Disease Reactions and Yield Performance of Peanut Genotypes Grown Under Groundnut Rosette and Rosette-Free Field Environments

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ABSTRACT

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Experimental field conditions were established to determine the effect of groundnut green rosette (caused by groundnut rosette virus [GRV and its satellite RNA] and groundnut rosette assistor virus [GRAV]) on susceptible and resistant peanut genotypes. Test genotypes were planted in alternate rows with susceptible genotype F 452.4 in which rosette-diseased plants, infested with viruliferous aphids, were transplanted every 1.5 m. When disease incidence was high (90–100%) but severity was moderate (measured on rating scale of 1–5) in susceptible genotypes in 1988, only 9–10% of the resistant plants of RMP 12 and RG 1 showed very mild mottle. In 1989, when incidence in susceptible plants was 100% and disease was very severe, about 87% of the resistant plants showed symptoms but only 2–5% had severe symptoms. In the rosette environment, seed yield was 33 times higher in resistant genotypes than in susceptible ones, and yields in general were comparable in the two groups under rosette disease-free conditions. We consider RMP 12 and RG 1 to be highly resistant to GRV and its satellite but not to GRAV, which causes no leaf symptoms by itself but is essential for aphid transmission of GRV and its satellite.

Production of peanut (*Arachis hypogaea* L.), a major source of oil and protein for humans and feed for livestock in many developing countries (11), is frequently limited by groundnut rosette disease throughout Africa. The disease appears to be restricted to that continent and can cause yield losses of up to 100% (11,16,22,23). Rosetted plants contain two viruses and a viral satellite RNA. The single-stranded RNA of groundnut rosette virus (GRV) can be mechanically transmitted (20), but so far, no particles have been associated with its infection (5,20). Groundnut rosette assistor virus (GRAV), a luteovirus, causes no overt symptoms but is essential for aphid transmission (in a persistent manner) of GRV (9,21) and its satellite RNA (12). It is theorized that GRV-RNA and the satellite RNA are encapsidated in GRAV coat protein when mixed infections occur (5,12,13). A GRV satellite RNA is required for both rosette symptom production (14) and aphid transmission (12). Variants of the satellite RNA are responsible for two forms of the disease, chlorotic rosette and green rosette (13). The most severe disease occurs when peanut plants are infected with all three components (1,14).

A few peanut genotypes resistant to the rosette causal agent(s) are available (7,8,16,19), but their use by farmers has been limited because they are small-seeded, late maturing, or have some other unacceptable agronomic characteristics. Peanut breeding, therefore, must be directed toward incorporation of resistance into high yielding genotypes that will be readily used by farmers. The primary objective of this study was to determine disease reactions of peanut genotypes believed to be susceptible or resistant under field conditions (plants infected with all three causal agents) in Nigeria where the green rosette form of the disease is predominant. Other objectives include a comparison of the impact of green rosette on yield of susceptible and resistant genotypes and an improvement of the understanding of the nature of resistance.

MATERIALS AND METHODS

Genotypes. The eight peanut genotypes used in these experiments were selected on the basis of previous reports with regard to reaction to groundnut rosette (reports variable with green rosette in Nigeria), maturity, and acceptable agronomic traits such as seed size, upright growth habit, and yield. Genotypes RMP 12, RG 1, RRB, and 55-437 were considered resistant; M1204.78I as moderately resistant; and MK 374, JL 24, and ICGS-56(E) were considered to be susceptible. RMP 12, RG 1, and MK 374 were considered late maturing; M1204.78I intermediate maturing; and the rest were early maturing.

Rosette environment. Rosette disease was established in field plots in both 1988 and 1989, following the general procedure described by Bock and Nigam (3). Virus inoculum came from field-grown peanut plants with green rosette containing GRV, GRAV, and GRV satellite RNA. The disease was maintained in susceptible peanut genotype F 452.4 with monthly transfers by *Aphis craccivora* Koch. About 6,000 peanut seedlings of genotype F 452.4, grown in the greenhouse to serve as a source of infectious agents in field experiments, were inoculated at the two- to three-leaf stage with viruliferous aphids. Two weeks after in-

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oculation when rosette symptoms had appeared, all plants were sprayed with the aphicide dichlorvos (Nuvan) to prevent death of the plants from the high population of aphids. A new population of aphids was placed on the infector plants 2 wk before transplanting them in the field.

The eight genotypes were planted (17 July 1988 and 6 June 1989) on research land at the Institute for Agricultural Research Samaru, Ahmadu Bello University, Zaria, Nigeria, in a randomized complete block design with four and three replications in 1988 and 1989, respectively. Seeds were treated with thiram (Fernasan D at 3.3 g/kg of seed). Before planting, 174.9 and 116.6 g of single superphosphate (P_2O_5) plus 81.1 and 54.1 g of muriate of potash (K_2O) fertilizers were applied to the plots in 1988 and 1989, respectively. The preemergence herbicide paraquat was used to reduce weeds; subsequent weed control was done by hand. Each plot consisted of 17 and 11 rows (3.6 m long) in 1988 and 1989, respectively. In 1988, a plot consisted of eight rows of a test genotype with an infector row planted in alternate rows. In 1989, each plot had five rows of a test genotype and six rows of infector plants. Spacing between plants and rows for both years was 20 and 75 cm, respectively. The average number of plants per genotype was 706 and 147 in 1988 and 1989, respectively.

Nine and 15 days after seeding in the field in 1988 and 1989, respectively, rosetted seedlings, heavily infested with aphids from the greenhouse, were transplanted at 1.5 m spacing in each of the infector rows.

Disease evaluation. Beginning 8 days after rosetted plants were transplanted to the infector rows, scorings for green rosette were done at weekly intervals during the first 4 wk and every 2 wk thereafter. Individual plants with symptoms were identified with regard to position within the row at each evaluation. Each plant was evaluated for time of initial symptom expression and disease severity on the basis of the following disease rating system: 1 = no symptoms, 2 = discernible rosette leaf symptoms (no stunting), 3 = rosette leaf symptoms plus stunting ranging from barely discernible to about 30% the size of symptomless plants, 4 = rosette leaf symptoms plus stunt ranging from about 30 to 70%, and 5 = rosette leaf symptoms plus stunt greater than 70%. (Degree of stunting was based on the observation of one evaluator.) Index values were determined by using a rating system similar to that described by Kuhn and Smith (10), as follows: $(A + 2B + 3C + 4D + 5E)$/ total number of plants assessed per plot, where A, B, C, D, and E equal the number of plants with ratings of 1, 2, 3, 4, and 5, respectively. For example, if seven plants were rated 1, 28 rated 2, six rated 3, one rated 4, and zero rated 5, then the index = $7 \times 1 + 28 \times 2 + 6 \times 3 + 1 \times 4 + 0 \times 5 = 85/42 = 2$.

Disease tissue analysis. In 1989, leaf samples of diseased plants were taken from all genotypes (including F 452.4) for nucleic acid and serological diagnostic tests, which were conducted at the University of Georgia. Young leaves of about the same age and size from at least four different rosetted plants per genotype per block were harvested and dried over calcium chloride in a desiccator. Rosette-free leaves were also harvested from RMP 12 and RG 1. All samples were labelled, packaged, and handled in compliance with a shipping permit issued by the Georgia Department of Agriculture and the Animal and Plant Health Inspection Service, U.S. Department of Agriculture.

dsRNA electrophoresis. A double-stranded (ds) RNA electrophoresis method described by Breyel et al (4) was used to detect and quantify a 900-base pair (bp) dsRNA associated with infections of GRV (4,20). Small quantities (one leaflet or about 0.1 g) of desiccated leaf tissue were ground in a mortar with 0.9 ml of water. Restriction endonuclease *Pst*I cut lambda DNA was used as the standard for molecular weights of nucleic acids. An estimate of the quantity of 900-bp dsRNA was made by rating the electrophoresis bands from 1 to 5 where 1 = low intensity and narrow band and 5 = high intensity and broad band.

ELISA. Tests for detection of GRAV were conducted with the double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) method described by Clark and Adams (6). A conjugate was prepared with antiserum raised to potato leaf roll virus (PLRV), a luteovirus serologically related to GRAV, supplied by R. Casper, Federal Biological Research Centre, Braunschweig, Germany. An extract from desiccated tissue (approximately 0.1 g + 0.9 ml of buffer) was prepared in ELISA antigen buffer (PBS-Tween 20 + 2% polyvinylpyrrolidone [Sigma PVP-40] + 0.01 M diethyldithiocarbamic acid [Na DIECA, Sigma D-3506]). Absorbance values at 405 nm were obtained using a Bio-Rad MICROELISA EIA Reader model 2550 (Bio-Rad Laboratories, Richmond, CA).

Rosette-free environment. In 1989, the eight test genotypes were also grown in a second experiment (planted 6 June) located 17 m from the rosette-infected plots. They were arranged in a randomized complete block design with four replications. Each plot of two rows was 8.2 m long. Spacing between plants and rows was 20 and 75 cm, respectively. Each row had 40 plants, thus, 80 plants per plot per genotype. Plots were surrounded by 5 m of corn, *Zea mays* L., planted earlier than the peanut to form a potential aphid barrier, and also by 2 m of resistant genotype RMP 12 between the corn and rosette-free plots.

Plots were fertilized with superphosphate (P_2O_5) and muriate of potash (K_2O) at 26.2 kg P/ha and 26.9 kg K/ha, respectively, broadcast and incorporated before planting. For protection against soil pests, insects, and foliar diseases, the following recommended chemicals were used: thiram (Fernasan D at 3.3 g/kg of seed) for seed treatment, carbofuran (Furadan 3G at 1.5 kg/ha) applied at time of seeding for soil pests, dimethoate (Rogor at 12.5 ml/L of water) applied at 10-day intervals for insects, and mancozeb (Dithane M-45 at 1.68 kg/ha) applied at 14-day intervals for control of fungal leaf spots. Paraquat at 5 L/ha was used to reduce the number of weeds, and subsequent weed control was done by hand.

Yield measurements. In 1989, data were collected on all plants for pod number and weight and seed weight and size. In rosette plots, pod numbers and weight and seed weight were determined for individual plants. In the rosette-free plots, harvests were made on a per plot basis and divided by the number of plants per plot. Seed weight was determined by weighing 100 seeds (to the nearest 0.1 g) per plot.

RESULTS

Disease reactions. RMP 12 and RG 1 were resistant (both reacted similarly for disease incidence and severity) and clearly distinct from six susceptible genotypes (Fig. 1 and Tables 1 and 2). Although disease incidence and severity varied in 1988 and 1989, the overall disease reactions of the eight genotypes to the green rosette disease were similar. In 1988, extremely mild mottling symptoms developed on about 8% of the plants of

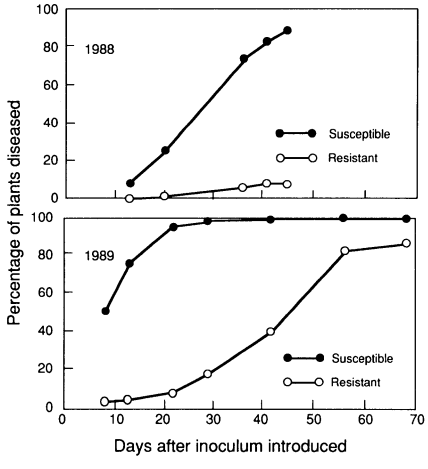

Fig. 1. Disease progress curves of rosette resistant and susceptible peanut genotypes grown in the field at Samaru, Nigeria, in 1988 and 1989. Data points represent the percent of diseased plants of two resistant and six susceptible genotypes (average of four and three replications in 1988 and 1989, respectively).

the resistant genotypes. In 1989, about 87% of their plants displayed mild mottling on young leaves, downward leaf rolling, and dark green color on older leaves, symptoms which were limited to a few leaves of a branch and a few branches. In 1989, 6–7% of the RMP 12 and RG 1 plants showed showed severe stunting (ratings of 3, 4, and 5) by the end of the season (Table 2). The susceptible genotypes showed distinct leaf symptoms by 13 and 8 days after exposure to inoculum in 1988 and 1989, respectively (Fig. 1).

Disease incidence. Disease incidence in the susceptible genotypes progressed more rapidly in 1989 than in 1988 (Fig. 1). By 45 days after introduction of inoculum, the incidence ranged from 83 to 94% in 1988, and that incidence level was attained in 15–20 days in 1989. By 3 wk after exposure to inoculum in 1989, more than 95% of susceptible genotype plants were showing symptoms. In 1988, when 25% (in 20 days) and 75% (in 40 days) of plants in the susceptible genotypes were diseased, only 1 and 7% of plants, respectively, of resistant genotypes were infected (Fig. 1). In 1989, 50% disease incidence in the susceptible genotypes occurred 8 days after exposure to inoculum, whereas it took 46 days for the same amount of infection to occur in resistant genotypes (Fig. 1).

Disease severity. The disease was more severe in 1989 than in 1988 (Tables 1 and 2). Disease index values ranged from 1.1 to 3.5 in 1988 and 2.0 to 4.1 in 1989. RMP 12 and RG 1 had the lowest values in both years, whereas the six susceptible genotypes had similar values, averaging 3.3 and 3.9 in 1988 and 1989, respectively. In 1988, all genotypes had numerous plants with no stunting (disease ratings 1 and 2). Susceptible genotypes with no stunting ranged from 30 to 47% whereas resistant ones ranged from 95 to 100% (Table 1). In 1989, all plants of the susceptible and 16–21% of the resistant genotypes were stunted (disease rating of 3 or higher) (Table 2); furthermore, only 2–5% of the resistant ones were severely stunted.

Yield. In 1989, the susceptible genotypes produced few pods and seeds (Table 3). Plants with disease ratings of 5 had a few pegs and no seeds, whereas those with a disease rating of 4 had many pegs but mostly unfilled pods and a few single-seeded pods. On most plants of RMP 12 and RG 1, pods and seeds appeared to be normal. The average number of pods, pod weight, and seed weight per plant on RMP 12 and RG 1 were 21, 35, and 33 times greater, respectively, than the average values for the six susceptible genotypes.

In 1989, both the susceptible and resistant genotypes were grown in a rosette-free environment (no viruses introduced; insecticides and fungicides were applied) within 17 m of the rosette experiment.

Table 1. Percentage of plants with groundnut rosette disease ratings and disease index values of eight peanut genotypes in field plots under moderate to severe disease conditions at Samaru, Nigeria, in 1988

Genotype	Percent plants/rating no.[a]					Disease index value	Disease classification
	1	2	3	4	5		
RMP 12	91	9	0	0	0	1.1	Resistant
RG 1	90	5	1	1	3	1.2	Resistant
M1204.78I	5	37	17	17	24	3.2	Susceptible
ICGS-56(E)	0	30	16	33	21	3.4	Susceptible
RRB	0	33	14	33	20	3.5	Susceptible
55-437	1	40	19	26	14	3.1	Susceptible
MK 374	1	46	16	24	14	3.0	Susceptible
JL 24	0	34	16	30	20	3.4	Susceptible
LSD						0.59	

[a] Rating scale: 1 = no symptoms, 2 = leaf symptoms, no stunting; and 3–5 = leaf symptoms with stunting varying from slight to more than 70%.

Table 2. Percentage of plants with groundnut rosette disease ratings and disease index values of eight peanut genotypes in field plots under severe disease conditions at Samaru, Nigeria, in 1989

Genotype	Percent plants/rating no.[a]					Disease index value	Disease classification
	1	2	3	4	5		
RMP 12	17	67	14	2	0	2.0	Resistant
RG 1	12	67	16	5	0	2.1	Resistant
M1204.78I	0	0	18	44	38	4.1	Susceptible
ICGS-56(E)	0	0	31	44	25	3.9	Susceptible
RRB	0	0	19	68	13	3.9	Susceptible
55-437	0	0	33	60	7	3.7	Susceptible
MK 374	0	0	19	68	13	4.1	Susceptible
JL 24	0	0	17	82	1	3.8	Susceptible
LSD						0.43	

[a] Rating scale: 1 = no symptoms, 2 = leaf symptoms, no stunting; and 3–5 = leaf symptoms with stunting varying from slight to more than 70%.

Table 3. Yield characteristics of eight peanut genotypes grown in field plots during 1989 at Samaru, Nigeria, under conditions of severe groundnut rosette

Genotype	Yield per plant			Seed size (mg/seed)
	Pod number	Pod weight (g)	Seed weight (g)	
RMP 12	26.3 ± 1.36	24.7 ± 1.68	16.6 ± 1.11	527 ± 8.1
RG 1	15.0 ± 2.87	14.6 ± 2.72	9.8 ± 1.95	457 ± 20.9
M1204.78I	2.3 ± 0.27	1.2 ± 0.44	0.8 ± 0.25	372 ± 25.9
ICGS-56(E)	1.1 ± 0.42	0.7 ± 0.27	0.5 ± 0.16	337 ± 24.2
RRB	0.3 ± 0.27	0.2 ± 0.16	0.0 ± 0.04	ND[a]
55-437	0.0 ± 0.00	0.0 ± 0.00	0.0 ± 0.00	ND
MK 374	2.3 ± 0.27	1.3 ± 0.18	1.0 ± 0.22	437 ± 10.9
JL 24	0.0 ± 0.00	0.0 ± 0.00	0.0 ± 0.00	ND

[a] No data obtained because of too few or no seeds.

Table 4. Yield characteristics of eight peanut genotypes grown in a rosette-free environment in field plots at Samaru, Nigeria, in 1989

Genotypes	Yield per plant			Seed size (mg/seed)
	Pod number	Pod weight (g)	Seed weight (g)	
RMP 12	43	55	37.5	605
RG 1	37	41	23.6	558
M1204.78I	52	64	36.4	573
ICGS-56(E)	33	46	19.4	438
RRB	33	33	16.8	368
55-437	50	41	22.0	368
MK 374	46	53	35.7	545
JL 24	36	36	20.8	415
Mean	41	46.25	26.41	483
LSD	7.65	10.38	14.67	49.7

No plants with any rosette type symptoms were observed. Various yield measurements differed among the genotypes (Table 4), but the differences usually were more closely related to seed size for each genotype than to resistance and susceptibility to rosette.

No yield data were taken in 1988 because of late planting and the adverse effect of the absence of rainfall after the first of October.

Diagnostic tests. Electrophoretic tests revealed the presence of a 900-bp dsRNA in rosetted plants of both susceptible and resistant genotypes (Fig. 2). The dsRNA was found in plants with disease ratings of 2–5 but not in symptomless plants (ratings of 1). The concentration of the dsRNA was consistently less in resistant RMP 12 and RG 1 than in susceptible genotypes (Fig. 2). In six tests using different dilutions of sap from infected plants, susceptible F 452.4 (not one of our primary genotypes) always had larger and more intense dsRNA bands than any of the eight test genotypes. Size and intensity of bands were smaller for RMP 12 and RG 1 than for the susceptible genotypes.

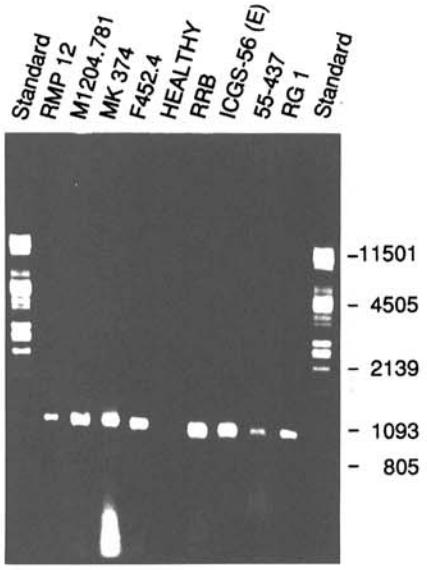

Fig. 2. Electrophoresis of dsRNA in sap from infected peanut genotypes classified susceptible (M1204.78I, MK 374, F 452.4, RRB, ICGS-56(E), and 55-437) and resistant (RMP 12 and RG 1) to groundnut rosette virus. (Susceptible JL 24 was shown to have the dsRNA in other electrophoresis tests.) Desiccated tissue (0.1 g) was macerated in a mortar with 0.9 ml of water. Standard = restriction endonuclease *Pst*I cut lambda DNA (base pairs).

ELISA results revealed the presence of the luteovirus GRAV in both susceptible and resistant genotypes (Table 5). Furthermore, GRAV was detected in resistant genotypes with no symptoms (disease rating of 1), presumably free of GRV. The level of GRAV antigen was consistently higher in F 452.4 than in other genotypes, a condition somewhat analogous to the high concentration of the GRV-related dsRNA in the same host.

DISCUSSION

The highly destructive nature of groundnut green rosette was demonstrated in these studies. Since the dramatic rosette epidemic in Nigeria in 1975 (22), epidemics of lesser severity occurred in 1983, 1985, and 1988. Their impact has been major in some areas, causing farmers to shift from peanut to cereal crops, which are less affected by disease epidemics and other production constraints. The development of rosette-resistant, agronomically acceptable peanut cultivars is required to prevent further decline in peanut production.

A high level of resistance to the green rosette was demonstrated in peanut cultivars RMP 12 and RG 1. Under moderate to severe disease conditions (relatively slow disease progress and high disease incidence by midseason in susceptible genotypes) in 1988, only 10% of the resistant plants developed symptoms and the symptoms were very mild. Under extreme disease conditions in 1989, about 86% of the resistant plants showed symptoms but only 2–5% had the severe rosetting and stunting associated with complete loss of yield. If fields were planted with either of the resistant cultivars, we believe the number of infected plants would be much lower and disease losses negligible, even though a rosette epidemic could be occurring in nearby fields planted with susceptible cultivars. In fact, no plants with rosette symptoms have been observed in several RMP 12 fields in recent epidemic years (1983, 1985, and 1988) in northern Nigeria (S. M. Misari and P. E. Olorunju, *personal observations*).

The impact of groundnut green rosette on yield of RMP 12 and RG 1 was not determined in these studies. About 86% of their plants were infected in 1989, and their yield (seed weight) was an average 57% less than in rosette-free plots in the same field only 17 m away. However, the latter plots were treated with various pesticides to control insects, soil pests, and foliage diseases. In general, foliage growth of the diseased resistant plants appeared to be affected only in a negligible way (P. E. Olorunju, *personal observation*).

Rosette symptoms on resistant genotypes grown in the field have been reported previously by de Berchoux (8), Nutman et al (16), and Bock et al (2). de Berchoux (8) worked with chlorotic rosette, but the other two reports are not specific with regard to the form of rosette used in the experiments. Nutman et al (16) and Bock et al (2) found about 1% of the resistant plants with rosette symptoms, much less than the 86% green rosette incidence we found in resistant RMP 12 and RG 1 in 1989. Presumably, the level of rosette incidence would be related to disease levels in nearby diseased susceptible peanut plants, aphid populations, and environmental factors, such as rain and wind currents, which affect movement of the vectors.

Similar to field studies by Bock et al (2) and Nigam and Bock (15), we found GRAV antigen in rosette-diseased plants of both susceptible and resistant peanut plants and in symptomless (presumably with no GRV) resistant plants. It is clear that rosette-resistant genotypes, such as RMP 12 and RG 1, can be infected by GRAV, but the relative levels of susceptibility or resistance in peanut genotypes have not been tested. GRV satellite RNA appears to be in lower concentration in resistant plants than in susceptible ones.

On the basis of our studies and those of Nutman et al (16), Bock et al (2), and Murant and Kumar (13), there are a few resistance mechanisms that may be operating in rosette-resistant peanut genotypes. First, it is difficult to initiate in-

Table 5. Detection of groundnut rosette assistor virus (GRAV) by ELISA in peanut genotypes susceptible and resistant to groundnut rosette virus (GRV)

Genotypes	Disease reaction[a]	Inoculation site[b]	Disease rating[c]	Samples positive/ samples tested (no.)	A_{405nm} of positive samples[d]
RG 1	R	Field	1	4/4	0.682
RMP 12	R	Field	1	1/4	0.501
RMP 12	R	Greenhouse	4	2/2	0.892
M1204.78I	S	Field	4	2/2	0.705
RRB	S	Field	4	2/2	0.563
MK 374	S	Field	4	1/2	0.609
F 452.4	S	Field	5	2/2	1.703
F 452.4	S	Greenhouse	5	3/3	1.176
Potato[e]	...	...	...	5/5	>2.000
Control[f]	...	Field	...	...	0.176

[a] R = resistant; S = susceptible.
[b] Field = natural infection; greenhouse = viruliferous aphids. Plants at both sites exposed to both GRV and GRAV.
[c] 1 = No symptoms; 4 and 5 = leaf symptoms and severe stunt.
[d] Positive samples had to have absorbance values at least twice as large as the negative control.
[e] Desiccated tissue from potato plants infected with potato leaf roll virus.
[f] Desiccated tissue from symptomless peanut plants in the rosette-free plots.

fection. Presumably all of the resistant plants can be infected, but under field conditions where 85–100% of susceptible plants become infected, usually 10% or less of the resistant plants develop symptoms. Second, initial symptom appearance is greatly delayed in resistant plants, suggesting a restriction of virus replication, virus movement within plants, or perhaps synthesis of the GRV satellite RNA. Restriction of virus movement is supported by the observation of symptoms on only a portion of leaves and branches on diseased plants.

Field screening for resistance to groundnut rosette requires extreme care to clearly distinguish susceptible and resistant genotypes (3) and individual plants in segregating populations (17). Three genotypes (RRB, M1204.78I, and 55-437) previously classified resistant were determined to be susceptible under conditions of high disease levels. We propose that screening for resistance to rosette can be done more effectively and with a major savings in time and cost by mechanical inoculation of GRV and its satellite RNA under greenhouse conditions (18). Inoculum efficiency can be controlled and the complexity of a mixed virus infection avoided.

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