Groundnut Rosette

A Virus Disease Affecting Groundnut Production in Sub-Saharan Africa

R. A. Naidu
ICRISAT, Lilongwe, Malawi

F. M. Kimmins
Natural Resources Institute, Chatham Maritime, Kent, UK

C. M. Deom
University of Georgia, Athens

P. Subrahmanyam
ICRISAT, Lilongwe, Malawi

A. J. Chiyembekeza
Chitedze Agricultural Research Station, Lilongwe, Malawi

P. J. A. van der Merwe
ICRISAT, Lilongwe, Malawi

Groundnut (peanut, *Arachis hypogaea* L.) is cultivated in the semiarid tropical and subtropical regions of nearly 100 countries on six continents between 40°N and 40°S (Fig. 1). For people in many developing countries, groundnuts are the principal source of digestible protein (25 to 34%), cooking oil (44 to 56%), and vitamins like thiamine, riboflavin, and niacin (65). In many countries, groundnut cake and haulms (straw, stems) are used as livestock feed. Groundnut is also a significant source of cash income in developing countries that contributes significantly to food security and alleviates poverty (68). In many sub-Saharan African (SSA) countries, women predominantly grow and manage the crop (Fig. 2). Therefore, groundnut cultivation has a direct bearing on the overall economic and financial well-being and nutritional status of women and children.

As a legume, groundnuts improve soil fertility by fixing nitrogen and thereby increase productivity of the semiarid cereal cropping systems (68). Groundnut requires few inputs, making it appropriate for cultivation in low-input agriculture by smallholding farmers. (Smallholding farmers have holdings of less than 1 ha and grow different crops, often in mixtures. Smallholding farmers use hand tools and have limited resources for agricultural operations.) Groundnuts are grown in most of SSA by smallholding farmers as a subsistence crop under rain-fed conditions, either once or twice a year, depending on rainfall patterns. A few commercial farms also grow groundnut in large areas under irrigation.

Groundnut rosette disease was first reported in 1907 from Tanganyika (85), now called Tanzania, and has since been reported in several other countries in SSA (Fig. 1). Symptoms similar to groundnut rosette disease have been reported in some countries of Asia and South America, but diagnostic tests to unequivocally confirm the presence of the disease have not been conducted (58). Thus, it is generally assumed that groundnut rosette disease is endemic to groundnut growing countries of SSA and its off-shore islands such as Madagascar. Since groundnut rosette disease is limited to SSA, it is likely that groundnut, introduced from South America sometime during the sixteenth century by the Portuguese, was infected by a pathogen endemic to SSA and is therefore an example of the new-encounter phenomenon. The new-encounter phenomenon occurs when a crop has been introduced into a new geographical region and pests and/or pathogens that evolved with other host species attack the newly introduced crop (14).

Although the debilitating impact of groundnut rosette disease epidemics was documented in a few instances (6,84), the longer term consequences of such epidemics on economic and sociological issues affecting the smallholder farmer need further study. The poor documentation of the impact of groundnut rosette disease in SSA is probably due to misdiagnosis because of the lack of adequate resources to conduct reliable surveys and a lack of knowledge of the disease. The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) estimates that groundnut rosette disease causes greater yield loss than any other virus disease affecting groundnut in the semiarid tropics of the world (Table 1, Fig. 3).

Epidemics of groundnut rosette disease in SSA often significantly reduce groundnut production and cripple the rural economy. In 1975, an epidemic in northern Nigeria destroyed approximately 0.7 million ha of groundnuts, with an estimated loss of $250 million (84). Recurrent epidemics have limited production to below the pre-1975 yields. Similarly, the epidemic that occurred in 1995 in eastern Zambia affected approximately 43,000 ha, causing an estimated loss of $4.89 million. In the following year, in the central region of neighboring Malawi, groundnut production was reduced 23% by groundnut rosette disease (6).

H. H. Storey began his pioneering work on groundnut rosette disease in South Africa more than 70 years ago and demonstrated that it was caused by a virus disease transmitted by the aphid *Aphis craccivora* Koch. (70–72). Much of the subsequent work until the 1970s was done by scientists of colonial powers in different Anglophone and Francophone countries of SSA: the British in Malawi, Tanzania, Nigeria and Uganda, and the French in Senegal, Burkina Faso, and Cote d'Ivoire. Most of these studies focused on breeding for resistance, insect vector control through chemical sprays, and modification of cultural practices such as date of sowing and plant densities (1,2,12,17,18,27–29,32,37,64,67,69,70). Despite these efforts, many of the recommendations to reduce the risk of groundnut rosette disease were not adopted by smallholder farmers because crucial socioeconomic factors were not considered by researchers (i.e., quality and duration of resistant varieties, labor constraints, etc.). Although it became clear in the 1960s that more than one virus was associated with the disease (36), detailed understanding of the etiology of groundnut rosette disease came about as a result of collaborations

Dr. Naidu's present address is: Department of Plant Pathology, University of Georgia, Athens 30602-7274; Fax: 706-542-1262; E-mail: naidu@arches.uga.edu

Publication no. D-1999-0519-02F

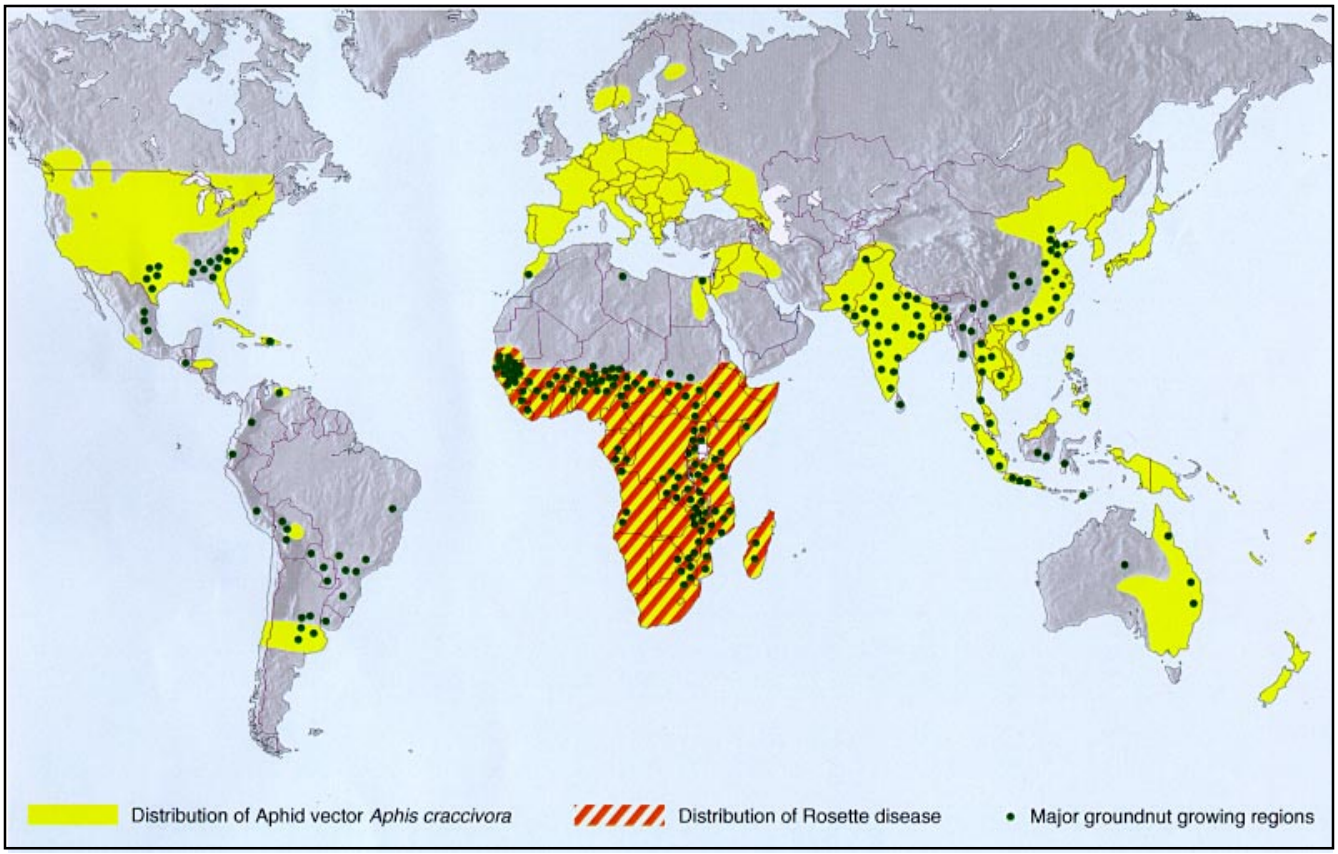


Fig. 1. Worldwide distribution of groundnut rosette disease (yellow- and red-hatched areas) and the aphid vector, *Aphis craccivora* (yellow areas). Major groundnut growing regions are indicated by green dots.

among scientists from the Scottish Crop Research Institute (SCRI) in the UK, ICRISAT, the University of Georgia in the United States, and the African national programs. Thus, by the early 1990s, it was established that groundnut rosette disease had a complex etiology involving three agents: groundnut rosette assistor luteovirus (GRAV; 44,66), groundnut rosette umbravirus (GRV; 45,76), and a satellite RNA (sat RNA; 7,48) of GRV (Table 2). It is quite understandable that this complex etiology and the lack of diagnostic tools were major constraints in understanding the epidemiology of and in developing management strategies for the disease.

Although plant virus diseases with similar etiological characteristics have been reported, the most compelling reasons for this article are the complex and fascinating etiology of groundnut rosette disease and the great economic importance of the disease to groundnut production and food security of smallholder farmers in SSA.

Biology of Groundnut Rosette Disease

Disease symptoms. The two predominant symptom types of groundnut rosette disease are "chlorotic" and "green" rosette (Figs. 4A and B, 5A and B). Chlorotic rosette is ubiquitous in SSA, while the distribution of green rosette is unknown.

Fig. 2. Smallholder farmer managing a groundnut crop on a small, family-owned farm in Malawi.

Fig. 3. Groundnut rosette disease in a susceptible line of groundnut on a smallholder farm.

Table 1. Virus diseases identified as major constraints to groundnut production in semiarid tropical countries[a]

Disease	Distribution	Yield loss[b]	Potential yield gain through crop improvement[b]
Groundnut rosette	Sub-Saharan Africa	156	121
Bud necrosis	Indian subcontinent, Thailand, China	89	45
Peanut mottle	Worldwide	59	35
Peanut clump	Indian subcontinent, West Africa	38	22
Peanut stripe	Asia, S. America, USA	36	18
Tomato spotted wilt	USA (Georgia)	43[c]	…

[a] ICRISAT medium-term plan (1994 to 1998).
[b] US dollars in millions.
[c] Reference 13.

The expression in young plants of either form of the disease affects the entire plant and causes severe stunting due to shortened internodes and reduced leaf size, leading to a bushy appearance. In contrast, plants infected late in their growth may show symptoms only in some branches or parts of branches. Infected plants may show symptoms other than the typical rosette (chlorotic and green) symptoms. For example, a low incidence of mosaic rosette commonly occurs in Eastern and Southern Africa (72). In addition, in some West African countries, symptoms of groundnut rosette disease (green rosette) resemble symptoms of peanut clump disease (caused by peanut clump furovirus; 50), making it difficult to differentiate and determine the distribution and impact of groundnut rosette disease on groundnut production. Variability in disease symptoms could be due to diversity among the causal agents, differences in genotype response, variable climatic conditions, and mixed infections with other viruses. Therefore, to confirm the presence of the disease and to document the variable symptom types, it is important that samples are tested for the three agents of groundnut rosette disease as well as for other viruses.

Effect on yield. Yield losses due to groundnut rosette disease depend on the growth stage of the plant when infection occurs. A 100% loss in pod yield due to either chlorotic or green rosette disease may result if infection occurs before flowering (Fig. 6). Yield loss is variable if infection occurs between flowering and the pod maturing stage, whereas subsequent infections cause negligible effects.

Unlike other members of the family *Luteoviridae*, which often cause yellowing, reddening, and/or stunting of their host plants, GRAV infection alone is asymptomatic in groundnut. Whether GRAV causes any deleterious effects on the host plant, either alone or together with GRV and its sat RNA in a synergistic manner, has not been determined.

Causal agents. Properties of the three agents of groundnut rosette disease are given in Table 2. GRAV, GRV, and sat RNA are intricately dependent on each other, and all three play a crucial role in the biology and perpetuation of the disease. The sat RNA was shown to be largely responsible for disease symptoms (48). Variants of sat RNA cause different forms of the disease (46,48), whereas GRAV or GRV alone cause asymptomatic infections. GRAV acts as a helper virus in vector transmission of GRV and sat RNA, in that GRV RNA, which does not encode for a coat protein, and sat RNA are packaged in the coat protein of GRAV to form virus particles that are aphid-transmissible. The sat RNA depends on GRV for replication, and GRV, although autonomous, depends on sat RNA for aphid transmission. The presence of sat RNA in the source plant is essential for encapsidation of GRV RNA (60), and therefore for the GRAV-dependent transmission of GRV (45). Thus, all three agents must occur together for transmission by the aphid vector and subsequent disease development.

The complete nucleotide (nt) sequence and genome organization of GRV are known. GRV RNA is 4,019 nt long and contains four open reading frames (ORF) (76). The sat RNA variants associated with chlorotic and green rosette disease from different regions of Africa are 895 to 903 nt long and are at least 87% identical (7).

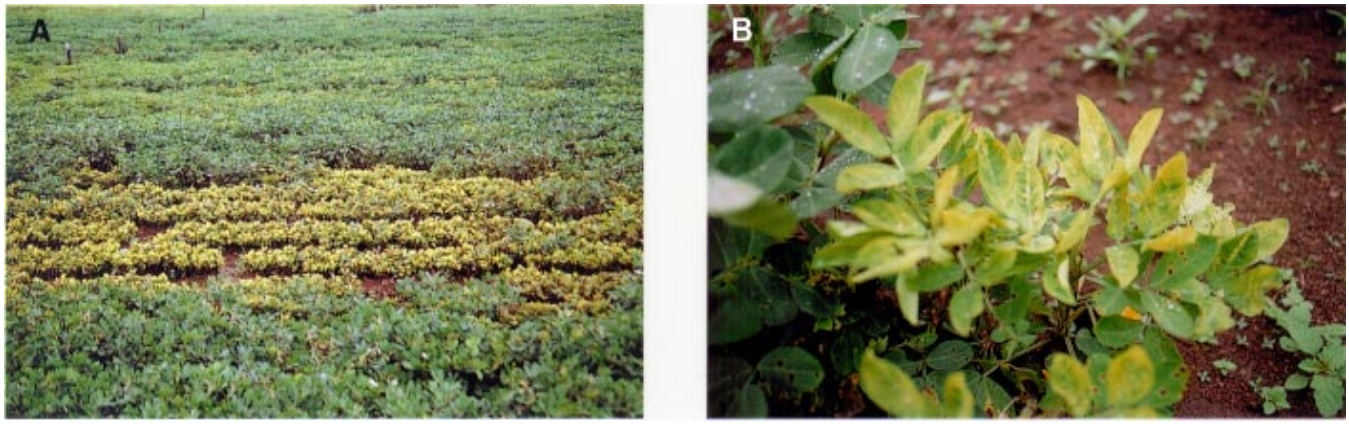


Fig. 4. (A) Groundnut plants in Malawi showing the chlorotic rosette symptoms of groundnut rosette disease (center). Typical disease symptoms are chlorosis, severe curling of leaflets, stunting, and the rosette appearance. (B) A close-up of a plant showing the chlorotic rosette symptoms of groundnut rosette disease.

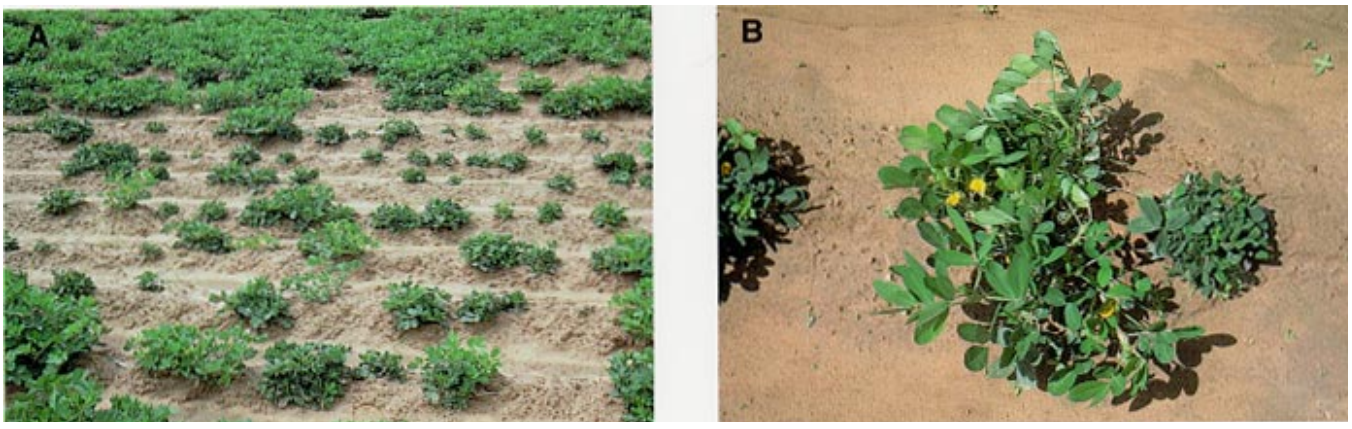


Fig. 5. (A) Groundnut plants in Nigeria showing the green rosette symptoms of groundnut rosette disease. (B) Close-up of a plant showing the green rosette symptoms of groundnut rosette disease (right) and a healthy plant (left). Typical disease symptoms are dark green coloring of leaflets, stunting, and the rosette appearance.

The sat RNA variants contain up to five potential ORFs in either the positive or the negative sense, but none of the ORFs are required for disease or symptom development. Therefore, the role of sat RNA in groundnut rosette disease is RNA-mediated. Some sat RNA variants were found to induce only mild symptoms in *Nicotiana benthamiana* and drastically diminished the replication of GRV. Domains within the sat RNA molecule have been identified that are responsible for sat RNA replication, down-regulation of GRV replication, and symptom production in *N. benthamiana* (74,75). GRAV is a distinct luteovirus based on coat protein sequence data (66); however, the complete genome organization of GRAV is needed to assign its taxonomic status within the family *Luteoviridae*. GRAV is phloem-limited, whereas GRV and sat RNA are not. Although the genome organization and replication strategies of the three agents are distinct, it is not clear how and where in the infected plant the packaging of GRV RNA and sat RNA into the coat protein of GRAV occurs.

Diagnosis. Groundnut rosette disease can be tentatively diagnosed in the field based on the characteristic symptoms in groundnut or by mechanical inoculation onto a suitable indicator host such as *Chenopodium amaranticolor.* Symptom development on *C. amaranticolor* indicates the presence of GRV, but this test is not always reliable when the indicator plants are subjected to the widely fluctuating temperatures of SSA. Improved diagnostic methods (Table 2) now include a triple antibody sandwich enzyme-linked immunosorbent assay (TAS-ELISA) for detection of GRAV (57), a dot blot hybridization (DBH) assay for detection of GRV and sat RNA (8), and reverse transcription-polymerase chain reaction (RT-PCR) that allows detection of each of the three agents (51). The advantage of the RT-PCR method is that it concurrently detects all three groundnut rosette disease agents in plants and aphids.

Fig. 6. Effects of groundnut rosette disease on yield. Farmer is holding a healthy plant in her right hand and a plant with green rosette symptoms of groundnut rosette disease in her left hand. Because there is a 100% loss in pod yield in the diseased plant, the plant was likely infected prior to flowering.

The aphid vector. *A. craccivora* (Homoptera, Aphididae; Fig. 7) is the principal aphid vector of groundnut rosette disease agents. *A. craccivora* is widely distributed in many countries around the world (Fig. 1; 5). In the tropics, only females have been recorded, and these reproduce parthenogenetically throughout the year. The adults have a black, shiny body with a prominent tail-end and are either winged (alatae) or wingless (apterae). The development of these different morphs is due to a combination of factors: tactile stimulation, host plant quality, and climatic conditions (24). Alatae produce only about half the progeny produced by apterae. The nymphs undergo four nymphal stages before developing into adults; under favorable conditions the development of one generation takes 6 to 8 days (an average of 5.5 days). An adult can produce larvae for 6 to 7 days at the rate of 2 to 3 per day or a total fertility of 13 to 14 descendants. Thus, with each generation, the degree of infestation on affected plants may increase five- to eightfold (41). This rate of reproduction is largely dependent on climatic factors, especially temperature, and the nutritional status of the plant host.

Disease–vector relationships. Groundnut rosette disease is of particular interest because GRV and sat RNA must be packaged within the GRAV coat protein to be aphid transmissible (Fig. 8). Thus, all virus particles, irrespective of whether they contain GRAV RNA or GRV RNA and sat RNA, are acquired by the aphid vector from phloem sap. Once acquired, the aphid

Fig. 7. *Aphis craccivora,* the aphid vector of groundnut rosette disease. Shown are black, winged adult aphids (alatae) and smaller brown nymphs.

Table 2. Properties of the three agents of rosette disease

Agent	Genus	Replication	Transmission on groundnuts		Symptoms	Detection[a]		
			Mechanical	Aphid		TAS-ELISA	DBH	RT-PCR
GRAV	*Luteovirus*	Autonomous	No	Yes	Symptomless infection (transient mottle)	Yes	Yes	Yes
GRV	*Umbravirus*	Autonomous	Yes	Yes, requires GRAV & sat RNA	Symptomless infection (transient mottle)	No	Yes	Yes
sat RNA	…	Requires GRV	Yes, requires GRV	Yes, requires GRAV & GRV	Chlorotic, green, mosaic, etc.	No	Yes	Yes

[a] TAS-ELISA, triple antibody sandwich enzyme-linked immunosorbent assay; DBH, dot blot hybridization; RT-PCR, reverse transcription-polymerase chain reaction.

can potentially transmit virus particles for up to 14 days and possibly for life (43,71,81). All developmental stages of the aphid may acquire and transmit the disease.

In previous studies (25,43,71,81), determining successful transmission of the agents causing groundnut rosette disease by *A. craccivora* was based solely on symptoms on the inoculated plants. Therefore, these reports determined only if transmission of GRV and its sat RNA occurred, since GRAV causes no obvious symptoms. Our recent studies indicate that the virus–vector relationships of groundnut rosette disease are complex and seem to differ from the typical persistent type of transmission by aphid vectors. Monitoring the probing and feeding of *A. craccivora* on groundnut by the Electrical Penetration Graph (EPG) technique (79) and testing the inoculated plants for GRAV, GRV, and sat RNA indicated that while GRAV, like other luteoviruses, must be inoculated into phloem cells, GRV and sat RNA infection can also occur if the aphid vector makes exploratory probes into mesophyll cells. (F. M. Kimmins, *unpublished data*). Detailed studies are in progress to understand the intricacies of transmission of the disease agents and to use this information in disease epidemiology and resistance breeding programs.

Epidemiology of Groundnut Rosette Disease

The epidemiology of groundnut rosette disease is complex, involving interactions between two viruses and a sat RNA, the aphid vector, and the host plant in the unpredictable environments of SSA. Therefore, a combination of disciplines and approaches is needed to understand the dynamics of the disease operating in different regions of SSA. Studies were initiated at several locations across SSA to better understand the epidemiology of the disease, but the lack of etiological knowledge and diagnostic tools precluded rapid advances. Therefore, several of the important aspects of rosette disease, such as the underlying causes of groundnut rosette disease epidemics, are unresolved (49).

Where does the disease come from? The seasonal cycle of infection is a major aspect of groundnut rosette disease epidemiology that remains unknown. Possible sources from which rosette could spread are infected groundnut plants surviving between cropping seasons (ground keepers), and/or alternative host plants. The available information on the relative importance of these primary sources in the spread of groundnut rosette disease from Tanzania (27), Uganda (16), Nigeria (12), and Malawi (3,35; R. A. Naidu, *unpublished data*) is conflicting. This is not surprising given the diversity of crop seasons and farming systems of SSA.

As groundnut rosette disease is endemic to SSA and its off-shore islands (Fig. 1), we assume that there are native African plants from which the disease spreads into groundnut. The vector, *A. craccivora*, is polyphagous, and as many as 142 plant species in 23 families in addition to groundnut have been identified on which the aphid can survive (3,26,42). Of these species, 83 are in the Leguminosae, suggesting that *A. craccivora* has a strong preference for legume hosts. One or more of these 142 plant species could be the source of the rosette complex. However, although host plants for GRAV and/or GRV and sat RNA have been identified under experimental conditions (3,25,36,44,59), groundnut is the only known natural host for the entire rosette complex. The development of the specific diagnostic techniques described earlier has enhanced the chances of identifying such alternative hosts and of understanding their role in the perpetuation of the disease.

Primary versus secondary spread of the disease. The relative importance of primary spread of groundnut rosette disease into, and secondary spread within, the groundnut crop is uncertain. Groundnut rosette disease is regarded as a polycyclic disease because each infected plant serves as a source for initiating subsequent spread in the field. Since the viral agents of the disease are not seed-borne, the primary infection must be introduced into the crop by viruliferous aphids. Secondary spread from the initial foci of disease within a field occurs by way of apterae and nymphs (12,16,27–29). The nature and pattern of disease spread can be influenced by plant age, crop density, timing and efficiency of transmission by viruliferous aphid vectors that reach the crop, proximity to the source of primary inoculum, climatic factors, and predators and parasitoids of vector populations within the crop.

The source of viruliferous aphids that initiate groundnut rosette disease is unknown. In areas with a single annual groundnut crop followed by a wide temporal gap, aphids that introduce the initial inoculum may have traveled in air currents over long distances. Alternatively, the aphids may travel in stages by successive generations, each moving relatively short distances. Although the behavior and long-distance migration of *A. craccivora* in

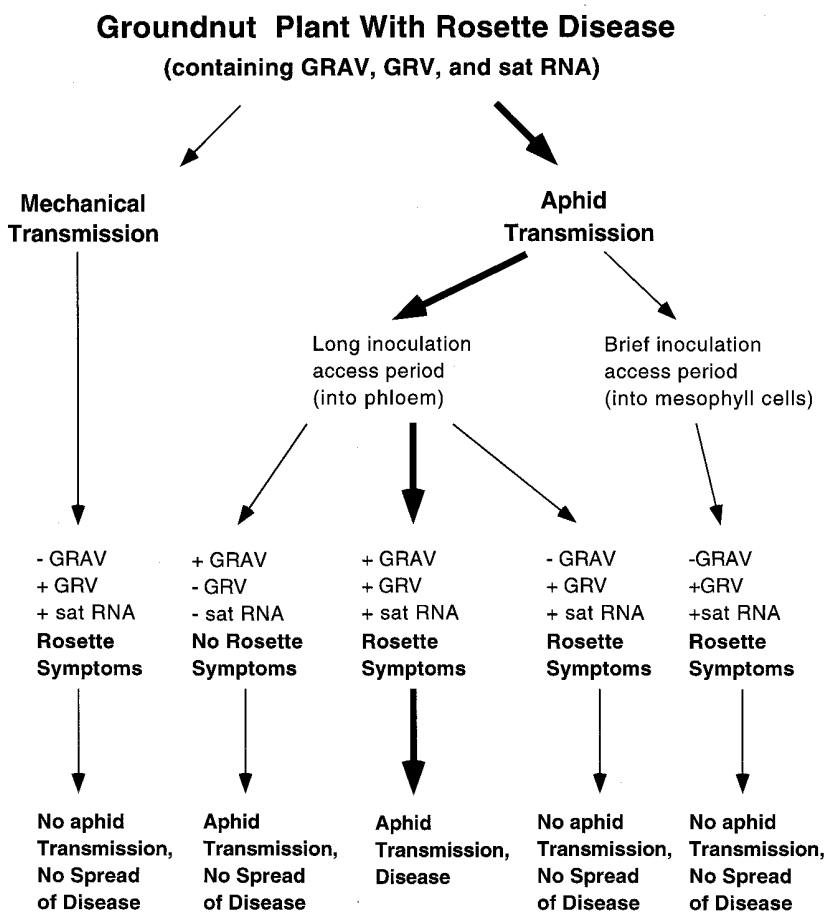


Fig. 8. Separation of groundnut rosette disease agents in time and space during mechanical or aphid inoculations. Aphids fail to transmit groundnut rosette disease in the absence of groundnut rosette assistor luteovirus (GRAV), and plants lacking groundnut rosette umbravirus (GRV) and satellite RNA do not show groundnut rosette disease symptoms. + indicates replication, – indicates no replication, bold arrows indicate epidemiological significance.

temperate Southeast Australia is known (33,38); there is no direct evidence for such migratory flights in tropical SSA. Nonetheless, high-altitude trappings and other circumstantial evidence from Africa (Rainey in 78) suggest that *A. craccivora* has the potential to disperse over long distances. Collection of data on aphid migrations would be operationally difficult in SSA, but these studies may be able to be conducted by utilizing the opportunities afforded by Geographic Information Systems (GIS) and radar-based technologies, as has been done with other migratory pests like desert locusts and armyworm (20).

In general, early primary infection provides a good opportunity for repeating cycles of infection to occur before crops mature and vector populations decline. However, the secondary spread of groundnut rosette disease is complicated because a single aphid may not always transmit the three causal agents (R. A. Naidu, F. M. Kimmins, J. Holt, D. J. Robinson, C. M. Deom, and P. Subrahmanyam, *unpublished data*). Expression of disease symptoms does not necessarily indicate the presence of aphid-transmissible GRAV in infected plants (51, Fig. 8). Plants that show symptoms but lack GRAV play no role in the spread of the disease because the coat protein of GRAV is needed for encapsidation and transmission of GRV and sat RNA (45,60). It is the number of plants containing all three agents that plays a crucial role in the secondary spread of the disease in a given field, while the total number of plants showing disease symptoms, irrespective of having GRAV, influences yield. This needs to be considered while correlating disease incidence with development of epidemics.

Aphid biotypes. Previous studies have reported variation in host plant preferences and vectoring ability among clones of *A. craccivora* obtained from distinct regions of SSA and from different host plants (39,53,71,81). Therefore, additional studies are required to understand the relation between aphid populations and transmission competence of different biotypes in a given population.

Disease forecasting. Aphid population dynamics, distribution and abundance of sources of inoculum, cropping patterns, and meteorological parameters are the key components in predicting the risk factors for groundnut rosette disease. Each of these factors will need to be considered to generate an infectivity index for developing a disease forecasting system. Considerable progress has been made in forecasting the incidence of other vector-borne viruses, including several that persist in aphid vectors such as potato leafroll luteovirus, beet yellowing luteovirus, and barley yellow dwarf luteovirus (34). A similar approach may be appropriate for groundnut rosette, as it would enhance and maximize the various crop management options being developed for smallholder farmers in SSA.

Management of Rosette Disease

Methods that have been investigated to manage groundnut rosette disease include pesticides to reduce vector aphid populations, cropping practices to delay onset and spread of both vector and disease, and breeding for virus and vector resistance. Limited success has been achieved with each, and in recent years, efforts have focused on the latter two tactics for disease management.

Control by chemical pesticides. Organophosphorus pesticides have been used to control aphid vector populations to minimize spread of groundnut rosette disease in the field (17,18,27,69). The timing, dosage, and type of insecticidal applications are critical for effectively diminishing the vector population and require an early forecast of vector migration into the crop. However, chemical pesticides are an unlikely control measure since this approach is not economically feasible for smallholder farmers in SSA. In addition, improper use of these potent chemicals might alter the delicate balance between aphid vectors and their natural enemies, and possibly result in the development of insecticide-resistant biotypes.

Cropping practices. Studies carried out in different parts of SSA, where a single groundnut crop alternates with a long dry season, indicate that early sowing and maintaining a uniformly dense stand of groundnut greatly reduces the incidence of groundnut rosette disease (1,2,12,19,28–30,37,64,67). The early sown crops cover the ground before the aphids' main period of flight activity. Such crops largely escape infection because aphids prefer younger crops and often alight preferentially on widely spaced plants. However, these practices are seldom adopted by the traditionally conservative smallholder farmers in SSA because they prefer sowing their main staple food crops (cereals like maize and sorghum) first. Groundnut, provided sufficient seed is available, is left until later in the season or until the second rains. In many countries, farmers do not remove volunteer groundnuts and ground-keepers that start growing with the first rains. This permits aphid vectors to colonize and infect such plants, which in turn serve as primary sources of inoculum for the subsequently sown crops. In addition, early sowing may not be effective in areas with overlapping groundnut crops where the vector and disease agents perpetuate throughout the year. Many times, early sown crops must be harvested during wet weather, causing problems of drying and predisposition to fungi, which may result in the accumulation of mycotoxins (23,82,83). Furthermore, weeding of groundnut fields is often delayed due to labor constraints and, in many cases, due to the preoccupation of farmers with other food and cash crops, resulting in poor plant growth and yield.

In many parts of SSA, groundnut is intercropped with cereals such as sorghum, maize, and finger millet or legumes like cowpea, beans, pigeonpea, and soybean. Intercropping with beans (30) or sorghum (4) was reported to be effective in reducing the disease incidence. Additional studies are required on the effectiveness, feasibility, and acceptability of this approach to groundnut rosette disease management. In addition, the role of varietal mixtures (a portfolio of cultivars with a range of desired traits) needs to be compared with monocultures of crop varieties for effectively managing groundnut rosette disease.

Host-plant resistance. Sources of resistance to groundnut rosette disease were first identified in groundnut land races of the late-maturing Virginia type (*A. hypogaea* subsp. *hypogaea*, var. *hypogaea*) from Burkina Faso and Cote d'Ivoire in West Africa (62,63). More recently, resistance to groundnut rosette disease has been identified in the early-maturing Spanish type (*A. hypogaea* subsp. *fastigiata*, var. *vulgaris*) (80). Resistance identified in races of the Virginia type was used in breeding programs throughout SSA and has contributed to the development of several disease-resistant cultivars (e.g., RMP 12, RMP 91, KH 241-D, and RG 1) (11,31). Resistance among these cultivars was found to be effective against both chlorotic and green rosette and was governed by two independent recessive genes (21,52).

The major disadvantage of these cultivars is that they require a long growing season (150 to 180 days) to attain maturity, making them susceptible to drought during the end of the season. They are also characterized by a spreading growth habit and small pods. The challenge was to combine groundnut rosette disease resistance with early-maturing (90 to 110 days), high-yielding Spanish types suitable for smallholder farmers in different ecosystems of SSA. The Southern African Development Community/International Crops Research Institute for the Semi-Arid Tropics (SADC/ICRISAT) Groundnut Project based at Chitedze Agricultural Research Station, Lilongwe, Malawi, launched a program in the early 1980s to develop such cultivars. An effective screening technique developed by K. R. Bock (9) (Fig. 9A) permitted rapid field evaluation of large numbers of segregating populations and breeding lines to identify lines with different growth characteristics and resistance to groundnut rosette disease. This resulted in identifying several high-yielding, agronomically acceptable, short-, medium-, and long-duration genotypes with good levels of resistance to chlorotic rosette disease. In field trials at Chitedze during the 1995-96

and 1996-97 crop seasons, several of these genotypes produced significantly higher yields compared with susceptible varieties (Table 3). On-farm evaluation of these genotypes for 3 years in different production systems of Malawi (15) showed that ICGV-SM 90704 (a medium-duration Virginia bunch type) and ICGs 12988 and 12991 (short-duration, Spanish type) have agronomic characteristics that are desired by Malawian farmers. Their performance is being evaluated for stable and effective resistance against both major forms of the disease (chlorotic and green rosette) in different regions of SSA.

Since 1990, nearly 6,800 germ plasm lines from the global collection of groundnut germ plasm have been screened against chlorotic rosette to identify additional sources of resistance (Fig. 9B). These lines originated from different countries of South America, Africa, and Asia and are available in the gene bank at ICRISAT-Patancheru, India. About 100 long-duration Virginia types and 15 early-maturing Spanish types have a high level of resistance to groundnut rosette disease (73). These additional sources should be invaluable in breeding programs to broaden the genetic base of resistance and ensure stability of resistance.

It is interesting to note that none of the groundnut rosette disease-resistant cultivars and germ plasm lines identified so far have resistance to GRAV (55,73). In addition, it is debatable whether the resistance to groundnut rosette disease conferred by the double recessive genes will remain stable and effective in all genotypes. Therefore, to maximize their effectiveness, all resistant material developed needs to be critically evaluated for performance against a range of variants of groundnut rosette disease agents in different environments.

Alternative breeding strategies. In all resistant cultivars and germ plasm lines which have been analyzed, resistance is to GRV. Resistance to GRV results in indirect resistance to sat RNA, and thus such genotypes do not develop symptoms (10). However, resistance to GRV does not amount to immunity and can be overcome under high inoculum pressure or adverse environmental conditions (10,53). In addition, all previous studies done on inheritance of disease resistance were based on visual symptoms and are applicable only to GRV and its sat RNA, but not GRAV.

In contrast, immunity to GRAV was identified in several wild *Arachis* species or accessions (47; R. A. Naidu, *unpublished data*). This provides an opportunity to transfer immunity to GRAV into cultivated groundnut through conventional breeding and/or through biotechnological approaches. Resistance to the aphid vector, identified in groundnut genotype EC36892 (56), represents another strategy that is being exploited in the resistance breeding programs. On plants of genotype EC36892, aphids initiate phloem feeding, but sustained feeding is not maintained, which results in a short feeding period and presumably the aphid-resistance phenotype. Pathogen-derived resistance (22) to groundnut rosette disease agents could be generated by transforming suitable groundnut cultivars with gene sequences from the GRAV replicase and coat protein genes, the GRV replicase and movement protein genes, and/or sat RNA-derived sequences that down-regulate GRV replication (74,75,77). Exploitation of the pathogen-derived resistance approach is delayed due to the lack of efficient protocols for transformation of groundnut.

Plant resistance combined with cultural practices are key components for developing a successful management program against groundnut rosette disease. Understanding the epidemiological principles of the disease combined with resistance will lead to the development of sustainable integrated disease management strategies.

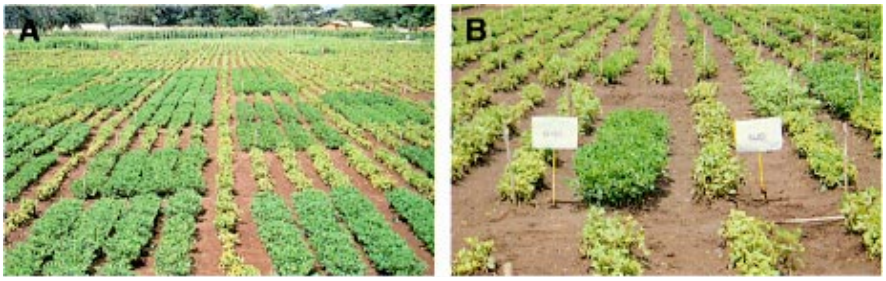


Fig. 9 (A) Groundnut rosette disease screening nursery at the Chitedze Agricultural Research Station, Lilongwe, Malawi. Nursery allows for large and rapid screening of germ plasm and breeding lines for resistance to chlorotic type of groundnut rosette disease and the identification of resistant lines with agronomically important traits. (B) Close-up of nursery showing a groundnut germ plasm line (Virginia type) with resistance to groundnut rosette disease (ICG 12415, left) and susceptible lines, such as Malimba (right).

Table 3. Performance of selected groundnut genotypes in field trials at Chitedze Agricultural Research Station, Lilongwe, Malawi[a]

Genotype	Groundnut rosette incidence (%)	Yield (t ha^{-1})		Shelling (%)
		Pod	Haulms[b]	
Virginia types (*Arachis hypogaea*, subsp. *hypogaea*, var. *hypogaea*)				
ICGV-SM 90704	5.0	0.59	3.27	67.0
ICGV-SM 91706	8.0	0.97	3.83	64.0
CG 7 (susceptible control)	53.0	0.23	3.19	62.0
Trial mean	21.9	0.69	3.62	64.8
LSD	6.2	0.15	0.47	2.3
CV (%)	34.5	27.0	15.9	4.4
Spanish types (*A. hypogaea*, subsp. *fastigiata*, var. *vulgaris*)				
ICGV-SM 93523	14.0	0.85	1.47	63.0
ICGV-SM 93524	14.0	0.87	1.88	64.0
ICGV-SM 93535	6.0	0.87	1.91	56.0
ICG 12988	12.0	1.42	2.04	75.0
ICG 12991	6.0	1.41	2.01	75.0
JL 24 (susceptible control)	67.0	0.34	1.80	61.0
Trial mean	19.6	0.96	1.85	65.6
LSD	2.0	0.16	0.30	2.3
CV (%)	10.1	16.4	16.0	3.4

[a] Average values of two growing seasons, 1995-96 and 1996-97. LSD, least significant difference ($P < 0.05$); CV, coefficient of variance.
[b] Peanut straw and stems.

Outlook

Information on the etiology and molecular characteristics of the causal agents of groundnut rosette disease provides a better understanding of the intricacies associated with the disease. This knowledge, together with recently developed diagnostic tools, gives impetus to addressing unresolved issues of the disease and to utilizing such information in groundnut improvement programs.

RNA viruses exist as "quasispecies" (61) in infected plants, and thus the population complexity of GRAV, GRV, and sat RNA in the field has the potential to be large. The potential permutations among variants of the three agents able to form viable alternatives and their capacity to adapt to diverse and changing eco-niches is enormous. With time, this continuous "evolution" of groundnut rosette disease agents under strong selection pressure can lead to new disease patterns. For example, in Nigeria, a clear shift occurred from

green to chlorotic rosette over a period of about 20 years (43,50,84). The shift could be due to changes in the genome sequences of the groundnut rosette disease agents or to different vector biotypes and cropping patterns. Therefore, the degree of diversity among disease agents, the population dynamics of the vector, the role of vector biotypes and their host plant affiliation, as well as other, as yet undefined, factors that contribute to changes in disease patterns need to be addressed to understand the pathosystem.

Groundnut rosette disease thrives under a variety of contrasting ecologies in the unpredictable semiarid tropical environments of SSA. Hence, the dynamics of the groundnut rosette disease pathosystem are influenced by many biotic and environmental factors. The most critical information lacking about groundnut rosette disease epidemiology involves the off-season survival of the disease agents and aphid vector, and their spread during the cropping season into and between groundnut crops (49). These gaps must be filled in to develop efficient technologies for groundnut rosette disease management. Indeed, this is a challenging task given the ecological complexity and institutional and sociological framework of SSA. Advances require an integrated and coordinated problem-solving approach if efficient technologies for groundnut rosette disease management are to be developed.

Although it is currently endemic to SSA, the potential for groundnut rosette disease to emerge in the future as a disease of groundnut in other regions of Asia and the Americas exists. *A. crassivora* is ubiquitous in semiarid environments where groundnut is mainly grown (Fig. 1). Therefore, the risk of introduction and subsequent establishment of groundnut rosette disease by the aphid vector to groundnut growing regions outside the SSA needs to be monitored. The introduction of virus diseases across continents has been documented in several crops (40).

A single strategy of development and deployment of cultivars resistant to GRV alone may be a risky proposition for a complex problem like groundnut rosette disease. Conventional breeding efforts need to be combined with alternative breeding strategies, such as pathogen-derived resistance, to broaden the genetic base of resistance and enhance its durability against different variants of groundnut rosette disease agents. The biodiversity value of varietal mixtures to respond to outbreaks of new/virulent forms of groundnut rosette disease yet increase production capacity must be addressed.

The social acceptability of improved groundnut varieties and the economics of their production must also be considered. Ideally, research in the future would focus on developing varieties with combined resistance to multiple pathogens, pests, and stresses. Combining such initiatives with innovative technologies to alleviate labor constraints of sowing, harvesting, and processing will contribute to the sustainability of groundnut in SSA.

Research efforts on groundnut rosette disease for groundnut improvement continues to be a collaborative endeavor. The existing partnerships between institutes, both within and outside Africa, in addressing strategic and applied research aspects of groundnut rosette disease, are a good example of complementarity and synergism aimed at developmental impact. African national agricultural research systems (NARS), farmer groups, community organizations, and nongovernmental organizations (NGOs) constitute vital links between technology development and its delivery to farmer's fields. Increasingly, combining an understanding of the epidemiology of groundnut rosette disease (49) with on-farm research is becoming vital for successful implementation and acceptability of new technologies under low input subsistence agriculture. Detailed research efforts are needed in virology, entomology, plant breeding, molecular biology, modeling, socioeconomics, and technology transfer through long-term coordinated support from donor agencies. A multidisciplinary team effort combining the skills of these rather disparate disciplines is critical in making rapid strides toward development of sustainable groundnut rosette disease management strategies.

Acknowledgments

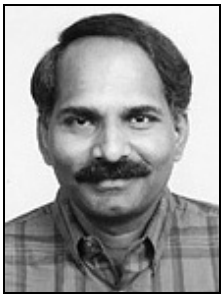
This publication is from research partially funded by the Department for International Development (DFID) of the United Kingdom (Project R 6811; Crop Protection Programme) for the benefit of developing countries and the Peanut Collaborative Research Support Program (Peanut CRSP) of the United States Agency for International Development (Grant LAG-G-00-96-90013-00). The DFID can accept no responsibility for any information provided or views expressed in this article. We thank J. L. Sherwood for critically reviewing the manuscript, J. M. Lenne' for encouragement and support, and the two women farmers for permission to take their pictures. Approved for publication as Journal article JA 2307 by ICRISAT.

Literature Cited

1. A'Brook, J. 1964. The effects of planting date and spacing on the incidence of groundnut rosette disease and of the vector, *Aphis craccivora* Koch, at Mokwa, Northern Nigeria. Ann. Appl. Biol. 54:199-208.
2. A'Brook, J. 1968. The effect of plant spacing on the numbers of aphids trapped over the groundnut crop. Ann. Appl. Biol. 61:289-294.
3. Adams, A. N. 1967. The vectors and alternative hosts of groundnut rosette virus in Central Province, Malawi. Rhodesia, Zambia, Malawi J. Agric. Res. 5:145-151.
4. Alegbejo, M. D. 1997. Survey of the effect of intercropping of groundnut with cereals on the incidence of groundnut rosette virus disease in Northern Nigeria. Intern. Arachis Newsl. 17:39-40.
5. Anonymous 1983. Distribution Map No. 99 (revised 1983). Commonwealth Institute of Entomology, London, Eng.
6. Anonymous 1996. SADC/ICRISAT Groundnut Project Annual Progress Report for 1996. Chitedze Research Station, PPO Box 1096, Lilongwe, Malawi.
7. Blok, V. C., Ziegler, A., Robinson, D. J., and Murant, A. F. 1994. Sequences of 10 variants of the satellite-like RNA-3 of groundnut rosette virus. Virology 202:25-32.
8. Blok, V. C., Ziegler, A., Scott, K., Dangora, D. B., Robinson, D. J., and Murant, A. F. 1995. Detection of groundnut rosette umbravirus infections with radioactive and non-radioactive probes to its satellite RNA. Ann. Appl. Biol. 127:321-328.
9. Bock, K. R. 1987. Rosette and early leaf spot diseases: A review of research progress, 1984/85. Pages 5-14 in: Proc. Regional Groundnut Workshop for Southern Africa, 2nd. K. R. Bock, D. L. Cole, J. A. Wightman, and S. N. Nigam, eds. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, Andhra Pradesh 502 324, India.
10. Bock, K. R., Murant, A. F., and Rajeshwari, R. 1990. The nature of the resistance in groundnut to rosette disease. Ann. Appl. Biol. 117:379-384.
11. Bocklee-Morvan, A. 1983. Les differentes varietes d'arachide. Repartition geographique et climatique, disponibilite. Oléagineux 38:73-116.
12. Booker, R. H. 1963. The effect of sowing date and spacing on rosette disease of groundnut in northern Nigeria, with observations on the vector, *Aphis craccivora*. Ann. Appl. Biol. 52:125-131.
13. Brenneman, T., and Culbreath, A. 1997. Peanut. Page 6 in: 1997 Georgia Plant Disease Loss Estimates. P. F. Bertrand, ed. University of Georgia Cooperative Extension Service, College of Agricultural and Environmental Sciences. Report # Path 98-007.
14. Buddenhagen, I. W., and de Ponti, O. M. B. 1984. Crop improvement to minimize future losses to disease and pests in the tropics. Pages 23-47 in: Breeding for Durable Disease and Pest Resistance. FAO Plant Prod. Prot. Pap. 55. Rome, Italy.
15. Chiyembekeza, A. J., Subrahmanyam, P., and Hildebrand, G. L. 1997. Identification and on-farm evaluation of rosette-resistant groundnut genotypes in Malawi. Pages 20-21 in: Groundnut Virus Diseases in Africa: Summary and Recommendations of the Sixth Meeting of the International Working Group, Agricultural Research Council, Plant Protection Research Institute, Pretoria, South Africa. D. V. R. Reddy, P. Delfosse, J. M. Lenne', and P. Subrahmanyam, eds. International Crops Research Institute for the Semi-Arid Tropics, Andhra Pradesh, India; and Belgian Administration for Development Cooperation, Belgium.
16. Davies, J. C. 1972. Studies on the ecology of *Aphis craccivora* Koch (Homoptera, Aphididae), the vector of rosette disease of groundnuts, in Uganda. Bull. Entomol. Res. 62:169-181.
17. Davies, J. C. 1975. Insecticides for the control of the spread of groundnut rosette disease in Uganda. Pest Articles News Summaries 21:1-7.
18. Davies, J. C. 1975. Use of Menazon insecticide for control of rosette disease of groundnuts in Uganda. Trop. Agric. (Trinidad) 52:359-367.
19. Davies, J. C. 1976. The incidence of rosette disease in groundnut in relation to plant density and its effects on yield. Ann. Appl. Biol. 82:489-501.
20. Day, R. K., Haggis, M. J., Ddiyo, P. O., Mallya, C., Norton, G. A., and Mumford, J. D. 1996. Wormbase: A data management and information system for forecasting *Spodoptera exempta* (Lepidoptera: Noctuidae) in eastern Africa. Entomol. Soc. Am. 89:1-10.
21. de Berchoux, C. 1960. La rosette de l'arachide en Haute-Volta. Comportement des

lignées résistantes. Oléagineux 15:229-233.
22. Deom, C. M. 1999. Engineered resistance. In: Encyclopedia of Virology. 2nd ed. A. Granoff and R. Webster, eds. Academic Press, London. In press.
23. Diener, U. L., Pettit, R. E., and Cole, R. J. 1982. Aflatoxins and other mycotoxins in peanut. Pages 486-519 in: Peanut Science and Technology. H. E. Pattee and C. T. Young, eds. American Peanut Research and Education Society, Yoachum, TX.
24. Dixon, A. F. G. 1985. Structure of aphid populations. Annu. Rev. Entomol. 30:155-174.
25. Dubern, J. 1980. Mechanical and aphid transmission of an Ivory Coast strain of groundnut rosette virus. Phytopathol. Z. 99:318-326.
26. Eastop, V. F. 1981. The wild hosts of aphid pests. Pages 285-298 in: Pests, Pathogens and Vegetation. J. M. Thresh, ed. Pitman Advanced Publishing Program, London.
27. Evans, A. C. 1954. Groundnut rosette disease in Tanganyika. I. Field studies. Ann. Appl. Biol. 41:189-206.
28. Farrell, J. A. K. 1976. Effects of groundnut sowing date and plant spacing on rosette virus disease in Malawi. Bull. Entomol. Res. 66:159-171.
29. Farrell, J. A. K. 1976. Effects of groundnut crop density on the population dynamics of *Aphis craccivora* Koch (Hemiptera, Aphididae) in Malawi. Bull. Entomol. Res. 66:317-329.
30. Farrell, J. A. K. 1976. Effects of intersowing with beans on the spread of groundnut rosette virus by *Aphis craccivora* Koch (Hemiptera, Aphididae) in Malawi. Bull. Entomol. Res. 66:331-333.
31. Gibbons, R. W. 1977. Groundnut rosette virus. Pages 19-21 in: Diseases of Tropical Crops. J. Kranz, J. Schutter, and W. Koch, eds. Verlag Paul Parey, Berlin.
32. Guillemin, R. 1952. Etudes agronomique sur l'arachide en A.E.F. (Oubangi-Chari, Tchad). Oléagineux 7:699-704.
33. Gutierrez, A. P., Nix, H. A., Havenstain, D. E., and Moores, P. A. 1974. The ecology of *Aphis craccivora* Koch and subterranean clover stunt virus in South-East Australia. III. A regional perspective of the phenology of the cowpea aphid. J. Appl. Ecol. 11:21-35.
34. Harrington, R., Mann, J. A., Plumb, R. T., Smith, A. J., Taylor, M. S., Foster, G. N., Holmes, S. J., Masterman, A. J., Tones, S. J., Knight, J. D., Oakley, J. N., Barker, I., and Walters, K. F. A. 1994. Monitoring and forecasting for BYDV control - the way forward? Aspects Appl. Biol. 37:197-206.
35. Hildebrand, G. L., Bock, K. R., and Nigam, S. N. 1991. Groundnut rosette virus: Recent research progress in Southern Africa. Pages 7-8 in: Groundnut Virus Diseases in Africa: Summary Proceedings of the Consultative Group Meeting, Montpellier, France. ICRISAT, Patancheru, A.P. 502 324, India.
36. Hull, R., and Adams, A. N. 1968. Groundnut rosette and its assistor virus. Ann. Appl. Biol. 62:139-145.
37. Jameson, J. D., and Thomas, D. G. 1954. Groundnut rosette disease. Uganda Dep. Agric. Record Investigations No. 3:17-20.
38. Johnson, B. 1957. Studies on the dispersal by upper winds of *Aphis craccivora* Koch. in New South Wales. Proc. Linnean Soc. N.S. Wales 82:191-198.
39. Jones, M. G. 1967. Observations on two races of the groundnut aphid, *Aphis craccivora*. Entomol. Exp. Appl. 10:31-38.
40. Matthews, R. E. F. 1991. Ecology. Pages 562-590 in: Plant Virology. 3rd ed. Academic Press, San Diego, CA.
41. Mayeux, A. 1984. Le puceron de l'arachide - Biologie et contrôle. Oléagineux 39:425-434.
42. Millar, I. M. 1994. A catalogue of the aphids (Homoptera: Aphidoidea) of Sub-Saharan Africa. Plant Prot. Res. Inst. Handb. No. 4.
43. Misari, S. M., Abraham, J. M., Demski, J. W., Ansa, O. A., Kuhn, C. W., Casper, R., and Breyel, E. 1988. Aphid transmission of the viruses causing chlorotic rosette and green rosette diseases of peanut in Nigeria. Plant Dis. 72:250-253.
44. Murant, A. F. 1989. Groundnut rosette assistor virus. CMI/AAB Descriptions of Plant Viruses, No. 345.
45. Murant, A. F. 1990. Dependence of groundnut rosette virus on its satellite RNA as well as on groundnut rosette assistor luteovirus for transmission by *Aphis craccivora*. J. Gen. Virol. 71:2163-2166.
46. Murant, A. F., and Kumar, I. K. 1990. Different variants of the satellite RNA of groundnut rosette virus are responsible for the chlorotic and green forms of groundnut rosette disease. Ann. Appl. Biol. 117:85-92.
47. Murant, A. F., Kumar, I. K., and Robinson, D. J. 1991. Current research on groundnut rosette at SCRI. Pages 18-20 in: Groundnut Virus Diseases in Africa: Summary Proceedings of the Consultative Group Meeting, Montpellier, France. ICRISAT, Patancheru, A. P. 502 324, India.
48. Murant, A. F., Rajeshwari, R., Robinson, D. J., and Raschke, J. H. 1988. A satellite RNA of groundnut rosette virus that is largely responsible for symptoms of groundnut rosette disease. J. Gen. Virol. 69:1479-1486.
49. Naidu, R. A., Bottenberg, H., Subrahmanyam, P., Kimmins, F. M., Robinson, D. J., and Thresh, J. M. 1998. Epidemiology of groundnut rosette virus disease: Current status and future research needs. Ann. Appl. Biol. 132:525-548.
50. Naidu, R. A., Kimmins, F. M., Delfosse, P., Alegbejo, M. D., and Waliyar, F. 1997. Groundnut rosette and peanut clump diseases in West Africa. Pages 49-51 in: Groundnut Virus Diseases in Africa: Summary and Recommendations of the Sixth Meeting of the International Working Group, Agricultural Research Council, Plant Protection Research Institute, Pretoria, South Africa. D. V. R. Reddy, P. Delfosse, J. M. Lenne', and P. Subrahmanyam, eds. International Crops Research Institute for the Semi-Arid Tropics, Andhra Pradesh, India; and Belgian Administration for Development Cooperation, Belgium.
51. Naidu, R. A., Robinson, D. J., and Kimmins, F. M. 1998. Detection of each of the causal agents of groundnut rosette disease in plants and vector aphids by RT-PCR. J. Virol. Methods 76:9-18.
52. Nigam, S. N., and Bock, K. R. 1990. Inheritance of resistance to groundnut rosette virus in groundnut (*Arachis hypogaea* L.). Ann. Appl. Biol. 117:553-560.
53. Nutman, F. J., Roberts, F. M., and Williamson, J. G. 1964. Studies on varietal resistance in the groundnut (*Arachis hypogaea* L.) to rosette disease. Rhod. J. Agric. Res. 2:63-77.
54. Okusanya, B. A. M., and Watson, M. A. 1966. Host range and some properties of groundnut rosette virus. Ann. Appl. Biol. 58:377-387.
55. Olorunju, P. E., Kuhn, C. W., Demski, J. W., Misari, S. M., and Ansa, O. A. 1991. Disease reactions and yield performance of peanut genotypes grown under groundnut rosette and rosette-free field environments. Plant Dis. 75:1269-1273.
56. Padgham, D. E., Kimmins, F. M., and Ranga Rao, G. V. 1990. Resistance in groundnut (*Arachis hypogaea* L.) to *Aphis craccivora* Koch. Ann. Appl. Biol. 117:285-294.
57. Rajeshwari, R., Murant, A. F., and Massalski, P. R. 1987. Use of monoclonal antibody to potato leaf roll virus for detecting groundnut rosette assistor virus by ELISA. Ann. Appl. Biol. 111:353-358.
58. Reddy, D. V. R. 1991. Groundnut viruses and virus diseases: Distribution, identification and control. Rev. Plant Pathol. 70:665-678.
59. Reddy, D. V. R., Murant, A. F., Duncan, G. H., Ansa, O. A., Demski, J. W., and Kuhn, C. W. 1985. Viruses associated with chlorotic rosette and green rosette diseases of groundnut in Nigeria. Ann. Appl. Biol. 107:57-64.
60. Robinson, D. J., Ryabov, E. V., Raj, S. K., Roberts, I. M., and Taliansky, M. E. 1999. Satellite RNA is essential for encapsidation of groundnut rosette umbravirus RNA by groundnut rosette assistor luteovirus coat protein. Virology 254:104-114.
61. Roossinck, M. J. 1977. Mechanisms of plant virus evolution. Annu. Rev. Phytopathol. 35:191-209.
62. Sauger, L., and Catherinet, M. 1954. La rosette chlorotique de l'arachide et les lignées selectionnées. Agron. Trop. 9:28-36.
63. Sauger, L., and Catherinet, M. 1954. Nouvelles observations sur la rosette chlorotique de l'arachide et les lignées selectionnées. Bull. Agron. Ministere France Outremer 11:204-216.
64. Sauger, L., Catherinet, M., and Durand, Y. 1954. Contribution a l'etude de la rosette chlorotique de l'arachide. Bull. Agron. Ministere France Outremer 13:163-180.
65. Savage, G. P., and Keenan, J. I. 1994. The composition and nutritive value of groundnut kernels. Pages 173-213 in: The Groundnut Crop: A Scientific Basis for Improvement. J. Smartt, ed. Chapman & Hall, London.
66. Scott, K. P., Farmer, M.-J., Robinson, D. J., Torrance, L., and Murant, A. F. 1996. Comparison of the coat protein of groundnut rosette assistor virus with those of other luteoviruses. Ann. Appl. Biol. 128:77-83.
67. Smartt, J. 1961. The diseases of groundnut in Northern Rhodesia. Empire J. Exp. Agric. 29:79-87.
68. Smartt, J. 1994. The groundnut in farming systems and the rural economy - a global view. Pages 664-699 in: The Groundnut Crop: A Scientific Basis for Improvement. J. Smartt, ed. Chapman & Hall, London.
69. Soyer, D. 1939. La 'rosette' de l'arachide. Recherches sur les vecteurs possibles de la maladie. INEAC Sér. Sci. No. 21.
70. Storey, H. H., and Bottomley, B. A. 1928. The rosette disease of peanuts (*Arachis hypogaea* L.). Ann. Appl. Biol. 15:26-45.
71. Storey, H. H., and Ryland, A. K. 1955. Transmission of groundnut rosette virus. Ann. Appl. Biol. 43:423-432.
72. Storey, H. H., and Ryland, A. K. 1957. Viruses causing rosette and other diseases in groundnuts. Ann. Appl. Biol. 45:319-326.
73. Subrahmanyam, P., Hildebrand, G. L., Naidu, R. A., Reddy, L. J., and Singh, A. K. 1998. Sources of resistance to groundnut rosette disease in global groundnut germplasm. Ann. Appl. Biol. 132:473-485.
74. Taliansky, M. E., and Robinson, D. J. 1997. Down-regulation of groundnut rosette virus replication by a variant satellite RNA. Virology 230:228-235.
75. Taliansky, M. E., and Robinson, D. J. 1997. Trans-acting untranslated elements of groundnut rosette virus satellite RNA are involved in symptom production. J. Gen. Virol. 78:1277-1285.
76. Taliansky, M. E., Robinson, D. J., and Murant, A. F. 1996. Complete nucleotide sequence and organization of the RNA genome of groundnut rosette umbravirus. J. Gen. Virol. 77:2335-2345.
77. Taliansky, M. E., Ryabov, E. V., and Robinson, D. J. 1998. Two distinct mechanisms of transgenic resistance mediated by groundnut rosette virus satellite RNA sequences. Mol. Plant-Microbe Interact. 11:367-374.
78. Thresh, J. M. 1983. The long-range dispersal of plant viruses by arthropod vectors. Philos. Trans. R. Soc. London Ser. B. 302:497-528.
79. Tjallingii, W. F. 1985. Membrane potentials as an indication for plant cell penetration by

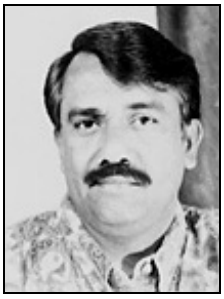
aphid stylets. Entomol. Exp. Appl. 38:187-193.
80. van der Merwe, P. J. A., and Subrahmanyam, P. 1997. Screening of rosette-resistant short-duration groundnut breeding lines for yield and other characteristics. Intern. Arachis Newsl. 17:14-15.
81. Watson, M. A., and Okusanya, B. A. M. 1967. Studies on the transmission of groundnut rosette virus by *Aphis craccivora* Koch. Ann. Appl. Biol. 60:199-208.
82. Wilson, D. M. 1995. Management of mycotoxins in peanut. Pages 87-92 in: Peanut Health Management. H. A. Melouk and F. M. Shokes, eds. American Phytopathological Society, St. Paul, MN.
83. Wilson, D. M., and Payne, G. A. 1994. Factors affecting *Aspergillus flavus* group infection and Aflatoxin contamination of crops. Pages 309-325 in: The Toxicology of Aflatoxins: Human Health, Veterinary, and Agricultural Significance. D. L. Eaton and J. D. Groopman, eds. Academic Press, San Diego, CA.
84. Yayock, J. Y., Rossel, H. W., and Harkness, C. 1976. A review of the 1975 groundnut rosette epidemic in Nigeria. Samaru Conf. Pap. 9. Institute of Agricultural Research, Ahmadu Bello University, Zaria, Nigeria.
85. Zimmermann, G. 1907. Über eine Krankheit der Erdnüsse (*Arachis hypogaea*). Der Pflanzer 3:129-133.



R. A. Naidu

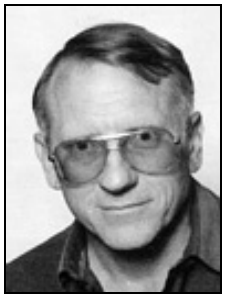
F. M. Kimmins

C. M. Deom



P. Subrahmanyam

A. J. Chiyembekeza



P. J. A. van der Merwe

Dr. Naidu is a consultant with the DFID–Crop Protection Programme, UK, based at Chitedze Agricultural Research Station, Lilongwe, Malawi. He earned his B.Sc. (1975) in biology, M.Sc. (1977) and Ph.D. (1984) in botany with plant virology specialization from Sri Venkateswara University, Tirupati, India. He lectured in botany (1985 to 1986) at Sri Krishnadevaraya University, Anantapur, India, then pursued postdoctoral research on sorghum and groundnut viruses at ICRISAT (1987 to 1988) and molecular biology of peanut stunt virus at the University of Kentucky (1989 to 1991). Subsequently, he worked as scientist and senior scientist at ICRISAT (1992 to 1998), focusing on molecular characterization and diagnosis of chickpea and groundnut viruses in India and sub-Saharan Africa. He was a visiting scientist at Scottish Crop Research Institute and Natural Resources Institute in the UK, the University of Florida Citrus Research and Education Center, and the University of Georgia. His current research focuses on groundnut viruses in sub-Saharan Africa, with major emphasis on rosette disease epidemiology and management.

Dr. Kimmins is an entomologist at the Natural Resources Institute of the University of Greenwich. She has a B.Sc. and Ph.D. from the University of London. Dr. Kimmins was a postdoctoral associate at the Wageningen Agricultural University prior to joining the Natural Resources Institute in 1985. Her research has focused on plant resistance mechanisms to homopteran pests of tropical crops, especially aphids and leafhoppers, and more recently on the epidemiology, biology, and management of insect-vectored virus diseases of rice and groundnut. During her research she has worked in the Philippines, India, Nigeria, Tanzania, and Malawi.

Dr. Deom is an associate professor with the Department of Plant Pathology, University of Georgia, Athens. He received his B.A. and M.A. in microbiology from Southern Illinois University, Carbondale, and his Ph.D. in microbiology from St. Louis University School of Medicine, St. Louis. Dr. Deom was a postdoctoral associate at Washington University prior to joining the University of Georgia. His research focuses on plant virus–host interactions, in addition to commitments to undergraduate and graduate education.

Dr. Subrahmanyam is a principal scientist (pathology) and ICRISAT country representative based at Chitedze Agricultural Research Station, Lilongwe. He received his Ph.D. in botany in 1975 from Sri Venkateswara University, Tirupati, India. Since joining ICRISAT in 1976, he has worked in India, West Africa, and currently in southern and eastern Africa. His major areas of research include host-plant resistance and integrated management of groundnut diseases with emphasis on foliar diseases and groundnut rosette. He was a visiting scientist at Texas A&M University Plant Disease Research Station at Yoakum, Texas, between 1984 and 1985. He is an editor of the Compendium of Peanut Diseases published by the American Phytopathological Society.

Dr. Chiyembekeza is a groundnut breeder with the Department of Agricultural Research and Technical Services, Ministry of Agriculture and Irrigation, Malawi, based at the Chitedze Agricultural Research Station, Lilongwe. He earned his B.S. in agriculture from the University of Malawi in 1978 and his M.Sc. and Ph.D. in agronomy with a minor in plant pathology at the University of Florida, Gainesville, in 1986 and 1992, respectively. He is currently the national coordinator for groundnut research in Malawi and carrying out on-farm multilocation trials and seed multiplication of improved groundnut varieties. His research is funded by GTZ/BMZ (Germany), the European Union, and USAID-Peanut CRSP (USA).

Dr. van der Merwe is a principal scientist (breeding) and manager of the SADC/ICRISAT groundnut project funded by GTZ/BMZ (Germany) based at Chitedze Agricultural Research Station, Lilongwe. He received his B.Sc. in 1965, his M.Sc. in 1968, and his D.Sc. in 1983 in agricultural genetics from the University of Pretoria, South Africa. He worked as an assistant director with the ARC-Grain Crops Institute in South Africa till 1996, focusing on control of major diseases of groundnut and sunflower through host-plant resistance. During this time, he was largely responsible for developing groundnut varieties resistant to pod rot and curbing the black pod rot epidemic of groundnuts. His current research focuses on developing groundnut varieties with multiple resistance to rosette, rust, and early and late leaf spot diseases, multilocation testing of improved groundnut varieties in collaboration with the national program scientists, and dissemination of groundnut production technologies in southern and eastern Africa.