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Sources of resistance to groundnut rosette disease in global groundnut germplasm*

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Summary

About 6800 groundnut germplasm accessions originating from South America, Africa, and Asia were evaluated for resistance to rosette disease using an infector row technique between the 1990/91 and 1996/97 growing seasons. Of these, 116 germplasm accessions, including 15 short-duration Spanish types, have shown high levels of resistance to rosette disease. A high percentage of these resistant accessions were from West Africa and a few were from Asia and southern Africa. Only one out of 1400 accessions from South America showed resistance to rosette disease. All disease-resistant accessions were susceptible to groundnut rosette assistor virus. This is the first report to identify sources of resistance to rosette disease in groundnut germplasm from Asia and South America. These additional sources of resistance provide an opportunity to broaden the genetic base of resistance to rosette disease. The origins of rosette resistance in groundnut are discussed.

Key words: Groundnut (peanut), chlorotic and green rosette, host-plant resistance, germplasm

Introduction

Rosette is the most destructive virus disease of groundnut (*Arachis hypogaea* L.) in Africa. The disease is endemic to the African continent, south of the Sahara, and to its off-shore islands (Reddy, 1991). Two forms of rosette, *viz.* chlorotic rosette and green rosette, are recognised on the basis of symptoms (Gibbons, 1977). Chlorotic rosette is the most prevalent type in southern and eastern Africa (Subrahmanyam *et al.*, 1997), while green rosette is the most common in West Africa (Subrahmanyam, Greenberg, Savary & Bosc, 1991). Although rosette disease epidemics are sporadic, yield losses approach 100% whenever the disease occurs in epidemic proportions.

Rosette disease is transmitted by the aphid, *Aphis craccivora* Koch, in a persistent, circulative manner (Okusanya & Watson, 1966). It is caused by a complex of three agents: groundnut rosette virus (GRV), genus *Umbravirus* (Murant, Robinson & Gibbs, 1995) and its

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satellite RNA (sat RNA, Blok, Ziegler, Robinson & Murant, 1994) 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 or transient mild mottle symptoms. The rosette symptoms are largely due to sat RNA (Murant, Rajeshwari, Robinson & Raschke, 1988), and variants of sat RNA are responsible for different forms of the rosette disease (Murant & Kumar, 1990). All three agents must be present together in the host plant for successful transmission of the disease by the aphid vector.

Previous work showed that rosette disease could be managed by insecticidal control of the vector and by cultural practices like manipulating date of sowing and plant density (A'Brook, 1964; Booker, 1963; Davies, 1975, 1976; Farrell, 1976*a*,*b*; Guillemin, 1952; Subrahmanyam & Hildebrand, 1994). However, these practices are seldom adopted by the smallholder farmers in Africa due to lack of resources, labour constraints and costs, sowing sequence of crops and differential crop priorities. Host-plant resistance, therefore, offers the best practical way for rosette disease management.

Pioneering research on the development of groundnut cultivars with resistance to rosette was done by IRHO (Institut de Recherches pour les Huiles et Oleagineux) in West Africa. Sources of resistance to rosette disease were first discovered in 1952, when an epidemic of this disease destroyed a large collection of groundnut germplasm at Bambey, Senegal (Catherinet, Sauger & Durand, 1954). However, a few germplasm lines originating from the frontier region between Burkina Faso and Cote d'Ivoire were able to withstand the epidemic. Resistance identified in those lines is effective against both chlorotic rosette and green rosette, and this resistance is governed by two independent recessive genes (Berchoux, 1960; Nigam & Bock, 1990). These sources formed the basis for rosette resistance breeding programs throughout Africa. However, most of these resistance sources are long-duration (between 120–130 days for maturity, 150–160 days in cooler climates at high altitude) Virginia types and therefore have a narrow genetic base. The Southern African Development Community (SADC)/International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) Groundnut Project based at Chitedze Agricultural Research Station, Lilongwe, Malawi, launched a program to screen the global collection of groundnut germplasm available in the gene bank at ICRISAT-Patancheru, India, to identify additional sources of resistance to groundnut rosette disease. This study identifies for the first time sources of resistance to rosette disease in groundnut germplasm accessions collected from Asia and South America in addition to those from West Africa.

Materials and Methods

Location

All field trials were conducted in the 1990/91, 1991/92, 1992/93, 1993/94, 1994/95, 1995/96, and 1996/97 growing seasons (December to April) at Chitedze Agricultural Research Station located 16 km west of Lilongwe, Malawi in southern Africa at 14°S and 33°45′E with an altitude of 1149 m.

Seed preparation and sowing

Seeds were obtained from the Genetic Resources Division, ICRISAT-Patancheru, India, and treated with a protectant fungicide (thiram at 3 g kg^{-1} $seed^{-1}$) before sowing. Seeds were sown singly at 10 cm (for Spanish and Valencia types) or 15 cm (for Virginia types) spacing along 60 cm raised ridges and received 40 kg P_2O_5 ha^{-1} as basal application of single super phosphate. All trials were conducted under rainfed conditions.

Preliminary screening

Each genotype was evaluated in unreplicated single row field plots of 3 m using the infector row technique (Bock & Nigam, 1988). A chlorotic rosette culture maintained in the greenhouse was used in all screening trials. Infector rows of a rosette susceptible groundnut (cv. Malimba) were arranged throughout the trial, one infector row flanking every two test rows. Potted spreader plants (cv. Malimba) showing severe rosette symptoms and heavily infested with aphids were raised in the glasshouse and transplanted in the infector rows (1 plant per 3 m row) 10 days after sowing. To minimise the chances of escape, each infector row was examined approximately 2 wk later and the plants that were free from rosette symptoms were infested with viruliferous aphids. Each entry was assessed for disease incidence at the pod-filling stage. The total number of plants in each plot and the number of plants showing rosette symptoms with severe stunting were counted and the percentage of disease incidence was computed.

Advanced screening

Those entries which showed low disease incidence (< 20%) in preliminary screening were further evaluated in advanced screening trials in the following growing season in replicated field plots using the technique described above. Each entry was grown in randomised block design with three to four replications. Plots consisted of two 6 m rows of the genotype. Disease incidence in each plot was assessed as described above.

In 1995/96, plants in each plot were evaluated on the basis of the following disease rating system adopted from Olorunju *et al.* (1991) with some modifications: 1 = plants with no visible disease symptoms on foliage and no stunting, 2 = plants with obvious rosette leaf symptoms and stunted to about 50% the size of symptomless plants, and 3 = plants with severe rosette leaf symptoms and stunting greater than 50%. Disease index values were determined by using a rating system similar to that described by Olorunju *et al.* (1991) as follows: (A+2B+3C)/total number of plants assessed per plot, where A, B, and C equal the number of plants with ratings of 1, 2, and 3 respectively. For example, if 35 plants were rated 1, 10 rated 2, and five rated 3, then the disease index = $35 \times 1 + 10 \times 2 + 5 \times 3 = 70 \div 50 = 1.4$.

Detection of GRAV

Only advanced rosette-resistant germplasm accessions were tested (50–60 days after aphid inoculations) for the presence of GRAV during the 1995/96 and 1996/97 growing seasons. Leaf samples were taken from 12 individual plants at random from two replications for each accession and tested by the triple antibody sandwich form of enzyme-linked immunosorbent assay (TAS-ELISA) as described by Rajeshwari, Murant & Massalski (1987). Equal quantities of tissue were taken for extraction in phosphate buffered saline (PBS) containing 0.01 M sodium diethyl dithiocarbamate (1:10 dilution, w/v) and 100 μl of the extract was added to each well of a microtitre plate (Greiner GMBH). GRAV IgG (1 μg ml^{-1}) was used to coat the plates and a monoclonal antibody to potato leafroll virus (PLRV), SCR 6, was used as the second antibody. Absorbance readings at A_{405} were taken in a Titertek Multiscan photometer (Flow Laboratories) after 4 h at room temperature followed by overnight incubation at 5°C. Each sample was assayed twice and readings of the extracts with more than twice the value of healthy plant extracts were considered as positive for GRAV.

Statistical analysis

Analysis of variance (ANOVA) for data on rosette disease incidence (%) from advanced screening trials in 1993/94, 1994/95, 1995/96, and 1996/97 and for disease index measured in 1995/96 were performed using the GENSTAT software package. Angular transformation,

Table 1. *Reaction of some Virginia type (variety* hypogaea*) groundnut genotypes to rosette disease in field screening trials during the 1993/94, 1994/95, 1995/96 and 1996/97 growing seasons at Chitedze, Malawi*

ICG No.[a]	Other identities	Country of origin	Source[b]	Seed colour	Rosette disease incidence (%)					Disease index (1995/96)
					1993/94	1994/95	1995/96	1996/97	Mean	
589	28-206 RR, EC 99219	Senegal	BL	Tan	6	8	8	3	6.3	1.3
3436	PI 246388, EC 99671	South Africa	LR	Tan	9	0	2	0	2.8	1.3
4540	69-101	Senegal	BL	Tan	6	8	2	5	5.3	1.3
6322	RMP 12	Burkina Faso	BL	Tan/White[c]	4	0	7	5	4.0	1.4
6323	RMP 91	Burkina Faso	BL	Tan	3	0	0	1	1.0	1.2
6325	48-37	Cote d'Ivoire	BL	Tan	4	4	2	2	3.0	1.2
6326	55-455	Cote d'Ivoire	BL	Tan	4	—	0	0	1.3	1.0
6333	RMP 89	Burkina Faso	BL	Tan	11	1	2	2	4.0	1.3
6395	RG 200, 56-408	Senegal	BL	Tan	3	—	7	1	3.7	1.3
6424	RG 204, 56-129	Senegal	BL	Tan	2	3	12	1	4.5	1.3
6428	RG 192, 48-35	Cote d'Ivoire	BL	Tan	2	5	0	0	1.8	1.2
6466	RG 199, 56-210	Senegal	BL	Tan	3	0	2	0	1.3	1.3
6482	RG 190, 52-14	Cote d'Ivoire	BL	Tan	0	—	2	0	0.7	1.1
7236	RMP 16, 311/75	Burkina Faso	BL	Tan/White	10	3	0	8	5.3	1.3
7237	RMP 40, 312/75	Burkina Faso	BL	Tan/White	5	0	4	2	2.8	1.2
7303	RMP 11, 309/75	Burkina Faso	BL	Tan/White	—	—	0	5	2.5	1.1
7346	M 53-76 (1) M	Nigeria	BL	Tan	5	0	0	0	1.3	1.2
7350	M 318-74 K	Nigeria	BL	Tan	4	0	7	2	3.3	1.3
7416	RMP 12, 310/76	Burkina Faso	BL	Rose	10	0	2	2	3.5	1.3
7436	M 121-74 S	Nigeria	BL	Red	3	2	8	7	5.0	1.4
7437	M 127-74 S	Nigeria	BL	Tan	10	0	—	15	8.3	—
7445	M 65-75 M	Nigeria	BL	Tan	10	—	6	5	7.0	1.4
7446	M 6-76 M	Nigeria	BL	Tan	2	2	—	2	2.0	—
7448	M 718-76 (1) M	Nigeria	BL	Tan	5	0	3	0	2.0	1.3
7449	M 843-76 (1) M	Nigeria	BL	Tan	5	0	0	—	1.7	1.2
7450	69-101 K	Senegal	BL	Tan	10	0	5	1	4.0	1.4
7452	M 170-72 K	Nigeria	BL	Tan	5	16	10	12	10.8	1.2
7454	M 399-72 K	Nigeria	BL	Tan	9	0	4	2	3.8	1.5

7455	M 32-73 K	Nigeria	BL	Tan	4	—	7	8	6.3	1.5
7456	M 290-73 K	Nigeria	BL	Tan	7	0	12	1	5.0	1.5
7458	M 104-74 K	Nigeria	BL	Tan	0	0	3	0	0.8	1.2
7459	M 237-74 K	Nigeria	BL	Tan	8	0	—	—	4.0	—
7461	M 705-74 K	Nigeria	BL	Tan	5	0	0	10	3.8	1.3
7463	M 100-74 K	Nigeria	LR	Tan	2	0	2	5	2.3	1.2
7469	M 925-74 K	Nigeria	BL	Tan	2	0	4	0	1.5	1.2
7483	RMP 12	Burkina Faso	BL	Tan/White	10	0	0	1	2.8	1.3
7492	M 569-74 K	Nigeria	BL	Rose	0	1	0	0	0.3	1.1
7625	M 1069-74 K	Nigeria	BL	Tan	11	0	2	1	3.5	1.1
7636	M 103-74 K	Nigeria	BL	Tan	2	4	0	0	1.5	1.1
7637	M 107-74 K	Nigeria	BL	Tan	2	0	0	2	1.0	1.1
7638	M 249-74 K	Nigeria	BL	Tan	7	2	0	1	2.5	1.3
7641	RMP 93, 313/75	Burkina Faso	BL	Tan	4	0	5	0	2.3	1.5
7645	2630-76 S	Nigeria	BL	Red	8	2	0	0	2.5	1.2
7648	M 1052-76 M	Nigeria	BL	Tan	10	5	4	0	4.8	1.3
7649	M 64-72 K	Nigeria	BL	Tan	5	7	3	3	4.5	1.6
7650	M 108-74 K	Nigeria	BL	Tan	2	0	3	4	2.3	1.5
7651	M 599-74 K	Nigeria	BL	Tan	9	8	6	0	5.8	1.5
7652	M 221-76 (1) K	Nigeria	BL	Tan	8	3	0	0	2.8	1.2
7675	M 460-74 K	Nigeria	BL	Purplish red	11	—	2	2	5.0	1.4
7677	M 884-74 K	Nigeria	BL	Rose	7	2	—	6	5.0	—
7693	M 669-74 S	Nigeria	BL	Tan	10	0	2	2	3.5	1.4
7726	M 79-76 (1) M	Nigeria	BL	Tan	11	8	0	4	5.8	1.3
7728	M 63-74 K	Nigeria	BL	Tan	1	0	2	2	1.3	1.4
7730	M 751-76 (1) M	Nigeria	BL	Tan	10	0	0	2	3.0	1.2
7736	M 27-73 K	Nigeria	BL	Tan	7	3	0	1	2.8	1.2
7739	M 896-76 (1)	Nigeria	BL	Tan	3	0	0	1	1.0	1.4
7743	M 25-68 (1)	Nigeria	BL	Tan	14	4	0	0	4.5	1.2
7745	661-74	Nigeria	BL	Tan	4	4	0	0	2.0	1.3
7749	M 380-72	Nigeria	BL	Tan	8	0	3	1	3.0	1.5
7752	RMP 49/2/1	Malawi	BL	Tan/White	7	0	2	0	2.3	1.4
7753	RMP 49/3	Malawi	BL	Tan/White	3	0	2	0	1.3	1.4

Table 1 (continued). *Reaction of some Virginia type (variety* hypogaea*) groundnut genotypes to rosette disease in field screening trials during the 1993/94, 1994/95, 1995/96 and 1996/97 growing seasons at Chitedze, Malawi*

ICG No.[a]	Other identities	Country of origin	Source[b]	Seed colour	Rosette disease incidence (%)					Disease index (1995/96)
					1993/94	1994/95	1995/96	1996/97	Mean	
7754	RMP 49/4/1	Malawi	BL	Red	9	0	0	1	2.5	1.3
7755	RMP 49/4/2	Malawi	BL	Red/White[c]	12	0	0	0	3.0	1.1
7756	RMP 49/5	Malawi	BL	Red	12	0	7	1	5.0	1.5
7758	RMP 30/1	Malawi	BL	Tan	13	4	6	3	6.5	1.4
7759	BS 1	Malawi	BL	Tan	12	4	0	0	4.0	1.3
7760	B 735	Malawi	BL	Tan	0	0	2	4	1.5	1.2
7995	48-37, PI 268960	Cote d'Ivoire	BL	Tan	7	0	4	0	2.8	1.2
8493	RG 170, Gambia 69	Gambia	LR	Tan	4	0	0	1	1.3	1.1
8494	RG 174, Volta 1172	Burkina Faso	LR	Tan	8	0	0	0	2.0	1.2
8728	56-204	Cote d'Ivoire	BL	Tan	3	0	—	—	1.5	—
8729	56-381	Cote d'Ivoire	BL	Tan	11	0	5	1	4.3	1.5
8730	56-383	Cote d'Ivoire	BL	Tan	8	—	16	1	8.3	1.5
8896	RC 044	Gambia	LR	Tan	7	0	10	2	4.8	1.4
9300	58-436	Eq. Guinea	LR	Tan	8	0	4	1	3.3	1.2
9475	79-73	Senegal	—	Tan/White	4	1	1	7	3.3	1.3
9549	RPM 134	Mozambique	LR	Tan	6	20	2	4	8.0	1.2
9558	RPM 167	Mozambique	LR	Tan	11	0	0	0	2.8	1.3
9723	VRR 731	India	LR	Tan	—	—	15	9	12.0	1.7
10183	52-13	Cote d'Ivoire	—	Tan	0	0	—	—	0.0	—
10275	75-105, No.1040	Burkina Faso	BL	Tan	5	0	0	0	1.3	1.5
10345	K 27-23	Nigeria	BL	Tan	4	0	4	2	2.5	1.4
10347	Lok Wow, PI 445925	China	LR	Tan	—	—	7	4	5.5	1.5
10541	No. 1036, PI 279617	Burkina Faso	—	Tan	10	2	0	0	3.0	1.3
10542	No. 1037, PI 279618	Burkina Faso	—	Tan	5	0	4	1	2.5	1.4
10543	No. 1040, PI 279619	Burkina Faso	—	Tan	0	0	6	0	1.5	1.4
11044	PI 162525	Argentina	LR	Tan	0	0	5	0	1.3	1.1

11116	48-34, PI 268958	Cote d'Ivoire	BL	Tan	14	0	8	1	5.8	1.6
11649	Lianzhan	China	LR	Tan	—	—	7	2	4.5	1.5
11735	RV 055	India	LR	Tan	—	—	7	7	7.0	1.5
11767	RV 093	India	LR	Tan	—	—	4	4	4.0	1.2
11788	RV 115	India	LR	Tan	—	—	3	2	2.5	1.2
11968	RS 105	Mali	LR	Tan/White	2	3	2	0	1.8	1.2
11971	RS 107-1	Mali	LR	Tan	7	3	0	14	6.0	1.5
11972	RS 107-2	Mali	LR	Tan	4	0	0	1	1.3	1.4
12622	RAP 154	India	—	Tan	—	—	2	0	1.0	1.3
12678	RV 14	India	—	Tan	—	—	9	3	6.0	1.5
12680	RV 15	India	—	Tan	—	—	11	5	8.0	1.4
12876	RT 12	Myanmar	—	Tan	—	—	13	6	9.5	1.5
12938	RG 1	Malawi	—	Tan	10	0	0	0	2.5	1.3
13063	GSS 181	India	—	Tan	—	—	7	1	4.0	1.2
Controls										
Resistant										
RG 1		Malawi	BL	Tan	4	0	0	5	2.3	1.2
RMP 40		Burkina Faso	BL	Tan/White	0	0	0	2	0.5	1.1
RMP 93		Burkina Faso	BL	Tan	6	0	0	0	1.5	1.3
RR1/24		Malawi	BL	Tan	1	0	0	0	0.3	1.2
Susceptible										
Chalimbana		Malawi	BL	Tan	100	90	94	79	90.8	2.9
Chitembana		Malawi	BL	Tan	87	91	98	82	89.5	2.9
CG 7		Malawi	BL	Red	100	95	99	87	95.3	2.9
Mani Pintar		Bolivia	BL	Red/White	100	98	94	97	97.3	2.8
df					192.0	178.0	204.0	240.0		204.0
SED					1.8	2.0	1.2	5.8		0.01
CV (%)					13.8	43.1	21.1	86.7		3.7

[a]ICRISAT groundnut accession number.
[b]BL = breeding line, LR = landrace, — = unknown.
[c]Variegated seed colour.

Table 2. *Reaction of some Spanish type (variety* vulgaris*) groundnut genotypes to rosette disease in field screening trials during the 1993/94, 1994/95, 1995/96 and 1996/97 growing seasons at Chitedze, Malawi*

ICG No.[a]	Other identities	Country of origin	Source[b]	Seed colour	Rosette disease incidence (%)					Disease index (1995/96)
					1993/94	1994/95	1995/96	1996/97	Mean	
6327	75-21	Burkina Faso	BL	Red	16	17	7	5	11.3	1.5
6337	69-102	Senegal	BL	Red	4	9	5	8	6.5	1.7
7457	M 19-74 K	Nigeria	BL	Tan	0	1	0	1	0.5	1.3
7623	M 253-72 K	Nigeria	BL	Tan	4	4	0	1	2.3	1.5
9188	75-21, KH 149 G	Burkina Faso	BL	Red	4	4	0	2	2.5	1.4
9190	75-23, KH 184-2 B	Burkina Faso	BL	Tan	11	5	0	6	5.5	1.4
9446	75-52, KH 149 F	Burkina Faso	BL	Tan	4	11	10	3	7.0	1.5
9447	75-54, KH 149 C	Burkina Faso	BL	Red	6	5	3	—	4.7	1.5
9450	75-57	Burkina Faso	BL	Red	1	1	0	1	0.8	1.4
9451	75-58, KH 313 B	Burkina Faso	BL	Red	7	10	9	4	7.5	1.8
10651	GH 327 A, PI 385935	Burkina Faso	BL	Tan	5	7	8	6	6.5	1.9
12988	US 22	India	—	Tan	—	—	5	10	7.5	1.5
12989	US 23	India	—	Tan	—	—	0	19	9.5	1.3
12991	US 25	India	—	Tan	—	—	0	6	3.0	1.4
12992	US 26	India	—	Tan	—	—	0	—	0.0	1.2
Controls										
Resistant										
KH 241 D		Burkina Faso	BL	Red	3	10	8	3	6.0	1.8
Susceptible										
Malimba		Malawi	BL	Tan	100	100	93	92	96.3	2.8
JL 24		India	BL	Tan	100	100	96	87	95.8	2.8
df					26	26	34	34		34
SED					1.1	1.3	3.4	4.3		0.08
CV (%)					5.6	6.2	26.7	25.3		6.0

[a]ICRISAT groundnut accession number.
[b]BL = breeding line, LR = landrace, — = unknown.

when applied to disease incidence (%), did not change the conclusions obtained from untransformed data. Accordingly, the results from untransformed data are presented.

Results and Discussion

In all screening trials, rosette disease development in infector rows was uniform and the disease incidence approached 100%. Infected plants were chlorotic and severely stunted. Heavy infestations of viruliferous aphids occurred on these plants and spread to the neighbouring test rows. These conditions ensured uniform disease development throughout the field.

Rosette disease incidence was very high in all susceptible controls in all seasons. Mean disease incidence for Virginia types ranged from 89.5% to 97.3% and for Spanish types from 95.8% to 96.3%. Disease index in 1995/96 for susceptible controls was also high, ranging from 2.8 to 2.9 for Virginia types (Table 1) and 2.8 for Spanish types (Table 2). However, disease incidence was low in all resistant controls in all seasons. Mean disease incidence for Virginia types ranged from 0.3% to 2.3% and for the Spanish control genotype it was 6.0%. Disease index was also low for resistant controls, ranging from 1.1 to 1. 3 for Virginia types (Table 1) and 1.8 for the Spanish control (Table 2). All the test entries showed low disease incidence (< 20%) in advanced screening in all the seasons. The disease indices varied from 1.0 to 1.7 for Virginia types (Table 1) and from 1.2 to 1.9 for Spanish types (Table 2). There was a good correlation between mean disease incidence and disease index for both Virginia ($r = 0.97$) and Spanish ($r = 0.96$) genotypes.

South American germplasm

A total of over 1400 accessions was evaluated in preliminary field trials in 1990/91, 1991/92, and 1992/93. Only one line, ICG 11044 (PI 162525), a long-duration Virginia bunch type landrace from Argentina showed resistance to rosette disease in 1992/93. It was further confirmed in advanced screening trials in 1993/94, 1994/95, 1995/96, and 1996/97 (Table 1).

African germplasm

Over 3400 germplasm accessions were evaluated during the 1991/92 and 1993/94 growing seasons. Eighty-nine long-duration Virginia types were identified as resistant to rosette disease (Table 1). A high percentage (76%) of them originated in West Africa (Nigeria 39.6%, Burkina Faso 13.9%, Cote d'Ivoire 9.9%, Senegal 6.9%, Mali 3.0%, Gambia 2.0%, and Equatorial Guinea 1.0%) and the rest were from southern Africa (Malawi 8.9%, Mozambique 2.0%, and South Africa 1.0%). In addition, 11 short-duration Spanish types were identified in the African germplasm originating from West Africa, especially Burkina Faso (Table 2). The majority of these germplasm lines originate from crosses involving rosette disease-resistant lines identified after the 1952 epidemic in West Africa and are probably the products of breeding efforts involving resistant parents and subsequent selection for high yield in various production systems. It is apparent that many of the resistant sources originating from Malawi, for instance, are either reselections from the original resistant sources (e.g. RMP series) from Burkina Faso or breeding lines (e.g. RG 1) developed through hybridization involving the sources of resistance from West Africa. Only eight of the resistant germplasm lines from Africa are the land races.

Asian germplasm

Out of a total of over 2000 accessions evaluated in preliminary screening trials in the 1994/95 growing season, 15 were found to be rosette disease-resistant genotypes. Of these, 11 are long-duration Virginia types (India 8, China 2, and Myanmar 1) (Table 1) and four are short-duration Spanish types (all from India) (Table 2). The reaction of these genotypes was further confirmed in advanced screening trials in 1995/96 and 1996/97. Several accessions are landraces collected from farmers' fields in India and China. For instance, ICG 9723 was collected in Alni and ICG 10347 from Sholapur of Maharashtra State, India; ICG 11735 in Paralur, ICG 11767 in Uthangarai, and ICG 11788 in Ardhanaripalyam of Tamil Nadu State. ICGs 10347 and 11649 are landraces from China.

Rosette resistance in Spanish types

Identification of rosette resistance in early-maturing Spanish type groundnuts is of great significance to the development of high-yielding short-duration rosette-resistant cultivars. Some progress has already been made in this direction by the SADC/ICRISAT Groundnut Project (Reddy & Subrahmanyam, 1996). This will accelerate the deployment of high-yielding, rosette-resistant, short-duration cultivars that are urgently required for various production systems in sub-Saharan Africa which are characterised by short and erratic rainy seasons. Two short-duration genotypes, ICGs 12988 and 12991, both originating in India, out yielded several short-duration genotypes under high and low rosette disease situations in 1996/97 and are in trials in farmers' fields in Malawi (van der Merwe & Subrahmanyam, 1997).

Resistance to GRAV

Most of the plants in all resistant germplasm accessions were symptomless. However, a small proportion of plants were partially infected where the symptoms were restricted to one or two branches while the rest of the plant remained symptomless. Leaf tissue from symptomless plants as well as from symptomless and symptom-showing branches of partially infected plants of all resistant germplasm accessions were tested for the presence of GRAV. Previous studies showed good correlation between symptoms and the presence of GRV and its sat RNA in either rosette susceptible or resistant accessions (Bock, Murant & Rajeshwari 1990; Blok *et al.,* 1995) therefore, none of these samples were tested for GRV and its sat RNA. TAS-ELISA results showed that GRAV antigen was present in all plants tested, irrespective of symptoms, suggesting that all rosette disease-resistant accessions are infected by GRAV. However, the level of GRAV accumulation, as indicated by O.D. values in ELISA, varied (data not shown). Detailed studies are required to understand more precisely the relative levels of susceptibility to GRAV and to find out whether quantitative resistance to GRAV multiplication exists among these accessions. The benefits of exploiting such quantitative resistance to GRAV are that plants with low levels of GRAV would be poor sources of virus for acquisition by the aphid vector and in the field the amount of virus spread from infected plants would be considerably lower than that from plants susceptible to virus multiplication as shown with other persistently transmitted luteoviruses (Barker & Harrison, 1986; Gray, Smith & Sorrells, 1994).

The present study also showed that resistance to disease symptoms is not absolute since a small proportion of plants or a few branches of plants in many resistant accessions had rosette disease symptoms. These observations together with earlier reports (Bock *et al.,* 1990; Nutman, Roberts & Williamson, 1964; Olorunju *et al.,* 1991) suggest that distinct mechanisms of resistance might operate against the three agents (GRV and its sat RNA, and GRAV) in the resistant material. The understanding of these mechanisms would enable

the development of better strategies for incorporating resistance to all agents of rosette disease.

Origin and occurrence of rosette disease

Groundnut is the only known natural host of the three agents of rosette disease (GRV, its sat RNA and GRAV). It is likely that both viruses have evolved and survived in host species native to Africa before the introduction of groundnut. After its introduction into Africa sometime in the 16th century, groundnut became an accidental host of rosette disease representing a case of the "new-encounter" phenomenon (Buddenhagen & de Ponti, 1984). It is possible that resistance to rosette came to Africa in some of the original introductions from the South American centre(s) of origin and due to recurrent epidemics in West Africa it was concentrated to a greater degree by natural out crossing and recombination.

A possible explanation for the occurrence of rosette resistance in a landrace collected from a secondary centre of diversity in South America (Argentina) and areas of introduction in Asia (India, Myanmar, and China), where the disease has never existed (Reddy, 1991), is that the resistance was present as a constituent trait in the ancestors of groundnut and was only expressed in the new encounter situation. During the course of evolution, as these genes did not possess any survival value in the absence of the disease, they may have been altered in the majority of the genotypes One of the prerequisites for the loss of traits during 'evolution' is their simple inheritance (Stebbins, 1950) and rosette resistance is governed by two independent major recessive genes (Nigam & Bock, 1990).

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