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An evaluation of the nature and magnitude of gene effect of resistance to rosette virus disease in groundnut (A*rachis hypogaea* L.)

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Groundnut rosette disease is one of the most damaging diseases militating against groundnut production in sub-Saharan Africa. The disease cause up to 100% yield loss whenever epidemic occurs. The most effective, economic and environmental friendly method to control the disease is through genetic resistance. Knowledge on inheritance of resistance to the rosette disease is required to accelerate breeding of resistant varieties. A study was conducted to understand the nature and magnitude of gene effect of resistance to the disease. Thus F_1, RF_1, F_2, RF_2, BC_1, RBC $_1$, BC_2, and RBC_2 progenies were derived from crosses of Otuhia × Manipintar, Otuhia × Shitaochi, ICGV 01276 × Manipintar, and ICGV 01276 × Shitaochi along with their parents were evaluated in a randomized complete block design at CSIR - CRI, Fumesua, under artificial infection. Generation mean analysis revealed that additive gene action effect was predominant on the resistance to the disease in all the crosses. Additive by dominance was the only form of non-allelic interaction observed to be significant in ICGV 01276 × Manipintar cross. Reciprocal differences suggested the presence of maternal effect involved in the inheritance of resistance to groundnut rosette disease. Estimates of broad and narrow sense heritability indicated that genetic effect was larger than the environmental effects in this study. Disease diagnosis using TAS ELISA revealed the presence of groundnut rosette assistor virus (GRAV) antigens in the resistant samples analyzed. Resistant genotypes containing GRAV were considered to be resistant to the GRV and its Sat-RNA, but not the GRAV which causes no obvious symptoms by itself. Pure line breeding with selection from early generation is suggested for the improvement of resistance to rosette virus disease, because additive genetic effect contributed significantly in controlling the inheritance of resistance to groundnut rosette disease (GRD).

Key words: Additive, dominance, heritability, inheritance, heterosis, rosette disease.

INTRODUCTION

Groundnut is an important food crop providing income and livelihoods to many of the farmers in Africa. Its production is constrained by several biotic and abiotic factors such as diseases, pest, aflatoxin and drought.

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Groundnut rosette disease (GRD) has been described as the most damaging disease of groundnut in sub-Saharan Africa causing yield losses approaching 100% whenever an epidemic occurs (Ntare et al., 2002). Yield loss due to GRD depends on the growth stage at which infection occurs. Seedling stage infection leads to 100% yield loss whilst infection at the pod filling stage causes negligible effects (Naidu et al., 1999a; Waliyar et al., 2007). The disease contributes to an annual loss of US $ 156 million across Africa (Nigam et al., 2012).

The disease is caused by three agents, these include groundnut rosette virus (GRV), a satellite RNA of GRV and groundnut rosette assistor virus (GRAV) (Bock et al., 1990). *Aphis craccivora* Koch transmits the virus complex in a persistent and circulative manner (Okusanya and Watson, 1966). Variants of Sat-RNA have been shown to be responsible for different rosette symptoms, such as green and chlorotic rosette (Murant and Kumar, 1990; Taliansky and Robinson, 1997). The complex interaction of these agents in causing the disease makes it a unique and fascinating virus disease whose origin and perpetuation in nature, in spite of substantial advance in knowledge, still remain inconclusive (Waliyar et al., 2007).

Farmers have adopted several cultural, biological and chemical methods to curb the spread of the disease but the adoption rate of these methods has been very low because they are not economical and effective (Olorunju et al., 2001). However, the most economic, ecological and environmentally–friendly method of control is the use of rosette resistant lines (Adu-Dapaah et al., 2004). Recent reports have shown that resistance is directed towards GRV and Sat-RNA, but not GRAV (Waliyar et al., 2007). Breeding for resistance to diseases remains a principal focus in the groundnut breeding programme in Ghana. Although genetics of resistance to the disease has been reported, the mechanism of resistance may be different in the sources of parents. To facilitate the design of breeding strategies to develop resistant cultivars to GRD, it would be beneficial to understand more completely the mode of inheritance of resistance to the disease. The objectives for the study were to determine the mechanism of gene action, contribution of maternal effect and heritability of resistance to GRD.

MATERIALS AND METHODS

Genetic materials

Parent materials used were selected based on germplasm screening of resistance to the disease by the Council for Scientific and Industrial Research (CSIR)-Crops Research Institute, Kumasi, Ghana. The resistant genotypes were Otuhia and ICGV 01276 while the susceptible genotypes were Manipintar and Shitaochi.

Hybridization

The conventional technique for hybridization in groundnut which was described by Norden (1973) and modified by Nigam et al. (1980) was adopted for this work. Direct, reciprocal and backcrossing crosses were carried out for developing the necessary progenies for evaluation. Thus F_1, Reciprocal F_1, F_2, Reciprocal F_2, BC_1, Reciprocal BC_1, BC_2, and Reciprocal BC_2 progenies were derived from crosses of Otuhia × Manipintar, Otuhia × Shitaochi, ICGV 01276 × Manipintar, and ICGV 01276 × Shitaochi.

Disease evaluation of parental and progenies

Disease evaluation of the parental and the progenies developed were done under high disease pressure environment created through aphid infestation on the field of Council for Scientific and Industrial Research (CSIR)-Crops Research Institute, Kumasi, Ghana. The trials were laid out in randomized complete block design with 3 replications. Each replicate consisted of one plot of each of the Parents, F_1, RF_1, backcross and two plots of each F_2 and RF_2 generations. Each plot was made up of a row, 2 m long with 0.4 m between rows and 0.2 m within plants giving 10 plants per row. Plants were sown at a rate of 1 seed per hill. Aphid colonies were reared on a highly infested genotype Manipintar in netted cages prior to planting of the experiments. Triple antibody sandwich enzyme linked immunosorbent assay (TAS ELISA) tests were done to detect the presence of (GRAV on those infested plants before the aphids colonies were collected from them. Five wingless viruliferous aphids were transferred onto 7 to 14 days old seedlings on the test materials using wet camel's hair brush as described by Naidu and Kimmins (2007). This was done to ensure effective inoculation by the vectors. Each of the test plants was evaluated for GRD symptoms at weekly interval for the first four weeks and every two weeks thereafter using a 1-5 rating scale (Pande et al., 1997; Olorunju et al., 2001) as follows: 1= No visible symptoms on leaves (Highly Resistant), 2= Rosette symptoms on 1 to 20% leaves, but no obvious stunting (Resistant), 3= Rosette symptoms on 21-50% leaves with stunting (Moderately Resistant), 4= Severe symptoms on 51 to 70% leaves with stunting (Susceptible), and 5= Severe symptoms on 71 to 100% leaves with stunting (Highly Susceptible).

Disease diagnosis

Leaf samples for serological test were taken from field plants rated 1 to 4 (Susceptibility and resistance). An indirect triple antibody sandwich- enzyme –linked immunosorbent essay method which entails the usage of beet western yellow virus (*Luteovirus*) antiserum was used for the detection of GRAV antigen in the various samples (Naidu et al., 1999b). Purified polyclonal antiserum (LgG) (AS-0049) raised against purified preparation of GRAV was diluted at recommended dilution in coating buffer, 200 µl was added to each well of a microliter plate. Each sample was allotted to two wells in the 96 well microplate. 200 µl of the monoclonal antibodies (MAb) (AS0049/1) to beet western yellow virus, which reacts with the GRAV coat protein was used as the secondary antibody. Since the monoclonal antibodies were not labeled, a secondary, animal species (mouse) antibody was used to react with the bound MAb. This anti mouse (RAM) antibody was labeled with alkaline phosphatase (AP) as reporter group for detecting the antibody. 200 µl aliquot of freshly prepared substrate [10 mg p-nitrophenyl phosphate (sigma, fluke)] dissolved in 10 ml of substrate buffer was added to each well of the plates. The plates were incubated at room temperature for 30-50 min to obtain a clear reaction. Samples were then assessed by visual and spectrophotometric measurement of absorbance at 405 nm. All TAS-ELISA kits and protocol used were supplied by the Leibniz Institute DSMZ, Germany.

Statistical and genetic analyses

Data collected were subjected to analysis of variance (ANOVA

Table 1. Mean rosette resistance scores, standard error and variance in ten generations of direct and reciprocal crosses in groundnut.

Generation	NP	O/S		O/M		I/S		I/M	
		Mean ± SE	S^2	Mean ± SE	S^2	Mean ± SE	S^2	Mean ± SE	S^2
P1	30	1.13 ± 0.06^{a}	0.12	1.13 ± 0.06^{a}	0.12	1.67 ± 0.08^{a}	0.23	1.67 ± 008 ^{a}	0.23
P2	30	4.00 ± 0.05^{g}	0.07	4.13 ± 0.06^{e}	0.12	4.00 ± 0.05^{g}	0.07	4.13 ± 0.06^{e}	0.12
MP		2.67		2.73		2.84		2.90	
F1	30	2.20 ± 0.07^{d}	0.17	3.03 ± 0.03^{d}	0.03	2.60 ± 0.09^{cd}	0.25	2.67 ± 0.11^{c}	0.27
RF1	30	2.63 + 0.09^{e}	0.24	2.87 ± 0.06^{d}	0.12	3.20 ± 0.07^{f}	0.17	3.10 ± 0.06^{d}	0.09
F2	60	1.57 ± 0.10^{b}	0.55	2.40 ± 0.15^{c}	1.29	2.23 ± 0.14^{b}	1.21	2.03 ± 0.13^{b}	0.95
RF2	60	2.13 ± 0.04^{cd}	0.12	3.13 ± 0.16^{d}	1.47	3.00 ± 0.13^{ef}	1.02	2.43 ± 0.13^{c}	1.00
BCI	30	2.17 ± 0.17^{d}	0.83	2.13 ± 0.13^{c}	0.53	2.70 ± 0.11^{de}	0.36	2.63 ± 0.12^{c}	0.45
RBCI	30	1.80 ± 0.07^{c}	0.17	1.67 ± 0.14^{b}	0.58	2.30 ± 0.09^{bc}	0.22	3.06 ± 0.20^{d}	1.17
BC2	30	2.67 ± 0.08^{e}	0.23	2.97 ± 0.09^{d}	0.24	2.73 ± 0.13^{de}	0.5	2.60 ± 0.16^{c}	0.73
RBC2	30	3.27 ± 0.08^{f}	0.20	2.93 ± 0.10^{d}	0.34	3.00 ± 0.15^{ef}	0.76	3.13 ± 0.15^{d}	0.67

*NP= Number of plants evaluated, O/S= Otuhia x Shitaochi, O/M= Otuhia x Manipintar. *I/S= ICGV 01276 x Shitaochi, I/M= ICGV 01276 x Manipintar. *SE= Standard error. *S^2 = Variance.

using GENSTAT statistical package (Discovery Edition 4). Means were separated using least significant difference (LSD) at 5%.

Generation mean analysis (GMA) (Mather and Jinks, 1982) was carried out to determine the types of gene action influencing the expression of groundnut rosette virus disease resistance trait. Gene effect based on a six parameter model was estimated using the PBTools, version 1.4, 2014. Weighted regression approach was used for the generation mean analysis. Two full models were fitted to the data. The first was "mean = 0 + m + a + d + aa + ad" and the other was "mean = 0 + m + a + d + aa + dd". For each model, backward regression procedure was used to obtain the best model. Mather and Jinks (1982) model describes the phenotype in terms of the mid- parental values [m], additive effects [a], dominance effects [d], additive by additive [aa], additive by dominance [ad], and dominance by dominance [dd] epistatic interaction effects. Broad sense (h^2_b) and narrow-sense (h^2_n) heritabilities were estimated using the variance component method (Wright, 1968) and variances of F_2 and back cross generations (Warner, 1952), respectively, as:

$$(h^2_b) = \{VF_2 – [(VP_1 + VP_2 + 2VF_1) /4]\}/ VF_2$$

$$(h^2_n) = [VF_2 – (VBC_1 + VBC_2) /2] / VF_2$$

RESULTS

Mean values, standard errors and variances for resistance to GRD of the four crosses are presented in the Table 1. Parents used in this research showed significant differences in rosette virus reactions. Otuhia (P_1) was the most resistant, followed by ICGV 01276 (P_1) and Manipintar (P_4) was highly susceptible. Means of the direct and reciprocal first filial generation (F_1) were significantly different in three of the crosses, except in Otuhia × Manipintar cross. The F_1 and F_2 were more resistant than that of their respective reciprocal crosses. The mean of the F_{1s} was less than the mid-parent value but higher than the mean of the parent with lowest disease score (P_1). Significant mean differences were detected in all the Backcrosses except that of the Otuhia and Manipintar backcross, with reciprocal cross of Otuhia × Manipintar recording the highest mean score (Table 1). Mean scores for BC_1 and BC_2 were significantly different from each other in two of the crosses that is, Otuhia × Shitaochi and Otuhia × Manipintar.

Results from the TAS-ELISA showed that all the 23 susceptible and 9 resistant samples tested positive for the GRAV causal agent, indicating that GRAV antigen occurred frequently in all the samples (Table 2).

Generation mean analysis for gene effect controlling inheritance of resistance to the groundnut rosette disease is presented in Table 3. The results provide estimates of the main and first order gene interaction. Mid parent value ranged from 0.14 to 4.01, it was lowest in the cross of Otuhia × Shitaochi and high in ICGV 01276 × Shitaochi. Additive gene action was the only significant gene action in the crosses of Otuhia × Shitaochi, Otuhia × Manipintar and ICGV 01276 × Shitaochi (Table 2). In contrast both additive and additive by dominance were significant in the cross of ICGV 01276 × Manipintar. Additive by dominance non-allelic interaction was the only significant non allelic interaction in all the crosses.

Table 4 shows broad sense and narrow sense heritability (based on mid parent value) for the groundnut rosette resistance in four different crosses. Heritability estimates varied between crosses. The broad sense heritability ranged from 76-95% in the various crosses with Otuhia × Manipintar cross recording the highest broad sense heritability value. Mean broad sense heritability in all the four crosses was 83% whilst mean narrow sense heritability was 43%. The highest narrow sense heritability value recorded was 67% with the cross Otuhia × Manipintar, in sharp contrast with a narrow sense heritability of 4% recorded with the cross Otuhia × Shitaochi (Table 4).

Table 2. Detection of groundnut rosette assistor virus (GRAV) by ELISA of groundnut genotypes resistance and susceptible to groundnut rosette virus (GRV).

S/N	Genotype	Field status	MOD	GRAV
1	(ICGV 01276 × Manipintar) × Manipintar (S_1)	Susceptible	0.570	+
2	(Shitaochi × ICGV 01276) × ICGV 01276	Susceptible	0.595	+
3	Manipintar × Otuhia	Susceptible	0.587	+
4	Otuhia × Manipintar	Susceptible	0.589	+
5	Shitaochi × Otuhia	Susceptible	0.580	+
6	(Manipintar × Otuhia) × Manipintar (S_1)	Susceptible	0.567	+
7	(ICGV 01276 × Manipintar) × Manipintar (S_2)	Susceptible	0.588	+
8	(Otuhia × Shitaochi) × Shitaochi	Susceptible	0.599	+
9	(ICGV01276 × Shitaochi) × Shitaochi	Susceptible	0.594	+
10	(Otuhia × Manipintar) × Manipintar	Susceptible	0.598	+
11	(Otuhia × Manipintar) × Otuhia	Susceptible	0.599	+
12	(ICGV01276 × Shitaochi) × ICGV 01276 (S_1)	Susceptible	0.584	+
13	(Manipintar × Otuhia) × Manipintar (S_2)	Susceptible	0.587	+
14	ICGV 01276	Susceptible	0.597	+
15	Manipintar	Susceptible	0.595	+
16	Shitaochi	Susceptible	0.594	+
17	Otuhia (S_1)	Susceptible	0.593	+
18	(Shitaochi × Otuhia) × Shitaochi	Susceptible	0.574	+
19	(ICGV 01276 × Shitaochi) × ICGV 01276 (S_2)	Susceptible	0.592	+
20	(Manipintar × ICGV 01276) × Manipintar (S_1)	Susceptible	0.593	+
22	Shitaochi × ICGV 01276	Susceptible	0.585	+
23	Manipintar × ICGV 01276	Resistance	0.603	+
24	ICGV 01276 × Manipintar	Resistance	0.594	+
25	(Manipintar × Otuhia) × Otuhia	Resistance	0.582	+
26	Otuhia (S_2)	Resistance	0.582	+
27	(Otuhia × Shitaochi) × Shitaochi	Resistance	0.593	+
28	(ICGV 01276 × Shitaochi) × ICGV 01276 (S_2)	Resistance	0.614	+
29	(Manipintar × ICGV 01276) ICGV 01276	Resistance	0.591	+
30	(Shitaochi × Otuhia) × Otuhia	Resistance	0.578	+
31	(Manipintar × ICGV 01276) × Manipintar	Resistance	0.593	+
32	Shitaochi × ICGV 01276	Susceptible	0.601	+
37	Negative Control	……	0.260	
38	Buffer	……	0.283	
39	Positive	……	0.588	

[a]MOD = Mean Optical Density (Average two wells for each sample). [b]+ = Positive (GRAV present). [c] - = Negative (GRAV absent). [d](S_1) = Sample One (1). [e](S_2) = Sample Two (2). [f]Positive samples had to have OD values twice as large as the negative control.

DISCUSSION

The significantly different mean GRD resistance scores detected among some of the direct and reciprocal crosses indicates that maternal effect played a major role in the GRD resistance. The inheritance of resistance to the rosette virus disease might therefore not be attributed solely to nuclear gene control. This suggests that the choice of maternal parent is relevant in hybridization programme that focuses on the improvement of groundnut for resistance to the disease. Generation mean analysis using the weighted regression approach was adequate to explain the genetic control of resistance to groundnut rosette disease in the four crosses involving two resistance parents and two susceptible parents. Additive gene effect was of the greatest importance in crosses of Otuhia × Shitaochi, Otuhia × Manipintar and ICGV 01276 × Shitaochi for resistance to GRD. On the other hand, both additive and additive × dominance gene effect were important for inheritance of rosette resistance in ICGV 01276 × Manipintar cross. With respect to epistatic effects, additive by dominance gene effect was the only non - allelic interaction observed to play a significant role in the inheritance of resistance to

Table 3. Estimate of gene effects for groundnut rosette resistance in *Otuhia/ Shitaochi* cross.

Parameter	Crosses			
	Otuhia (R) × Shitaochi (S)	Otuhia (R) × Manipintar (S)	ICGV 01276 (R) × Manipintar (S)	ICGV 01276 (R) × Shitaochi (S)
M	0.14 ± 1.30	3.60 ± 1.06*	0.93 ± 1.07	4.01 ± 1.04*
A	-1.43 ± 02.5*	-1.50± 0.19*	-1.23 ± 0.21*	-1.17± 0.29*
D	3.60 ± 3.37	-2.88 ± 2.63	3.62 ± 2.60	-3.74 ± 2.78
aa	2.40 ± 1.19	-0.96 ± 1.04	1.98 ± 1.05	-1.24 ± 1.00
ad	1.50 ± 1.15	1.10 ± 0.77	2.32 ± 0.75*	1.57± 1.00
dd	-1.17 ± 2.2	2.18 ± 1.65	-1.62 ± 1.62	2.75± 1.83

*Significant ≤ 0.05. [m]= mean, [a] = additive, [d]= dominance, [aa]= additive*additive, [ad]= additive*dominance, [dd]= dominance*dominance effect, R= Resistant, S= Susceptible.

Table 4. Percentage broad and narrow sense heritability of rosette virus disease resistance in groundnut crosses.

Cross	Heritability (%)	
	Broad sense	Narrow Sense
Otuhia × Shitaochi	76	4
Otuhia × Manipintar	95	67
ICGV 01276 × Manipintar	84	65
ICGV 01276 × Shitaochi	77	37

groundnut rosette disease. In general, additive and additive by dominance gene effect were the only forms of gene effects involved in the inheritance of resistance to the disease in this study. On the contrary however, Nalugo et al. (2013) found the interaction of dominance by dominance with duplicate epistatic effect to be the only type of epistatic effect on the resistance to groundnut rosette disease. Probably this contradiction would have come as a result of the differences in the parent genotypes which were used in the studies. The presence of epistasis has important implications for any plant breeding program. Due to epistasis, selection has to be delayed after several cycles of crossing until a high level of gene fixation is attained. The negative sign for additive effects depend on which parent is chosen as P_1 (Cukadar-Olmedo and Miller, 1997; Edwards et al., 1975; Azizi et al., 2006). The sign for dominance effect is a function of the F_1 mean value in relation to the mid-parental value and indicates which parent is contributing to the dominance effect (Cukadar-Olmedo and Miller, 1997). It is suggested that pure line selection at early generation would be appropriate due to the large significant contribution of additive gene effect to the inheritance of resistance to GRD. Whereas selection at later generation would be appropriate for the additive by dominance type of epistasis for that fact that it will allow favorable gene combinations to be in a homozygous state before practicing final selection (Azizi et al., 2006).

Detection of GRAV antigen in the resistant plants tested, is in agreement with results obtained by Bock and Nigam (1988) who observed GRAV antigen present in all plants of six rosette-resistant groundnut lines that had been exposed to aphid inoculation in Malawi. These lines were RG 1, RMP 91, RMP 40, RMP 93, RRI/24 and RRI/16. Similar findings were also reported by Olorunju et al. (1992), who reported that GRAV was detected in 11 of 15 symptomless plants of R × R and RMP × M1204.781 crosses. The detection of GRAV antigen in resistance genotype can be attributed to the lower concentration of GRV (SatRNA) in the genotype resulting in no symptoms expression as compared to the susceptible ones (Olorunju et al., 1992). Naidu and Kimmins (2007) reported that GRV and its Sat-RNA may not always occur in the same tissue together with GRAV which explain the transmissions of GRAV alone. All resistance samples tested positive, indicating that genes conferring resistance to GRV and its sat RNA were successfully introgressed in those varieties but those genes did not confer resistance to the GRAV. These observations infer that symptoms alone cannot be a reliable basics for screening of groundnut plants for their resistance to the causal agents of the disease, as demonstrated by this study.

High average broad sense heritability of 86% observed in the study for the trait indicated that genetic variation was high and that it will respond readily to selection. This findings is in agreement with a high realized broad sense heritability reported by Kayondo et al. (2014).

The generally high broad sense heritability estimate in all the four crosses indicates that the environment in which the plants were evaluated had a lower effect on the expression of resistance to GRD. A high narrow sense heritability recorded for Otuhia × Manipintar and ICGV 01276 × Manipintar crosses suggested that the trait is largely governed by additive genes and may not require more cycles for selection.

Conclusions

Detection of GRAV antigens in the resistant samples suggests that introgressed gene conferred resistance to GRV and its sat RNA, but not GRAV. The significant difference between the direct and reciprocals suggested that maternal effect contributed significantly to the inheritance of the resistance to the rosette disease. This indicates that when developing breeding populations for resistance to GRD, the choice of a maternal parent is very important. Additionally, generation mean analysis revealed that inheritance of resistance to the disease is control by both additive and non-additive gene action. The additive gene component was predominant over the non-additive. Additive by dominance form of non-allelic interaction was the only form of epistasis revealed in this study. Due to the significant additive gene action it is suggested that selection from early generation would be effective. High heritability estimates suggest low environment influence on resistance to GRD

Conflict of Interests

The authors have not declared any conflict of interests.

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