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Additional sources of resistance to groundnut rosette disease in groundnut germplasm and breeding lines

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Summary

Groundnut rosette, a virus disease of groundnut (*Arachis hypogaea*) transmitted by the aphid, *Aphis craccivora* Koch, reduces yield in susceptible cultivars by 30-100%. Additional sources were sought in germplasm accessions involving 2301 lines from different sources and from 252 advanced breeding lines derived from crosses involving earlier identified sources of resistance to rosette. The lines were evaluated in field screening trials using an infector row technique during 1996 and 1997 growing seasons. Among the germplasm lines, 65 accessions showed high levels of resistance while 134 breeding lines were resistant. All rosette disease resistant lines were susceptible to groundnut rosette assistor virus. This work identified germplasm and breeding lines that will contribute to an integrated management of groundnut rosette disease. These new sources also provide an opportunity to eliminate yield losses due to the rosette disease.

Key words: Groundnut (peanut), resistance to rosette, germplasm

Introduction

Groundnut rosette disease is regarded as the most destructive virus disease of groundnut (*Arachis hypogaea* L.) in sub-Saharan Africa (Reddy, 1991). The major areas of disease occurrence include Burkina Faso, Ghana, Nigeria, Malawi, Mozambique and Uganda. The aphid, *Aphis craccivora* Koch, transmits the disease in a persistent manner (Okusanya & Watson, 1966). It is caused by a complex of three agents: groundnut rosette virus (GRV), genus *Umbravirus* (Murant *et al.*, 1995) and its satellite RNA (sat RNA, Blok *et al.*, 1995) and groundnut rosette assistor virus (GRAV), genus *Luteovirus* (Casper *et al.*, 1983; Reddy *et al.*, 1985; Murant, 1989). On their own, either GRAV or GRV cause symptomless infection. All the agents must be present together in the host plant for successful transmission of the disease by the vector. Symptoms associated with the disease are variable and two types (chlorotic and green rosette) are known. These symptoms are largely due to sat RNA (Murant *et al.*, 1988) and variants of sat RNA are responsible for different forms of the rosette disease (Murant & Kumar, 1990). Chlorotic rosette is the most prevalent type in southern and eastern Africa (Subrahmanyam *et al.*, 1997), while green rosette is most common in West Africa (Subrahmanyam *et al.*, 1991).

Rosette epidemics are sporadic, causing yield losses ranging from 10% to 30% each year in endemic areas, but reach 100% whenever the disease occurs in epidemic proportions. For example, in Nigeria alone, the rosette epidemic of 1975 destroyed 0.7 million ha of groundnut incurring a loss of approximately US$ 250 million in regional trade (Yayock *et al.*, 1976). Subsequent epidemics have limited crop production in West Africa.

Research on the development of groundnut cultivars with resistance to rosette was initiated in the early 1950s by the French Institut de Recherches pour les Huiles et Oléagineux (IRHO) in West Africa. Sources of resistance to rosette were first discovered in groundnut landraces of late-maturing Virginia (*A. hypogaea* L. subsp. *hypogaea* var. *hypogaea*) from Burkina Faso (then Haut Volta) and Cote d'Ivoire in 1952 (Sauger & Catherinet, 1954). These sources formed the basis for the rosette resistance breeding programmes throughout Africa. These attempts resulted in the development of long-duration varieties such as 69-101 (130 days to maturity), RMP 12, RMP 40 and RG 1 (140-150 days) and early-maturing (90 days) Spanish (*A. hypogaea* L subsp. *fastigiata* var. *vulgaris*) types such as KH149 A, KH 241C, KH 241 D, CN94C and QH 243C (Bockelée-Morvan, 1983). Resistance among these cultivars was found to be effective against both chlorotic and green rosette and was governed by two independent recessive genes (de Berchoux, 1960; Nigam & Bock

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1990). Unfortunately, the rosette resistant long-duration varieties are not adapted to the short growing seasons of the dry savannah of West Africa, where the bulk of the crop is grown. The few short-duration rosette-resistant varieties were not widely adopted by farmers. The challenge was to widen the genetic base by combining groundnut rosette disease resistance with early maturity (90 to 110 days), high yielding Spanish types suitable for smallholder farmers in semi-arid tropics of Africa. The International Crops Research Institute for Semi-Arid Tropics (ICRISAT) launched a programme in Malawi in the early 1980s and in West Africa in the late 1980s to develop such varieties. These programs have produced a wide range of early-, medium- and late maturing varieties suitable for various cropping systems.

A number of accessions in the global groundnut germplasm have been screened for resistance to GRV and GRAV with several sources of resistance being reported (Subrahmanyam *et al.*, 1998). The objective of this research was to identify additional sources of resistance to rosette disease from groundnut germplasm and breeding lines.

Materials and Methods

Sites

Field trials were conducted in 1996 and 1997 growing seasons (June to October) at Samaru (latitude 11°8'N, longitude 7°E) and Bagauda (11°40'N, 8°30'E) in northern Nigeria. A basal dose of 100 kg ha^{-1} of single super phosphate fertiliser was incorporated into the soil during land preparation. Seeds were hand sown during the last week of June each year and trials were all rainfed.

Plant material

The plant material consisted of 2301 accessions obtained from the International Crops Research Institute for Semi-Arid Tropics (ICRISAT) world groundnut germplasm collection and 252 breeding lines from ICRISAT and Institute for Agricultural Research (IAR), Nigeria, breeding programmes (Table 1). Lines with the prefix 'ICG are germplasm lines and those with Prefix 'ICGV' and/or 'IS' and 'SM' are advanced breeding lines of diverse pedigrees developed by ICRISAT in West Africa and Malawi, respectively. Others are from IAR.

1996 season

In 1996, germplasm accessions were screened in non-replicated single row plots 2 m long in a rosette disease nursery. Breeding lines divided into three maturity groups (late-, medium-and early maturing) were screened in replicated single row plots 2 m long in a randomised complete block design with two replications. Due to limited seed quantities, the germplasm accessions were evaluated at Samaru and the breeding lines were evaluated at Samaru and Bagauda. An infector row technique described by Bock & Nigam (1988) was used. This technique results in a disease incidence of 99% in susceptible entries. A large number of seedlings of a susceptible groundnut (cv. 55-437) were raised in the screen house and inoculated with GRV, using a screen house culture of viriluferus aphids, which had been reared on GRV-infected plants. Infector rows of 55-437 were arranged throughout the trial, one infector row flanking every two-test rows. A resistant groundnut (cv. RMP12) was sown throughout the nursery every 20 rows. Ten days after sowing, potted spreader plants (cv. 55-437) showing severe rosette symptoms and heavily infested with aphids were transplanted in the infector rows (one plant per 2 m row). Eight days after inoculation, scoring for rosette was done at weekly intervals during the first 4 wk and every 2 wk thereafter. Disease severity (DS) was rated for each plant based on a scale of 1 to 5: 1 = plants with no visible disease symptoms on foliage, 2 = plants with obvious rosette symptoms and no stunting (1-20% foliage affected), 3 = plants with rosette symptoms plus stunting (21-50 % foliage affected), 4 = plants with severe rosette leaf symptoms and stunted (51- 70% foliage affected), and 5 = plants with severe rosette leaf symptoms and stunting or dead plants (71-100% foliage affected). The last disease score was used to calculate a disease severity index (DSI) for each plot as described by Olorunju *et al.* (1992), as follows: (1A + 2B + 3C + 4D + 5E)/total number of plants assessed per plot, where A, B, C, D, and E are the number of plants with ratings of 1, 2, 3, 4, and 5, respectively. The DSI has a range from 1.0 for no diseased plant rated to 5.0 for uniform mortality of rated plants. Each entry was assessed for disease incidence 60 days after planting. Disease incidence (DI) was determined by recording the percentage of plants with rosette symptoms. No yield data were recorded in all the trials.

Table 1. *Number of lines screened and selected in field screening trials during 1996 and 1997 growing seasons*

Source	Number screened	Resistant 1996	Resistant 1997
Germplasm accessions	2301	65	65
Late maturing lines	64	64	56
Medium-maturity	66	56	56
Early maturity	122	22	22
Total	2553	207	199

1997 season

Entries that showed low disease incidence (< 10%) and produced pods in the 1996 season were grown in a randomised complete block design with two replications at Samaru using the technique described above.

Detection of Rosette disease components

Previous studies (Bock *et al.*, 1990; Blok *et al.*, 1995) showed good correlation between rosette symptoms and the presence of GRV and its sat RNA in either rosette susceptible or resistant. Therefore, only GRAV was detected in the 1997 season. Leaf samples from plants without symptoms as well as from branches of partially infected plants of all resistant lines were tested for the presence of GRAV using a triple anti-body sandwich of enzyme-linked immunosorbent assay (TAS-ELISA) as described by Rajeshwari *et al.* (1987).

Statistical analysis

In 1996, disease incidence and severity values from unreplicated plots were used to classify genotypes into resistant and susceptible groups. In 1997, analysis of variance was performed on disease severity index data using GENSTAT software package (Lane & Payne, 1996).

Results

In all screening trials rosette disease development was uniform. Disease incidence in the susceptible genotypes progressed more rapidly in 1996 than in 1997 as most of the susceptible lines in 1997 had been eliminated in the 1996 screening. By 3 wk after exposure to inoculum in 1996, more than 95% of susceptible plants were showing symptoms. Disease spread on the infector rows was very good indicative of the even distribution of inoculum and effective screening. The susceptible line had 100% infection within 20 days of inoculum introduction whereas the resistant line RMP 12 showed mild symptoms on few young leaves towards the end of the season.

Plants showing severe symptoms were stunted and bushy in appearance due to reduced internode length. Leaves of the infected plants were reduced in size and the plants did not produce pods. In the 1996 screening, most of the accessions were susceptible with a disease incidence of more than 90%. Most of the accessions did not produce harvestable pods. The incidence of chlorotic rosette was 99% at Samaru and 98% at Bagauda. The remaining 1.5% and 2.5% of plants developed green rosette with mild chlorosis. DSI of germplasm lines ranged from 1 to 5 in 1996 and 1 to 3 in 1997. Fifty three percent (or 1224 lines) of the germplasm lines had a DSI of 5 and disease incidence of 100%. Forty four percent (or 1009 lines) had a DSI of 4 with a diseases incidence of greater than 80%. The remaining 3% (or 68) lines had a DSI ranging from 1 to 2 with less than 10% disease incidence. Lines were considered resistant when no susceptible plants were found within the complete entry (0% incidence) and highly susceptible when no resistant plants were present (100% incidence). The majority of the resistant lines were from Nigeria, Burkina Faso, Cote d'Ivoire, Equatorial Guinea and Democratic Republic of Congo and the rest were from Malawi, Mozambique and Zimbabwe (Table 2). Among these lines, accessions ICG 7466, ICG 7694, ICG 7759 and ICG 11968 showed mild symptoms of the disease which appeared late in the season on the distal part of the branches. Similar symptoms were also observed in a few plants of RMP 12 (used as a resistant check). Among the resistant lines, ICG 7490, ICG 7638, ICG 7727, ICG 7623, ICG 7636, ICG 7625, and ICG 7637 were early maturing (100-110 days). All the resistant plants formed normal pegs and produced well-filled pods. The rest of the germplasm lines were either killed or did not produce any harvestable pods due to the disease.

The reaction of breeding lines to rosette disease is presented in Tables 3, 4 and 5. Disease severity scores ranged from 1 to 2 in both years in Samaru and Bagauda. Of the 64 late-maturing lines only ten showed a few plants with mild late symptoms. For the medium maturity group, 37 lines showed no visible symptoms while 17 showed mild symptoms late in the season. Thirty lines in the early maturity group did not show symptoms at both locations while the rest were highly susceptible.

All the resistant germplasm and breeding lines tested positive to the GRAV antigen, but the concentration of GRAV was variable (data not shown).

Discussion

The main goal of this research was to identify additional sources of resistance to rosette. The results look very promising since both germplasm and breeding lines were identified that can be used in integrated management of rosette. Some of the germplasm lines are already in good agronomic background and can be used directly for direct production. Out of the 65 resistant germplasm lines, 42 were also found resistant in Malawi (Subrahmanyam *et al.*, 1998). This indicated stable resistance and should be useful sources of resistance for breeding programmes in Africa.

A total of 134 breeding lines in various maturity groups showed levels of resistance comparable to RMP 12. Those in the early- and medium-maturity groups require a much shorter growing period than the resistant check. These lines offer an opportunity to eliminate 30-100% yield loss in the semi-arid zone

Table 2. *Reaction of groundnut germplasm accessions resistant to rosette disease in field screening trials at Samaru during 1996 and 1997 growing seasons*

(ICG) No.[a]	Alternate name	Source	DI (DSI)[b] 1996	1997
643	69-101	Senegal	0 (1.0)	0 (1.0)
6322	RMP 12	Burkina Faso	0 (1.0)	0 (1.0)
6323	RMP 90	Burkina Faso	0 (1.0)	0 (1.0)
6325	48-37	Cote d'Ivoire	0 (1.0)	0 (1.0)
6326	55-455	Cote d'Ivoire	0 (1.0)	0 (1.0)
6333	RMP 89	Burkina Faso	0 (1.0)	0 (1.0)
6388	RG 188	Cote d'Ivoire	0 (1.0)	0 (1.0)
6395	RG 200	Senegal	0 (1.0)	0 (1.0)
6428	RG 194	Cote d'Ivoire	0 (1.0)	0 (1.1)
6466	RG 199	Senegal	0 (1.0)	0 (1.1)
6482	RG 190	Cote d'Ivoire	0 (1.0)	0 (1.0)
6745	M19	Malawi	0 (1.0)	0 (1.0)
6747	Runner	Zimbabwe	0 (1.0)	0 (1.0)
7236	RMP 16	Burkina Faso	0 (1.0)	0 (1.0)
7348	M white	Malawi	0 (1.0)	0 (1.0)
7350	M 318-74 K	Nigeria	0 (1.0)	0 (1.0)
7445	M 65-75 M	Nigeria	0 (1.0)	0 (1.0)
7446	M 6-76 M	Nigeria	0 (1.0)	0 (1.0)
7448	M 718-76	Nigeria	0 (1.0)	0 (1.1)
7451	M 25-68 K	Nigeria	0 (1.0)	0 (1.0)
7452	M 170-72 K	Nigeria	5 (1.5)	0 (1.0)
7454	M 399-72 K	Nigeria	0 (1.0)	0 (1.0)
7456	M 290-73 K	Nigeria	3 (2.0)	0 (1.0)
7458	M 104-74 K	Nigeria	5 (1.3)	7 (1.4)
7460	M 287-74 K	Nigeria	0 (1.0)	0 (1.0)
7461	M 705-74	Nigeria	0 (1.0)	0 (1.0)
7466	M 649 K	Nigeria	6 (2.0)	4 (1.4)
7467	M 699-75 K	Nigeria	0 (1.0)	0 (1.0)
7468	M 688-75 K	Nigeria	0 (1.0)	0 (1.0)
7484	RMP 91	Burkina Faso	0 (1.0)	0 (1.0)
7490	M 57-72 K	Nigeria	0 (1.0)	0 (1.0)
7623	M 253-72 K	Nigeria	2 (1.3)	5 (1.3)
7624	M 285-74 K	Nigeria	0 (1.0)	0 (1.0)
7625	M 1069-74	Nigeria	0 (1.0)	0 (1.0)
7637	M 107-74 K	Nigeria	0 (1.0)	0 (1.0)
7636	M 103-74 K	Nigeria	0 (1.0)	0 (1.0)
7641	RMP 93	Burkina Faso	0 (1.0)	0 (1.0)
7644	M 103-74 S	Nigeria	0 (1.0)	0 (1.0)
7648	M 1052-76	Nigeria	0 (1.0)	0 (1.0)
7652	M 221-76	Nigeria	0 (1.0)	0 (1.0)
7693	M 699-72 S	Nigeria	0 (1.0)	0 (1.0)
7694	M 515-76	Nigeria	5 (2.0)	3 (1.4)
7727	M 86-73 K	Nigeria	0 (1.0)	0 (1.0)
7730	M 751-76	Nigeria	0 (1.0)	3 (1.2)
7749	M 308-72	Nigeria	0 (1.0)	0 (1.0)
7751	RMP 49/6	Malawi	0 (1.0)	0 (1.0)
7752	RMP 49/2/1	Malawi	0 (1.0)	0 (1.0)
7753	RMP 49/3	Malawi	0 (1.0)	0 (1.0)

cont.....

Table 2 (continued)

(ICG) No.[a]	Alternate name	Source	DI (DSI)[b] 1996	1997
7754	RMP 49/4/1	Malawi	0 (1.0)	0 (1.0)
7755	RMP 49/4/2	Malawi	0 (1.0)	0 (1.0)
7756	RMP 49/5	Malawi	0 (1.0)	0 (1.0)
7758	RMP 30/1	Malawi	0 (1.0)	0 (1.0)
7759	BS1	Malawi	3 (2.0)	2 (1.3)
7760	B 735	Malawi	0 (1.0)	0 (1.0)
7995	48-37	Cote d'Ivoire	0 (1.0)	0 (1.0)
8725	48-37	Cote d'Ivoire	0 (1.0)	0 (1.0)
8730	56-383	Cote d'Ivoire	0 (1.0)	0 (1.0)
9300	58-436	E. Guinea	0 (1.0)	0 (1.0)
9549	RMP 134	Mozambique	0 (1.0)	0 (1.0)
9558	RMP167	Mozambique	0 (1.0)	0 (1.0)
10542	PI 27961	Burkina Faso	0 (1.0)	0 (1.0)
11968	RS 105	Mali	2 (1.1)	1 (1.2)
12938	RG 1	Malawi	0 (1.0)	0 (1.0)
Controls				
Resistant				
RMP 12		Cote d'Ivoire	1 (1.2)	0 (1.0)
Susceptible				
55-437		Hungary	100 (5.0)	100 (5.0)
df				68
SED[c]				0.02

[a]ICRISAT groundnut accession number
[b]DI = Disease incidence, DSI = Disease severity index
[c]SED is for the disease severity index

Table 3. *Reaction of late- maturing breeding lines to rosette disease in field screening trials during the 1996 and 1997 growing season at Bagauda and Samaru*

Line[a]	Pedigree	DI (DSI)[b] Bagauda 96	Samaru 96	Samaru 97
ICGV-SM 88711	Mani Pintar / RMP91	3 (2.0)	0 (1.0)	0 (1.0)
ICGV-SM 88734	Chitemabana / RMP 93x	0 (1.0)	0 (1.0)	0 (1.1)
ICGV-SM 88735	Mawanga/RMP 93	0 (1.0)	0 (1.0)	5 (2.0)
ICGV-SM 88736	Mani Pintar/RMP 91	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 88737	Mani Pintar/RR1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 88761	SP 1 / RMP 91	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 88762	Egret/ RMP 91	8 (2.0)	0 (1.0)	0 (1.0)
ICGV-SM 88763	RG1/ Mani Pintar	1 (1.2)	0 (1.0)	0 (1.0)
ICGV-SM 88764	Mani Pintar/RMP 40	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 88769	Mani Pintar/RMP 91	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89749	SP1/RR1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89750	Mukuru Red/RR1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89751	Mawanga/RMP 93	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89752	Mani Pintar/RMP 91	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89753	RG1/JL 24	2 (2.0)	1 (1.2)	0 (1.0)
ICGV-SM 89755	RG1/JL 24	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89756	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)

cont.....

Table 3. (continued)

Line[a]	Pedigree	DI (DSI)[b]		
		Bagauda 96	Samaru 96	Samaru 97
ICGV-SM 89758	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89759	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89760	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89762	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89763	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89764	RMP 40/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89766	ICGM 48/RG1/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89767	JL 24/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89768	Mani Pintar/RMP 40	7 (2.0)	8 (2.0)	0 (1.0)
ICGV-SM 89786	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89787	RG1/ICGM 48	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89789	RMP 40/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89790	RMP 40/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 89791	Robut 33-1/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 90002	Mawanga/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 90003	Robut 33-1/RG1	1 (1.2)	0 (1.0)	0 (1.0)
ICGV-SM 90701	RMP 40/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 90702	RMP 40/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 90703	RMP 40/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 90704	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 91701	RG1/Mani Pintar	1 (1.2)	0 (1.0)	0 (1.0)
ICGV-SM 91705	RG1/Mani Pintar	1 (1.2)	0 (1.0)	0 (1.3)
ICGV-SM 91706	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.3)
ICGV-SM 91707	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.3)
ICGV-SM 91708	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 91709	RMP 40/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 91710	RG1/Mani Pintar	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 91717	Malimba/RR1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 92507	Malimba/RR1	0 (1.3)	0 (1.0)	0 (1.0)
ICGV-SM 93526	RG1/JL 24	0 (1.2)	0 (1.0)	0 (1.0)
ICGV-SM 93531	ICGV-SM 83030/RG1	3 (2.0)	1 (1.2)	0 (1.0)
ICGV-SM 93532	ICGV-SM 83030/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93534	ICGV-SM 83030/RG1	0 (1.1)	0 (1.0)	0 (1.0)
ICGV-SM 93557	ICGV-SM 885023/RG1	0 (1.1)	0 (1.0)	0 (1.0)
ICGV-SM 93560	ICGMS 197/RMP 40	1 (1.2)	0 (1.0)	0 (1.0)
Controls				
Resistant				
RMP 12		1 (1.0)	1 (1.2)	0 (1.0)
249-85		0 (1.0)	0 (1.0)	0 (1.0)
M554-76		1 (1.0)	1 (1.0)	0 (1.0)
Susceptible				
55-437		100 (5.0)	100 (4.7)	100 (4.9)
df		70	70	70
SED[c]		0.04	0.01	0.03

[a]ICGV-SM = ICRISAT breeding lines selected in Malawi, ICGMS = germplasm lines selected in Malawi
[b]DI=diseases incidence, DSI=Disease severity index
[c]SED is for disease severity index

Table 4. *Reaction of medium-maturing lines to rosette disease in field screening trials during the 1996 and 1997 growing seasons at Bagauda and Samaru*

Line[a]	Pedigree	DI (DSI)[b]		
		Bagauda 96	Samaru 96	Samaru 97
ICGV-IS 96801	ICGV-SM 85035/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96802	ICGV-SM 85035/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96803	RG1/ICGV-SM 85725	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96804	ICGV-SM 85045/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96805	ICGV-SM 85048/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96806	RG1/ICGV-SM 85725	4 (1.4)	8 (1.0)	0 (1.0)
ICGV-IS 96807	RG1/ICGV-SM 85725	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96808	ICGV-SM 85045/RG1	4 (1.2)	7 (1.4)	0 (1.0)
ICGV-IS 96809	ICGMS 522/RG1	4 (1.3)	7 (1.2)	0 (1.0)
ICGV-IS 96810	ICGV-SM 85035/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96811	ICGV-SM 85035/RG1	0 (1.0)	0 (1.1)	0 (1.0)
ICGV-IS 96812	ICGV-SM 85035/RG1	15(2.0)	18 (2.0)	12 (2.0)
ICGV-IS 96813	RG1/Flamingo	1 (1.2)	2 (1.2)	0 (1.0)
ICGV-IS 96814	RG1/ICGM 484	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96815	RG1/ICGV-SM 85725	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96816	RMP 40/ ICGV-SM 85048	2 (1.3)	5 (1.4)	0 (1.0)
ICGV-IS 96817	RG1/ICGMS 484	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96818	RG1/ICGMS 484	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96819	RG1/ICGMS 484	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96820	ICGV-SM 85001/RG1	4 (1.3)	5 (1.3)	2 (1.3)
ICGV-IS 96821	ICGV-SM 85035/RG1	2 (1.4)	3 (1.2)	0 (1.0)
ICGV-IS 96822	ICGV-SM 85035/RG1	4 (1.2)	8 (1.2)	0 (1.0)
ICGV-IS 96824	ICGV-SM 85035/RG1	1 (1.2)	2 (1.2)	0 (1.0)
ICGV-IS 96825	ICGV-SM 85048/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96826	ICGMS 522/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96827	ICGMS 522/RG1	1 (1.0)	1 (1.2)	0 (1.0)
ICGV-IS 96828	ICGMS 522/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96829	ICGMS 522/RG1	2 (1.3)	0 (1.0)	4 (1.2)
ICGV-IS 96830	ICGMS 56/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96831	ICGMS 56/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96832	ICGMS 522/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96833	ICGMS 5/ICGMS 5	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96834	ICGMS 42/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96835	ICGV-SM 886021/ICGX	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96836	ICGMS 42/ICGV-SM 88711	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96837	ICGMS 42/ICGV-SM 88734	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96838	ICGMS 42/ICGV-SM 88734	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96839	ICGV-SM 85718/ICGV-SM 88709	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96840	ICGV-SM 86021/ICGX-SM 82040	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96842	ICGMS 42/ICGV-SM 88711	2 (1.3)	3 (1.0)	0 (1.0)
ICGV-IS 96843	ICGV-SM 83708/ICGV-SM 88711	4 (1.4)	8 (1.4)	0 (1.0)
ICGV-IS 96844	ICGV-SM 85718/ICGV-SM 88709	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96845	ICGV-SM 86021/ICGX-SM 82040	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96846	ICGV 87157/ICGV-SM 82051	2 (1.2)	4 (1.2)	0 (1.0)
ICGV-IS 96847	ICGV-SM 86021/ICGX-SM 82040	1 (1.3)	1 (1.2)	0 (1.0)
ICGV-IS 96848	ICGV 86015/ICGV-SM 82051	2 (1.2)	5 (1.4)	0 (1.0)
ICGV-IS 96849	ICGV-SM 86021/ICGX-SM 82040	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96850	ICGV-SM 86021/ICGX-SM 82051	1 (1.4)	1 (1.0)	0 (1.0)

cont.....

Table 4. (continued)

Line[a]	Pedigree	DI (DSI)[b] Bagauda 96	Samaru 96	Samaru 97
ICGV-IS 96852	ICGV-SM 86021/ICGX-SM 82040	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96854	ICGV 87157/ICGV-SM 82051	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96855	ICGSM 56/ICGV-SM 88711	1 (1.2)	1 (1.2)	0 (1.0)
ICGV-IS 96856	ICGSM 56/RG1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96857	ICGSM 56/KH 241D	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96898	ICGSM 63/ICGX-SM 82051	0 (1.0)	0 (1.1)	0 (1.0)
ICGV-IS 96899	ICGSM 63/ICGX-SM 82051	0 (1.0)	0 (1.3)	0 (1.0)
Controls				
Resistant				
RMP 12		0 (1.0)	1 (1.0)	0 (1.0)
Susceptible				
55-437		100 (4.6)	100 (4.9)	100 (5.0)
df		66	66	66
SED[c]		0.02	0.01	0.01

[a]ICGV-IS = ICRISAT breeding lines selected in West Africa
[b]DI=diseases incidence, DSI=Disease severity index
[c]SED is for disease severity index

of West Africa where short-duration cultivars are required.

The results of this study indicated a higher percentage of resistance in both medium and late maturing lines compared to the early-maturing lines. This trend has been observed in many other studies involving rosette resistance in the three maturity groups (Subrahmanyam *et al.*, 1998). The availability of an efficient screening technique reduces the risk of having escapees in the selection.

The present study showed that resistance to disease symptoms was not absolute since small portions of plants or a few branches of plants in resistant lines had rosette symptoms. All the genotypes resistant GRV were susceptible to GRAV indicating lack of resistance to this component of the rosette complex. The results indicated variability of the virus complex and probably the behaviour of transmission efficiency of *A. craccivora*. Thus resistance to GRV could be overcome under high inoculum pressure or adverse environmental conditions (Naidu *et al.*, 1999). These results along with earlier reports (Bock *et al.*, 1990; Olorunju *et al.*, 1991) suggest that distinct mechanisms of resistance might operate against the three agents (GRV and its satellite RNA, and GRAV) in the resistant material. An understanding of these mechanisms would enable the development of better strategies for incorporating resistance to all agents of rosette disease.

By examining the pedigree of the selected germplasm and breeding lines, it was revealed that the majority owed their source of resistance from RMP 40, RMP91 and RG1, lines that were developed in the 1960s. The only groundnut land race (runner type) from Equatorial Guinea was also found resistant with no apparent symptoms. This indicated a narrow genetic base of these lines.

A recent analysis of strategies of breeding for resistance to rosette suggest that all resistant material developed needs to be critically evaluated for performance against a range of variants of groundnut rosette disease agents in different environments (Naidu *et al.*, 1999). Immunity to GRAV has been identified in several wild *Arachis* species (Subrahmanyam *et al.*, 1998). This provides an opportunity to transfer immunity to GRAV into cultivated groundnut through conventional breeding and/or biotechnological approaches. Resistance to GRAV will reduce virus inoculum build-up considerably. Rosette resistant lines identified in this study will contribute to such an incorporation programme.

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Table 5. *Reaction of early-maturing breeding lines to rosette disease in field screening trials during 1996 and 1997 growing seasons at Bagauda and Samaru*

Line[a]	Pedigree	DI (DSI)[b]		
		Bagauda 96	Samaru 96	Samaru 97
ICGV-IS 96859	ICGM 197/KH 241D	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96868	ICGM 284/KH 241D	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96870	ICGM 284/KH 241D	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-IS 96871	ICGM 284/KH 241D	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93525	RG 1/JL 24	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93528	ICGV-SM 83030/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93530	ICGV-SM 85027/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93533	ICGV-SM 83030/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93535	ICGM 522/RG 1	0 (1.0)	6 (1.4)	3 (1.2)
ICGV-SM 93537	ICGV-SM 83030/RG 1	2 (1.3)	3 (1.2)	1 (1.1)
ICGV-SM 93561	ICGM 197/RMP 40	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93581	ICGV-SM 85018/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93585	ICGM 522/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93586	ICGM 522/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICGV-SM 93587	ICGM 522/RG 1	0 (1.0)	0 (1.0)	0 (1.0)
ICIAR 9AR	KH 241D/ICGV 86055	0 (1.0)	0 (1.0)	0 (1.0)
ICIAR 18AR	KH 241D/ICGV 87922	0 (1.0)	0 (1.0)	0 (1.0)
ICIAR 6 AT	KH 241D/55-437	0 (1.0)	0 (1.0)	0 (1.0)
ICIAR 9AT	KH 241D/ICGV 86055	0 (1.0)	0 (1.0)	0 (1.0)
ICIAR 12AT	KH 241D/ICGV 86061	0 (1.0)	3 (1.2)	0 (1.0)
ICIAR 18AT	KH 241D/ICGV 87922	0 (1.0)	0 (1.0)	0 (1.0)
ICIAR 19BT	KH 241D/ICGV 87922	0 (1.0)	0 (1.0)	0 (1.0)
Controls				
Resistant				
RMP 12		0 (1.0)	0 (1.0)	0 (1.0)
Susceptible				
55-437		100 (5.0)	100 (5.0)	100 (5.0)
df		122	24	24
SED[c]		0.01	0.03	0.01

[a]ICIAR = lines jointly bred by ICRISAT and IAR in Nigeria
[b]DI=diseases incidence, DSI=Disease severity index
[c]SED is for disease severity index

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