heterosis, breeding methods such as recurrent selection and single seed descent may be used.

Success of genetic improvement is attributed to the magnitude and nature of variability present for a specific character (Wambi, 2014). Moderate to high level GCV (17.1-57.8%) and high PCV (20.4-70.1%) were noticed for all crosses (Table 2), indicating higher magnitudes of heritable variations for GRD resistance in these crosses. This could be attributed to genotypes with very little effect of the environment. According to Oyiga and Iguru (2011) and Vishnuvardhan *et al.* (2012), when the magnitude of genetic variance is higher than the environmental variance, it may indicate a predominance of additive gene actions, which could result to high response to phenotypic selection in early generations due to high influence of the genetic component to the total variance of the trait under study.

Information on genotypic coefficients of variation (Table 2) reveals the existence genetic variability present in the genotypes for GRD resistance, but does not provide full scope to assess the variation that is heritable. In our study, all crosses had high broad sense heritability values (64.1-73.7%) (Table 2). The findings are comparable with those of several other researchers (Van der Merwe, 1998; Adu Dapaah, *et al.*, 2007; Kayondo *et al.*, 2014), who reported high broad sense heritability estimates for GRD resistance (67- 93%). High heritability indicates a high response to selection due to reduced environment influence and predominance role of additive gene effects (Tafere *et al.*, 2013), in the control of GRD resistance, which could result to efficient response to selection in early generations.

However, it should be noted that Broad Sense Heritability coefficients comprise all the genetic influences in its expression, instead of only the additive effects of additive genes. This cannot

be used as an indicator for obtaining a precise estimation of selection gains. Therefore, h^2_n coefficients that comprised only of additive effect of additive genes were computed in our study. Moderate values (31-41.9%) for narrow sense heritability were observed in all crosses (Table 2), indicating greater dominance effects on GRD resistance than the additive.

In all crosses, the dominance variance component (V_D) exhibited relatively higher magnitudes, compared with additive component (V_A), except for NuMex-M₃ x ICGM-SM 90704 which had a relatively lower magnitude. It is generally verified that an increase in magnitude of V_D implies a decrease in h^2_n in the reference to F₂ generation. Hence selection of genotypes from initial generations for GRD disease score in these crosses may be difficult due to the higher influence of dominance effects. According to Kormsa-art *et al.* (2002), selection for such traits controlled by dominance becomes ineffective when carried out in early generations. Therefore, selection based on this trait is more effective when undertaken in subsequent generations of all crosses. In this way, the occurrence of heterozygotes would be reduced and the available additive variance for selection increased, thereby providing higher possibilities of selection gains for the trait.

The genetic advance, as percentage of mean (GAM), was high in all the crosses (21-50.7%)(Table 2), except for Valencia C x ICGV-SM 96801 (14.67%), M₃ x ICGV-SM 99566 (18%) and NuMex-M₃ x ICGV-SM 96801 (13.5%), which exhibited moderate GAM. According Wambi (2014), high GAM indicates a predominant role of additive gene actions in control of GRD resistance, which is very important for the improvement of a crop through breeding; while moderate GAM reflects additive and non-additive effects in control of the trait under study. Breeding methods such recurrent selection and biparental mating would be useful for improvement of such traits controlled by additive and non-additive effects (Nidagundi *et al.*, 2012).

CONCLUSION

The study has revealed presence of considerable heritable variation for GRD resistance, revealing

a greater success of these exotic materials to genetic improvement. Moderate narrow sense heritability (31-41.9%) was evident in all crosses; however, the heritability estimates depend on the genetic background of the parents that were used. Therefore, selection based on this trait is more effective in subsequent generations in all crosses when the available additive variance for selection is increased, thereby providing higher possibilities of selection gains for the trait. Breeding methods such recurrent selection and biparental mating can be used for improvement of the materials for GRD resistance.

ACKNOWLEDGEMENT

This study was funded by the United States Agency for International Development (USAID) under the Peanut Collaborative Research Support Program (Peanut CRSP) grant ECG-A-00-07-0001-00. We acknowledge the Legume Improvement Programme, National Semi-Arid Resources Research Institute (NaSARRI) of the National Agricultural Research Organisation (NARO), Uganda, for providing germplasm, glasshouse for hybridisation operations and field for evaluation of breeding materials.

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