

## GENETICS OF RESISTANCE TO GROUNDNUT ROSETTE VIRUS DISEASE

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### ABSTRACT

Groundnut Rosette Virus disease (GRD) has long been regarded a major limiting biotic constraint to groundnut production in Sub-Saharan Africa (SSA). The disease is caused by a complex of three viral components that interact in a synergistic fashion resulting into severe crop losses. A study was conducted to better understand the genetics of inheritance of GRD resistance. Nineteen groundnut genotypes among which twelve F<sub>2</sub> families populations arising from a 3x4 North Carolina II mating design, were evaluated for their percentage disease severity (PDS) and incidence (PDI). There was significant genetic variability for resistance to GRD among the materials studied with more significant additive gene action as compared to non additive. However, since specific combining ability effects were not so consistent among the F<sub>2</sub> family populations, evaluation and testing of progenies alongside with their parents would be more meaningful and selection in the early generations would be the most effective strategy. Further, narrow sense heritability of 53% suggests that performance of groundnut progenies could be partly predicted by both parental and individual cross means.

*Key Words:* *Arachis hypogaea*, disease severity, inheritance, heritability

### RÉSUMÉ

La rosette, une maladie virale de l'arachide (GRD) a pendant longtemps été considérée comme une contrainte biotique majeur à la production de l'arachide en Afrique Sub Saharienne (SSA). La maladie est causée par un complexe de trois composants viraux qui interagissent de façon synergétique causant ainsi de pertes lourdes de la culture. Une étude était menée afin de mieux comprendre l'acquisition génétique de la résistance de GRD. Dix neuf génotypes d'arachides parmi douze populations de famille F<sub>2</sub> provenant de la disposition du mating 3x4 de Nord Caroline II étaient évalués sur base de leur pourcentage de sévérité (PDS) et incidence maladie (PDI). Une variabilité significative de la résistance au GRD parmi le matériel étudié avec plus d' action additive significative du gène en comparaison à la non additive. Par ailleurs, du fait que les effets de combinaison des aptitudes n'étaient pas consistants parmi les populations de familles F<sub>2</sub>, l'évaluation et le test des progénies avec leurs parents pourraient être plus significatif et la sélection parmi les générations précoces pourrait être une stratégie la plus efficace. Aussi, l'heritabilité de 53% suggère que la performance des progénies d'arachide pourrait partiellement être prédit par le moyen de croisements parentaux et individuels.

*Mots Clés:* *Arachis hypogaea*, sévérité maladie, acquisition, héritabilité

## INTRODUCTION

Groundnut (*Arachis hypogaea* L.), is a valuable vegetable oil crop, widely grown in the semi-arid areas of Sub-Saharan Africa (SSA). It is the second most widely grown legume crop after beans in Uganda (Okello *et al.*, 2010). Groundnut production is largely constrained by biotic stresses, with groundnut rosette virus disease (GRD) contributing to annual losses of US\$156 million across Africa (Nigam *et al.*, 2012).

Groundnut rosette virus disease is caused by synergistic interaction of three viral agents, namely, groundnut rosette virus (GRV), its satellite RNA (Sat RNA) and groundnut rosette assistor virus (GRAV). GRAV plays an important role in aiding aphid transmission, alongside the other two viral components. It has been reported that absence of GRAV in the viral combination results into symptomless but infected plants (Waliyar *et al.*, 2007). GRD has been reported to occur sporadically, resulting into total yield loss in susceptible genotypes (Naidu *et al.*, 1999). The fast spread of GRD is facilitated by the cowpea aphid (*Aphis craccivora* Koch) that is widely distributed in the tropics and mediterranean regions (Waliyar *et al.*, 2007). While the initial GRD epidemics reported in early 1970's were characteristic of chlorotic rosette symptoms, mosaic and green forms have also been reported in some epidemics (Naidu *et al.*, 1999). Plants affected by either of the three major forms are often severely stunted and bushy, with leaves being curled and distorted (Nigam *et al.*, 2012).

Variations in GRD symptoms have been attributed to the existence of variant strains of the Sat RNA of GRV (Olorunju *et al.*, 2001). Key market class cultivars, including landraces have succumbed to GRD, resulting in yield reduction to as low as 800 kg ha<sup>-1</sup>, compared with 3,000 kg ha<sup>-1</sup> reported from on-station plots in Uganda (Okello *et al.*, 2010). Cultural, chemical and biological measures have not effectively curbed the spread of GRD, hence the low farmer adoption rate of such control measures (Olorunju *et al.*, 2001). However, use of resistant cultivars to GRD is considered as an effective alternative in managing the disease at the farm level (Nigam *et al.*, 2012). Resistant cultivars like

Serenut 2, Serenut 3 and Serenut 8 have been made available but have low marketability and farmer adoption resulting in the need to introgress the available resistance genes into the farmer preferred landraces.

The objective of this study was to determine the mechanism of gene action controlling GRD resistance and estimate its heritability in potential sources.

## MATERIALS AND METHODS

**Genetic materials.** Three GRD resistant groundnut lines, namely, Serenut 2, Serenut 3 (ICGV-SM 93530), and Serere 8 (ICGV99019) and four GRD susceptible cultivars; Acholi white, Egoromoit, Red Beauty and Serenut 1 were crossed in 4 x 3 North Carolina 2 mating design generating 12 F<sub>1</sub> offsprings families. All the 12 F<sub>1</sub> offsprings families consisting of 3-5 plants per family were planted in plastic bowls and advanced to F<sub>2</sub> generation progenies in an aphid free screen house.

**Field setting and experimental design.** Field evaluation of genetic materials for GRD resistance was conducted at the National Semi-Arid Resources Research Institute (NaSARRI) in Uganda, at an elevation of 1085 m above sea level (masl), 1°29'39"N and 33°27'19"E, during the 2011 growing season. NaSARRI has a bimodal rainfall pattern, with an annual mean of 1427 mm. The experiment was laid out in a completely randomised block design, in 3 replications, with each plot consisting of 20 plants per plot arranged in two rows. Seven parental materials and 12 F<sub>2</sub> families were evaluated in a field following early planting (1 week) of infector rows containing Acholi White cultivar. Each row was flanked by two infector rows of Acholi white to augment disease pressure. Disease assesment on an individual plant basis was done by recording both disease incidence and severity at 40, 60, and 80 days after planting. The disease incidence rating scale used was based on the percentage of disease incidence (PDI) to interpret genotype response according to Waliyar *et al.* (2007) method as follows:

PDI of  $\leq 10$  (highly resistant), 11 – 30 PDI (Resistant), 31 – 50 PDI (moderately resistant) and more than 50 PDI (susceptible).

Disease severity was assessed visually, mainly by focusing on the percentage of the leaf area showing GRD symptoms on each individual plant, using a quantitative scale adapted from Waliyar *et al.* (2007) as follows: No visible symptoms on leaves (Highly Resistant), Rosette symptoms on 1- 20% leaves, but no obvious stunting (Resistant), Rosette symptoms on 21-50% leaves with stunting (Moderately Resistant), Severe symptoms on 51-70% leaves with stunting (Susceptible), and Severe symptoms on 71-100% leaves with stunting (Highly Susceptible).

**Pathogen inoculation.** Virulent aphids were collected from groundnut plants showing chlorotic and green rosette symptoms from farmers fields and NaSARRI seedling nurseries. These aphids were transferred onto symptomless potted plants raised in the screen house. A dense population of the viruliferous aphids was sustained by periodically replacing the aging diseased plants with new two week old potted plants. This allowed for maintenance of large stocks of virulent aphids needed for continued supply during the field experiment. The potted plants in the screen house showed both chlorotic and green symptoms.

Presence of all GRD causal agents in the infector plants was verified and confirmed by randomly collecting 20 leaf samples for RT-PCR laboratory tests as described by Kumar (2007). Potted plants infested with virulent aphids were transferred into the infector rows 14 days after setting up the field evaluation experiment. Within the infector rows, potted plants were placed at a spacing of 1 metre apart and replaced after two weeks following the infector row technique method described by Olurungu *et al.* (1991).

**Genetic analysis.** Analysis of genetic variability among the test materials was performed using GenStat 14<sup>th</sup> Edition (Payne *et al.*, 2011). The analyses of variance components of the test materials was further partitioned into variation due to parental genotypes,  $F_2$  progenies as well as the interaction between parents and crosses. A fixed factor model was used to determine the

combining ability of resistance to GRD (Singh and Chaudhary, 2004). The level of genetic variability to GRD among parental lines was determined as follows:

$$y_{ijk} = \mu_{ij} + GCA_i + GCA_j + SCA_{ij} + B_k + e_{ijk}$$

Where:

- $y_{ijk}$  = Observed mean  $ijk^{\text{th}}$  observation;  
 $\mu_{ij}$  = Overall mean;  
 $GCA_i$  = GCA effects of  $i^{\text{th}}$  parent  
 $GCA_j$  = GCA effects of  $j^{\text{th}}$  parent  
 $SCA_{ij}$  = SCA effects of the  $ij^{\text{th}}$  genotype;  
 $B_k$  = The effect of the  $k^{\text{th}}$  block.  
 $e_{ijk}$  = Environmental effect of the  $ijk^{\text{th}}$  observation

Two tailed t-tests were used to determine the level of significance of the GCA and SCA effects at the 0.05 level. Narrow and broad sense heritabilities were estimated using a formula suggested by Dabholkar (2006) as follows:

$$h^2 = \frac{\sigma_{GCA_i}^2 + \sigma_{GCA_j}^2}{\sigma_{GCA_i}^2 + \sigma_{GCA_j}^2 + \sigma_{SCA_{ij}}^2 + \sigma_e^2}$$

Where:

- $h^2$  = Estimated narrow sense heritability;  
 $\sigma_{GCA}^2$  = Variance due to additive effects;  
 $\sigma_{SCA}^2$  = Variance due to dominance effects;  
 $\sigma_e^2$  = Environmental error variance component;

$$H^2 = \frac{\sigma_{GCA_i}^2 + \sigma_{GCA_j}^2 + \sigma_{SCA_{ij}}^2}{\sigma_{GCA_i}^2 + \sigma_{GCA_j}^2 + \sigma_{SCA_{ij}}^2 + \sigma_e^2}$$

Where:

- $H^2$  = Estimated broad sense heritability;  
 $\sigma_{GCA}^2$  = Variance due to additive effects;

$\sigma_{SCA}^2$  = Variance due to non additive effects;  
 $\sigma_e^2$  = Environmental error variance component;

GCA effects were calculated and tested for significance from zero using a t-test at 22 degrees of freedom for the error mean square as follows:

$$t - test_{GCA\ effects} = \frac{GCA - 0}{SEM}$$

Where:

GCA = General Combining Ability value;  
SEM = Standard Error of Means;

## RESULTS AND DISCUSSION

**Mechanism of gene action.** The results for resistance to GRD showed highly significant differences ( $P < 0.001$ ) among parents (Table 1) indicating that there was wide genetic variability among the genotypes used in this study. The genotype means (Table 2) indicated that Serenut 2, Serenut 3 and Serenut 8 were resistant; while the rest were susceptible. Similar results were reported by Chiyembekeza *et al.* (1988) who suggested that these groundnut materials were bred for resistance to rosette infection under Malawian environment. The resistance to GRD in these parental lines suggests that parents have resistance to the two causal agents responsible

TABLE 1. Analysis of variance among parental genotypes for reaction to GRD

Source	D.f.	Mean square for severity	Mean square for incidence
		80 DAP	80 DAP
Rep	2	0.5	178.2
Genotype	6	2083.7***	3531***
Rep. genotype	12	12.1	356.1
Residual (plant error)	75	23	
F-Value (genotype):		172.32	9.9
F-Prob. (genotype):		$P < 0.001$	$P < 0.001$

\* Significant at  $P < 0.05$ , \*\* Significant at  $P < 0.01$ , \*\*\* Significant at  $P < 0.001$ , <sup>ns</sup> not significant at  $P = 0.05$ , Df = degrees of freedom

TABLE 2. Genotype mean for disease severity and percentage disease incidence on seven parental genotypes of drought tolerant groundnut

Genotype	Means for disease severity (%)	Mean disease incidence(%)
Serenut 2	18.1	28.9
Serenut 3	19.6	39.33
Serenut 8	17.1	25
Egolomoit	67.4	96
Red Beauty	58.3	95.1
Acholi White	67.3	93.9
Serenut 1	74.1	93.3
LSD (0.05)	6.2	33.6

\* Significant at  $P < 0.05$ , \*\* Significant at  $P < 0.01$ , \*\*\* Significant at  $P < 0.001$ , <sup>ns</sup> not significant at  $P = 0.05$ , DAP = Day after planting

for disease symptom development but not to GRAV. Similar findings have been reported by Nigam *et al.* (2012), Adu-Dapaah *et al.* (2007) in Ghana, and Chancellor *et al.* (2002) in Uganda. Lack of total immunity among the resistant parents could be attributed to the existence of GRAV that was earlier confirmed to exist in the infector rows plants and failure to have genotypes with disease severity of <1%. Serenut 1 showed the highest PDS; followed by Acholi White and Egoromoit. Okello *et al.* (2010) reported that these genotypes had no resistance genes to GRD. Therefore the resistant genotypes identified could be used to introgress resistance to GRD to the susceptible genotypes.

Table 3 shows results of F<sub>2</sub> progenies which depicted highly significant variation (P<0.001) among F<sub>2</sub> generation for reaction to GRD. The GCA and SCA mean squares were highly significant (P< 0.001) based on PDS. This indicates that both additive and non-additive gene action were important in conditioning resistance to GRD among the crosses.

**Combining ability.** Results from the analysis of variance for general and specific combining abilities for PDI and PDS are presented in Table 3. It is clear that additive gene action was more important than its non-additive gene counterpart

as depicted by the baker's ratio of 0.57 suggesting that early generation selection of genotypes for resistance to GRD would be more effective than selection in the later generation.

The general combining ability effects for the male and female parents for PDS are shown in Table 4. Three parents had negative GCA effects and four had positive GCA effects. Egoromoit had the lowest significantly negative GCA effects at P<0.001 followed by Serenut 1 and Serenut 2. The low negative GCA effects of these parents indicated that the two genotypes transferred less susceptibility to GRD, and hence, they were considered the best general combiners for resistance to GRD.

Among the resistant parents, highly significant negative GCA effects (P<0.001) were observed for Serenut 2, indicating a strong contribution to GRD resistance due to the efficacy of the resistance genes associated with resistance to GRD. Selection of progenies from parents with highly significant negative GCA effects might result in transgressive segregants with inherent potential to have better resistance than their parents (Saleem *et al.*, 2010). Red Beauty was the worst combiner, followed by Acholi White, with highly significant (P<0.001) positive GCA effects. These parents are undesirable for breeding resistant genotypes to GRD, since their

TABLE 3. Analysis of variance of reaction to GRD in F<sub>2</sub> population

Source of variation	Df	MS for PDS	MS for PDI
Replication	2	86.3 <sup>ns</sup>	361.6 <sup>ns</sup>
Genotypes	18	1272.9 <sup>***</sup>	2392.9 <sup>***</sup>
Parents (P)	6	2083.7 <sup>***</sup>	3531 <sup>***</sup>
Crosses (C)	11	935.2 <sup>***</sup>	1914.6 <sup>***</sup>
Parents versus crosses	1	123.9 <sup>ns</sup>	825 <sup>ns</sup>
GCA <sub>Susceptible</sub> (local)	3	1953.6 <sup>***</sup>	4101.2 <sup>**</sup>
GCA <sub>Resistant</sub> (exotic)	2	432.8 <sup>***</sup>	1084.8 <sup>ns</sup>
SCA/R*S	6	593.4 <sup>***</sup>	1097
Rep. cross	22	31.4	359.5
Error (plant by plant)	131	28.9	

\* Significant at P<0.05, \*\* Significant at P<0.01, \*\*\* Significant at P<0.001, <sup>ns</sup>not significant at P = 0.05, Df = degrees of freedom

TABLE 4. Estimates of general combining ability effects for reaction to GRD by groundnuts parents

Parental genotype	Percentage disease severity
<b>Exotic resistant</b>	
Serenut 2	-6.9 <sup>***</sup>
Serenut 3	3.7
Serenut 8	3.2
S.E.	1.6
<b>Local susceptible</b>	
Acholi white	10.5 <sup>***</sup>
Red beauty	14.0 <sup>***</sup>
Egolomoit	-17.2 <sup>***</sup>
Serenut 1	-7.2 <sup>***</sup>
S.E.	1.8

\* Significant at P<0.05, \*\* Significant at P<0.01, \*\*\* Significant at P<0.001, <sup>ns</sup>not significant at P = 0.05

progenies show increased levels of susceptibility. Also, in spite of Serenut 8 (mean:17.1) and Serenut 3 (mean:19.6) having low mean disease severity values for GRD (Table 2), they combined badly for resistance to GRD suggesting that Serenut 8 and Serenut 3 were poor in transferring resistance to GRD. Choice of potential genotypes for future breeding work could therefore be based on both the GCA and mean values.

Specific combining ability effects for reaction to GRD among  $F_2$  generation populations indicated only four out of twelve crosses having negative SCA effects for PDS (Table 5). Based on SCA estimates two superior crosses, Serenut 1 x Serenut 2 (mean:11.8\*\*\*), and Egoromoit x Serenut 3 (mean:19.1\*) with statistically significant negative SCA effects for PDS were observed. Negative SCA effects depicted better performance of a specific cross over and above the expected performance based on the GCA of their respective parents.

However, one cross, Red Beauty x Serenut 8 (mean:15.9), performed better than expected, despite having inferior parental background that showed highly positive GCA effects values. The crosses; Serenut 1 x Serenut 8 (mean:15.3), Red Beauty x Serenut 2 (mean:10.5) and Egolomoit x Serenut 8 (mean:8.4) had the highest positive SCA

effects for percentage disease severity at 80 DAP. Crosses with significant SCA effects indicated that such crosses were markedly resistant or susceptible to GRD, than would be predicted from their parent genotypes. Crosses with high positive SCA effects are poor specific combiners, hence would not constitute good materials for any breeding programme due to their potential to produce high frequencies of susceptible progenies in future generations (Acquaah, 2008; Falconer and Mackay, 2009).

**Heritability estimation.** Broad sense and narrow sense coefficients of genetic determination (BS-CGD and NS-CGD) for resistance to GRD among  $F_2$  populations were calculated on a single-plot basis (Table 6). BS-CGD was used to approximate broad sense heritability (H) while the NS-CGD approximated narrow sense heritability ( $h^2$ ), since the crosses used were fixed in effect (Singh and Chaudhary, 2004).

High broad sense heritability of 93% were observed in this study giving a reflection of the magnitude of genetic contribution towards the phenotypic variance (Falconer and Mackay, 2009). This indicates that there was consistency of disease scores in the different replications.

However, high heritability values in the broad sense for PDS were in contrast with those reported by Adu-Dapaah *et al.* (2007) in Ghana. They reported broad sense heritability of 75% resistance to GRD based on percentage disease scores in advance breeding populations. The contrasting results might be due to the difference in the generations on which GRD evaluation was done. In this study, the heritability estimates were derived from  $F_2$  population, which was still undergoing segregation; while Adu-Dapaah *et al.* (2007) used  $F_4$  generation and a local genotype that were more stable and homozygous for GRD resistance. The fact that Adu-Dapaah *et al.* (2007) used a population that had undergone 3 successive generations of selfing implies that the non-additive components in their population must have been greatly reduced hence broad sense heritability values getting closer to the narrow sense heritability (Dabholkar, 2006).

The narrow sense heritability observed in this study (53%) is shown in Table 6. It is apparent that resistance to GRD was highly heritable, since

TABLE 5. Estimates of specific combining ability effects for reaction to GRD by  $F_2$  generation of drought tolerant groundnut

Crosses	Percentage disease severity
Acholi White x Serenut 2	4.5 <sup>ns</sup>
Acholi White x Serenut 3	3.2 <sup>ns</sup>
Acholi White x Serenut 8	-7.7 <sup>*</sup>
Red Beauty x Serenut 2	10.5 <sup>*</sup>
Red Beauty x Serenut 3	5.4 <sup>ns</sup>
Red Beauty x Serenut 8	-15.9 <sup>***</sup>
Egolomoit x Serenut 2	2.0 <sup>ns</sup>
Egolomoit x Serenut 3	-10.3 <sup>*</sup>
Egolomoit x Serenut 8	8.4 <sup>*</sup>
Serenut 1 x Serenut 2	-16.9 <sup>***</sup>
Serenut 1 x Serenut 3	1.7 <sup>ns</sup>
Serenut 1 x Serenut 8	15.3 <sup>***</sup>
S.E.	3.2

\* Significant at  $P < 0.05$ , \*\* Significant at  $P < 0.01$ , \*\*\* Significant at  $P < 0.001$ , <sup>ns</sup> not significant at  $P = 0.05$

TABLE 6. Heritability estimates for percentage disease severity and incidence by F<sub>2</sub> generation of drought tolerant groundnut

Source of variation	Df	PDS		PDI	
		MS	VC	MS	VC
GCA/Susceptible (local)	3	1953.6 <sup>***</sup>	213.6	4101.2 <sup>**</sup>	415.8
GCA/Resistant (exotic)	2	432.8 <sup>***</sup>	33.5	1084.8 <sup>ns</sup>	60.4
SCA/R*S	6	593.4 <sup>***</sup>	187.3	1097 <sup>*</sup>	246.1
Error	22	31.4	31.4	359.5	359.5
Baker's ratio		0.57		0.66	
NS-CGD (genotype mean basis)		0.53		0.44	
BS-CGD (genotype mean basis)		0.93		0.67	

\*Significant at P<0.05, \*\* Significant at P<0.01, \*\*\* Significant at P<0.001, <sup>ns</sup>not Significant at P = 0.05, Df = Degree of freedom, PDS = Percentage disease severity, PDI = Percentage disease incidence MS = Mean squares, VC = Variance component

it was more than 50% (Singh and Chaudhary, 2004). This further suggested that resistance to GRD is conditioned by both additive and non-additive gene action. Therefore, selection for resistance to GRD should be effective due to close correspondence between the phenotype and genotype since environment had a small impact on the phenotype (Fehr, 1987). This means that dependence on phenotypic predictions values for GRD as a breeding strategy may be informative and reliable (Chahal and Gosal, 2002; Dabholkar, 2006;).

On the other hand, based on PSI data, narrow sense heritability was 44% while broad sense heritability was 67% (Table 6). Similar findings have been reported by Van der Merwe *et al.* (1998). They reported broad sense heritabilities for GRD PDI as 63 and 74% at high and medium disease pressures respectively, among twelve entries in Malawi. In contrast, Adu-Dapaah *et al.* (2004) reported intermediate broad sense heritability of 54%, after assessing advanced lines for GRD incidence towards harvesting time. High broad sense heritability for PDI in this study and the previous related studies imply that the environment has limited influence on expression of resistance to GRD among the cross (Acquaah, 2008). Therefore this affirms the fact that there was consistency in GRD disease scores across the different replications. The slightly moderate narrow sense heritability value for PDI of 44% revealed by the present study implied that progeny performance could be partly predicted from their parental performance although

individual cross evaluation would still be meaningful.

## CONCLUSION

The parent lines used in this study show variable responses to GRD implying that these materials are genetically diverse. Two genotypes; Serenut 2 and Serenut 3 with resistance to GRD are good sources of resistance for future breeding efforts. However, none of the resistant parents has 0% PDS, implying that there is no total resistance to GRD among the resistant parents; which could be attributed to the possible presence of GRAV, an important GRD causal agent.

From this study, significant general combining ability observed reflected the effective contribution of additive genetic variance towards GRD resistance as revealed by the baker's ratio of 0.57. This indicated the preponderance of additive gene action over the non additive component for resistance to GRD implying that early generation selection and testing would be more effective.

Narrow sense heritability values being slightly higher than 50% for PDS, and 44% for PDI indicate that performance of crosses would be partly predicted by both parental GRD score values and individual cross means. The high broad sense heritability estimates of between 67 and 93% reported in this study are indicative of low environmental interaction and influence in the inheritance of GRD resistance.

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