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Resistance to groundnut rosette disease in wild *Arachis* species

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Summary

One hundred and sixteen accessions representing 28 species in the genus *Arachis* were evaluated for resistance to groundnut rosette disease using an infector row technique during the 1996/97, 1997/98, 1998/99 and 1999/2000 growing seasons at Chitedze, Malawi. Of these, a total of 25 accessions belonging to *Arachis diogoi* (1 accession), *A. hoehnei* (2), *A. kretschmeri* (2), *A. cardenasii* (2), *A. villosa* (1), *A. pintoi* (5), *A. kuhlmannii* (2), *A. appressipila* (3), *A. stenosperma* (5), *A. decora* (1), and *A. triseminata* (1) showed resistance to the groundnut rosette disease. No visible disease symptoms were observed in several accessions belonging to *A. appressipila, A. cardenasii, A. hoehnei, A. kretschmeri, A. villosa, A. pintoi, A. kuhlmannii,* and *A. stenosperma*. Some accessions in *A. appressipila, A. diogoi, A. stenosperma, A. decora, A. triseminata, A. kretschmeri, A. kuhlmannii, and A. pintoi* were resistant to all three components of rosette, *Groundnut rosette assistor virus* (GRAV), Groundnut rosette virus (GRV) and its satellite RNA (sat. RNA). Two accessions in *A. stenosperma* and one accession in *A. kuhlmannii* showed the presence of all three components of the rosette disease. Several wild *Arachis* accessions were resistant to GRAV. All the accessions of *A. batizocoi* (4), *A. benensis* (2), *A. duranensis* (46), *A. dardani* (1), *A. ipaensis* (1), *A. magna* (1), *A. monticola* (3), *A. oteroi* (1), *A. pusilla* (4), and *A. valida* (2) were susceptible to rosette disease. In all these accessions, infected plants were chlorotic and severely stunted. The value of exploitation of the resistance in wild *Arachis* species in rosette resistance breeding programmes is discussed.

Key words: Groundnut (peanut), rosette disease, wild *Arachis* species, germplasm, host-plant resistance

Introduction

Rosette is the most destructive virus disease of groundnut (*Arachis hypogaea* L.) in sub-Saharan Africa. Although rosette disease outbreaks are sporadic and unpredictable, yield losses approach 100% when the disease occurs in epidemic proportions (Subrahmanyam *et al.*, 1991; Subrahmanyam *et al.*, 1997; Naidu *et al.*, 1999*a*). The disease is transmitted by the aphid, *Aphis craccivora* Koch (*Homoptera: Aphididae*) (Okusanya & Watson, 1966). Rosette is caused by a complex of three agents: *Groundnut rosette assistor virus* (GRAV) (Casper *et al.*, 1983; Reddy *et al.*, 1985; Murant, 1989), *Groundnut rosette virus* (GRV) (Murant *et al.*, 1995) and its satellite RNA (sat. RNA)(Blok *et al.*, 1994). The disease symptoms are mainly due to sat. RNA (Murant *et al.*, 1988), and the variants of sat. RNA are responsible for different forms of the disease (Murant & Kumar, 1990). Plants infected by GRAV or GRV alone show no obvious symptoms or only transient mild mottling. Although aphids can transmit GRAV alone, for successful transmission of the disease all the three agents must be present together in the host (Naidu *et al.*, 1999*b*).

Management of groundnut rosette disease by insecticidal control of the vector has been recognized since the mid-1960s. Cultural practices such as early sowing at optimal plant densities are known to reduce the disease incidence. However, smallholder farmers in Africa, for a number of reasons, seldom adopt these practices (see Subrahmanyam & Hildebrand, 1994; Naidu *et al.*, 1999*a*). Therefore, host-plant resistance to the disease is regarded as the most viable and sustainable solution.

Resistance to rosette was first discovered in groundnut land races originating from Burkina Faso and Cote d'Ivoire (Catherinet *et al.*, 1954). Resistance identified in these lines is effective against both chlorotic and green rosette, and is governed by two independent recessive genes (Berchoux, 1960; Nigam & Bock, 1990; Olorunju *et al.*, 1992). These sources formed the basis for rosette resistance breeding programmes throughout Africa and have contributed to the development of several high-

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yielding, rosette-resistant groundnut varieties (Naidu *et al.*, 1999*a*). However, most of the rosette-resistant varieties released to date are late maturing and are not suitable for many production systems in Africa, due to short rainy seasons. In 1990, ICRISAT launched a program at Chitedze, Malawi on screening of global germplasm for resistance against rosette in order to diversify the genetic base of rosette resistance. Over 12 000 groundnut germplasm lines have been screened and several new sources of field resistance to rosette disease have been identified (Subrahmanyam *et al.*, 1998). None of these germplasm lines, however, have shown resistance to GRAV. Resistance in these lines is not absolute as a small proportion of plants or a few branches of plants in most resistant genotypes show rosette symptoms (Subrahmanyam *et al.*, 1998). Such plants act as a potential inoculum source for vector acquisition and disease survival. Although a high percentage of plants of these germplasm lines do not show visible symptoms in the field, the yield reduction in such plants is substantial under rosette epidemic situations (P Subrahmanyam, unpublished) possibly because of their susceptibility to GRAV. This necessitated the search for sources of resistance to GRAV for utilization in breeding programmes. Since GRAV is the main component involved in aphid transmission, identification of GRAV-resistant sources would help restrict the spread of the disease. Preliminary attempts in the past indicated the presence of resistance to GRAV in some wild *Arachis* species (Murant *et al.*, 1991). Identification of combined resistance to GRAV, GRV and its sat. RNA is vital to broaden the genetic base of rosette resistance in groundnut.

This paper reports the results of evaluation of 116 accessions in 28 wild species belonging to the genus *Arachis* against groundnut rosette disease and discusses the opportunities for their utilization in breeding programmes.

Materials and Methods

Field screening of wild Arachis *species*

All field trials were conducted at Chitedze Agricultural Research Station located 16 km west of Lilongwe, Malawi during the 1996/97, 1997/98, 1998/99 and 1999/2000 growing seasons as described by Subrahmanyam *et al.* (1998). Seeds of wild *Arachis* species were obtained from ICRISAT-Patancheru, India. Two Malawian groundnut cultivars, Malimba and CG 7 were used as susceptible controls. Seeds were treated with thiram (3 g kg seed^{-1}) and sown singly at 15 cm spacing along 60 cm raised ridges fertilized with single super phosphate (40 kg P_2O_5 ha^{-1}) as basal application.

Each entry was evaluated in unreplicated single row field plots of 3 m using the infector row technique (Bock & Nigam, 1988; Subrahmanyam *et al.*, 1998). Entries were visually assessed for rosette disease incidence at 120 days after sowing. The total number of plants in each plot and the number of plants showing rosette symptoms with severe stunting were counted and the percentage incidence was calculated.

Those entries which showed low disease incidence (< 10%) in the preliminary screening were further evaluated in replicated field trials as described above. Plots consisted of two 6 m long rows with three replications arranged in a randomised block design.

Detection of GRAV, GRV and sat. RNA

Leaf samples were tested for GRAV by triple antibody sandwich (TAS)-ELISA as described by Rajeshwari *et al.* (1987). Six leaves were collected at random from 12 individual plants for each accession from two replications and equal quantities of tissue was extracted in phosphate buffered saline containing 0.02% Tween-20 and 1% polyvinyl pyrrolidone (mol. wt 40 000). One hundred µl of the extract was loaded into the wells of an ELISA plate (Nunc, Denmark) coated with GRAV IgG at 1µg ml^{-1} concentration. *Chickpea stunt virus* monoclonal antibody (2B2-F3) was used as second antibody and anti-mouse Fc antibody (Sigma, USA) conjugated with alkaline phosphatase (ALP) was used for detection. *p*-nitrophenylphosphate (PNP) was used at 1 mg ml^{-1} in 10% diethanolamine buffer, pH 9.8 and readings were taken at 405 nm after 2 h incubation at room temperature or overnight at 4°C. A_{405nm} values > 2 × those of healthy were considered GRAV positive.

GRV and sat. RNA were detected by RT-PCR as described by Naidu *et al.* (1998*a*). A RNeasy Plant Mini Kit (Qiagen, Germany) was used to extract total RNA from 0.2 g of leaf tissue. Oligonucleotide primers GRV-1 and GRV-2 were used to amplify a 863-bp fragment specific to GRV, and SAT-1 and SAT-2 to amplify a 890 bp fragment specific for the sat. RNA. Ten µl of the amplified products was analysed by 1% agarose gel electrophoresis, DNA was stained with ethidium bromide and visualised on an UV transilluminator.

Statistical analysis

Analysis of variance (ANOVA) for data on rosette disease incidence collected from replicated field trials was performed using the GENSTAT software package. Angular transformations, when applied to disease incidence (%), did not change the conclusions obtained from untransformed data. Accordingly, the results from untransformed data are presented.

Results

Resistance to groundnut rosette disease

The development of rosette disease was uniform in all four seasons and the disease incidence reached 100% in Malimba and CG 7, the two susceptible cultivars used in infector rows. Rosette incidence was very high (about 100%) in susceptible groundnut controls in all seasons (Table 1). Infected plants were chlorotic and severely stunted, and there was no pod formation in these plants.

Out of 116 accessions of wild *Arachis* species evaluated, 79 accessions were scored susceptible (80% to 100% disease incidence), 12 were moderately resistant (25% to 50% disease incidence) and 25 were resistant (< 10% disease incidence) to groundnut rosette disease. Data on moderately resistant and susceptible entries are not presented.

All accessions belonging to *A. diogoi* Hoehne (ICRISAT groundnut accession number, ICG 4983), *A. hoehnei* Krapov. & W C Gregory (ICGs 8190 and 13232), *A. kretschmeri* Krapov. & W C Gregory (ICGs 8191 and 13224), *A. cardenasii* Krapov. & W C Gregory (ICGs 8216 and 11558), *A. villosa* Benth. (ICG 13168), *A. pintoi* Krapov. & W C Gregory (ICGs 13222, 14855, 14856, 14888 and 14907), and

Table 1. *Reaction of some wild* Arachis *species to groundnut rosette disease in field screening trials during the 1997/98, 1998/99 and 1999/2000 growing seasons at Chitedze, Malawi*

ICG		Rosette disease incidence (%)			Presence of		
No.[a]	Species	1998	1999	2000	GRAV[b]	GRV[c]	sat. RNA[d]
4983	*A. diogoi*	ni[e]	0	2	nd[f]	nd	nd
8127	*A. appressipila*	ni	0	ni	nt[g]	nt	nt
8945	*A. appressipila*	0	0	0	nd	nd	nd
14860	*A. appressipila*	0	0	2	nd	nd	nd
8190	*A. hoehnei*	0	6	ni	nt	nt	nt
13232	*A. hoehnei*	ni	0	ni	nt	nt	nt
8191	*A. kretschmeri*	ni	0	2	nd	nd	nd
13224	*A. kretschmeri*	ni	0	ni	nt	nt	nt
8216	*A. cardenasii*	ni	0	ni	nt	nt	nt
11558	*A. cardenasii*	10	0	ni	nt	nt	nt
13168	*A. villosa*	0	0	ni	nt	nt	nt
13171	*A. stenosperma*	0	1	1	nd	nd	nd
13173	*A. stenosperma*	0	3	3	nd	nd	nd
13187	*A. stenosperma*	0	4	3	++[h]	p[i]	p
13210	*A. stenosperma*	0	0	ni	nt	nt	nt
14872	*A. stenosperma*	0	1	1	+++[h]	p	p
13222	*A. pintoi*	ni	0	0	nd	nd	nd
14855	*A. pintoi*	0	0	0	nd	nd	nd
14856	*A. pintoi*	0	3	0	nd	nd	nd
14888	*A. pintoi*	0	0	0	nd	nd	nd
13225	*A. kuhlmannii*	0	0	0	nd	nd	nd
14862	*A. kuhlmannii*	0	0	0	+[h]	p	p
14875	*A. triseminata*	0	3	1	nd	nd	nd
14946	*A. decora*	0	0	1	nd	nd	nd
Controls							
Malimba	*A. hypogaea*	100	100	100	+++	p	p
CG 7	*A. hypogaea*	86	100	100	+++	p	p
df[j]		28	43	46			
Trial mean		18.2	10.9	15.1			
SED[k]		±12.1	±2.4	±1.5			
CV[l] (%)		82	27	12			

[a]ICRISAT groundnut accession number.
[b]GRAV = Groundnut rosette assistor luteovirus
[c]GRV = Groundnut rosette umbravirus
[d]sat. RNA = satellite ribonucleic acid
[e]ni = not included
[f]nd = not detected
[g]nt = not tested
[h]+ = optical density 0.4 to 0.5, ++ = 0.5 to 1.2, and +++ = >1.2
[i]p = present
[j]df = degrees of freedom
[k]SED = standard error of differences
[l]CV = coefficient of variation

A. kuhlmannii Krapov. & W C Gregory (ICGs 13225 and 14862) were resistant to rosette disease (Table 1). Out of six accessions of *A. appressipila* Krapov. & W C Gregory evaluated, only three (ICGs 8127, 8945, and 14860) were resistant (Table 1) and others were either moderately resistant or susceptible to rosette. Of 10 accessions of *A. stenosperma* Krapov. & W C Gregory, five (ICGs 13171, 13173, 13187, 13210, and 14872) were resistant and others were either moderately resistant or susceptible. Of two accessions of *A. decora* Krapov., W C Gregory & Valls tested, only ICG 14946 was resistant and the other was susceptible to the disease. Interestingly, one accession in *A. triseminata* Krapov. & W C Gregory (ICG 14875) was resistant to rosette and the other was susceptible. No rosette disease incidence was observed in several accessions of *A. appressipila* (ICGs 8127 and 8945), *A. cardenasii* (ICG 8216), *A. hoehnei* (ICG 13232), *A. kretschmeri* (ICG 13224), *A. villosa* (ICG 13168), *A. pintoi* (ICGs 13222, 14855, 14888 and 14907), *A. kuhlmannii* (ICGs 13225 and 14862) and *A. stenosperma* (ICG 13210). Plants were vigorous and did not show any visible disease symptoms.

All the accessions in *A. batizocoi* Krapov. & W C Gregory (four accessions), *A. benensis* Krapov., W C Gregory & C E Simpson (2), *A. duranensis* Krapov. & W C Gregory (46), *A. dardani* Krapov. & W C Gregory (1), *A. ipaënsis* Krapov. & W C Gregory (1), *A. magna* Krapov., W C Gregory & C E Simpson (1), *A. monticola* Krapov. & Rigoni (3), *A. oteroi* Krapov. & W C Gregory (1), *A. pusilla* Benth. (4), and *A. valida* Krapov. & W C Gregory (2) were susceptible to rosette disease. In all these accessions, infected plants were chlorotic and severely stunted. Infected plants did not produce any pods.

Resistance to GRAV, GRV and its sat. RNA

The rosette susceptible groundnut varieties, CG 7 and Malimba showed the presence of all three components of the disease, GRAV, GRV and its sat. RNA. Of those tested, some accessions of *A. appressipila* (ICGs 8945 and 14860), *A. diogoi* (ICG 4983), *A. stenosperma* (ICGs 13171 and 13173), *A. decora* (ICG 14946), *A. triseminata* (ICG 14875), *A. kretschmeri* (ICG 8191), *A. kuhlmannii* (ICG 13225), *and A. pintoi* (ICGs 13222, 14855, 14856, 14888, and 14907) did not show the presence of GRAV, GRV and its sat. RNA. Two accessions of *A. stenosperma* (ICGs 13187 and 14872) and one accession of *A. kuhlmannii* (ICG 14862) showed the presence of all three components of the rosette disease (Table 1).

It is interesting to note that several accessions of *A. appressipila* (ICGs 8946 and 8128), *A. rigonii* (ICGs 8186 and 8904), *A. paraguariensis* Chodat & Hassl (ICG 8970), and *A. matiensis* Krapov., W C Gregory & C E Simpson (ICG 11557), which were scored only moderately resistant to the disease, did not show the presence of GRAV. These accessions however, were not tested for the presence of GRV and its sat. RNA. Accessions of *A. batizogaea* Krapov & Av. Fernández (ICG 13208), *A. stenosperma* (ICG 14868) and *A. stenophylla* Krapov. & W C Gregory (ICG 8215) which were scored moderately resistant to the disease, were resistant to GRAV but susceptible to GRV and its sat. RNA. One accession of *A. paraguariensis* (ICG 8973) which was also scored moderately resistant to the disease, did not show the presence of all three components (data not presented).

Discussion

Results of the present study showed that several accessions in different wild species of the genus *Arachis* are free from all three components of groundnut rosette disease. It is likely that these accessions possess resistance to all the components of groundnut rosette. Detailed studies by experimental inoculation of GRAV alone and GRV and sat. RNA are essential to understand precisely the type of resistance offered by these wild species. Nevertheless, several *Arachis* accessions (ICGs 8946, 8128, 8186, 8904, 8970, and 11557) are free from GRAV. Although GRV and sat. RNA are responsible for rosette symptoms, absence of GRAV limits transmission. Thus GRAV-resistant accessions have potential to contribute to disease control. Some accessions (ICGs 13187 and 14862), though positive to GRAV, showed a low level of virus accumulation (Table 1). This suggests the possible existence of quantitative resistance to GRAV multiplication. Plants possessing such resistance would be poor sources for virus acquisition by aphids and could be exploited for GRAV resistance breeding. The fact that *A. batizogaea*, a hybrid derivative between *A. batizocoi* × *A. hypogaea,* has been found to be resistant to GRAV in the present study, indicates that it should be possible to breed groundnut cultivars with combined resistance to all three components of rosette disease. It has been shown under field conditions that the rate of potato leafroll virus spread from partially resistant plants is significantly lower than that from plants susceptible to virus multiplication (Barker & Harrison, 1986). All rosette-resistant groundnut germplasm lines identified prior to this study were found to be susceptible to GRAV (Olorunju *et al.*, 1991; Subrahmanyam *et al.*, 1998). The resistance appeared to be against GRV, which provides indirect resistance to its sat. RNA. This resistance does not amount to immunity and is known to be overcome under high inoculum pressure and environmental conditions that favour disease development (Nutman

et al., 1964; Bock *et al.*, 1990). Although GRAV alone causes no visible symptoms, it does appear to interact with the other two agents in disease development (Naidu *et al.*, 1998*b*; Ansa *et al.*, 1990). It is possible that, in spite of being symptomless, GRAV causes direct yield losses in groundnut. Hence, exploitation of resistance to GRAV in wild *Arachis* species is necessary to reinforce resistance in cultivated groundnut and retard further spread of the disease from infected groundnut crops. In addition, transfer of resistance to any of the three components of rosette disease from the wild species to the cultivated groundnut should broaden the genetic base of resistance.

Wild *Arachis* species have been shown to be generally crossable within sections. Within section *Arachis* however, the cultivated groundnut and its immediate wild progenitor *A. monticola* are the only tetraploid species, the other 25 species being diploid (Krapovickas & Gregory, 1994). *A. monticola* is readily crossable with *A. hypogaea*, but was found to be indistinguishable from *A. hypogaea* according to molecular markers (Kochert *et al.*, 1991; Halward *et al.*, 1991). This essentially isolates the cultivated groundnut reproductively. Several routes have been investigated to introgress genes from wild diploid species into polyploids. These include direct hybridisation, which results in sterile triploid hybrids, followed by chromosome doubling to a hexaploid and elimination of chromosomes either spontaneously or through repeated backcrossing to a tetraploid (Stalker & Moss, 1987), diploid by tetraploid crosses using 2n gametes and somatic doubling of a diploid followed by crossing with a tetraploid (Simpson, 1991). Recently a synthetic amphidiploid, TxAG-6 (Simpson, 1991; Simpson *et al.*, 1993), has been produced in this way, and has been used to introgress root-knot nematode resistance into cultivated groundnut (Burow *et al.*, 1996). Introgressed inter-specific groundnut germplasm lines have been thwarted by low fertility as a result of linkage drag from the wild species. Molecular markers have been used to study the transmission of chromatin from wild into cultivated germplasm (M D Burow, personal communication) and thus have the potential to reduce linkage drag by reducing the contribution of wild germplasm. Genetic transformation also offers opportunities for the utilisation of wild *Arachis* germplasm, irrespective of crossability barriers. It is interesting to note that several of these *Arachis* species which are resistant to groundnut rosette (to all three components) are also resistant to early leaf spot (*Cercospora arachidicola* Hori.) (P Subrahmanyam, unpublished) and should be useful in breeding for multiple resistance in groundnut.

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