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Epidemiology of groundnut rosette virus disease: current status and future research needs[@]

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Summary

Rosette is the most destructive virus disease of groundnut in sub-Saharan Africa. It is caused by a complex of three agents, namely groundnut rosette assistor virus, groundnut rosette virus and its satellite RNA. The disease appears to be indigenous to Africa as it has not been recorded elsewhere. Thus rosette represents a new-encounter situation as the disease is thought to have spread to the introduced groundnut from indigenous host plants. Rosette has been known since 1907 and much information has been obtained on the main features of the disease, *viz.* its biology, transmission, viral aetiology and diagnosis, and the impact of chemical control of the aphid vector, cultural practices and virus-resistant varieties on disease management. However, there are still many gaps in the available knowledge, especially the reasons for the large and unpredictable fluctuations in the incidence and severity of rosette disease throughout sub-Saharan Africa. Three unresolved issues of particular importance concern the nature of the primary source(s) of inoculum, the means of survival of virus and vector during unfavourable periods, and the distances over which the aphid vector can disperse and disseminate virus. Now that the aetiology of the disease is understood and diagnostic tools have been developed, the time is opportune for new initiatives in understanding the ecology and epidemiology of rosette. Substantial progress can be made by developing a co-ordinated multi-disciplinary research programme and making full use of the latest techniques, approaches and experience gained elsewhere with other insect-borne viruses. This information would help to explain the sporadic disease epidemics that cause serious crop losses and sometimes total crop failure, and would also facilitate the development of disease forecasting methods and sustainable integrated disease management strategies.

Key words: Aphid vector, *Aphis craccivora*, biotypes, diagnosis, epidemiology, groundnut rosette disease, groundnut rosette assistor virus, groundnut rosette virus, satellite RNA, integrated disease management, RT-PCR, TAS-ELISA, transmission, variability

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Introduction

Rosette disease of groundnut (*Arachis hypogaea* L.) was first described from Tanganyika (now Tanzania) (Zimmermann, 1907) and it was subsequently found to be widely distributed throughout west, east and southern Africa and offshore islands, including Madagascar. Although studied at different times and in several Anglophone and Francophone countries of Africa including Nigeria, Senegal, Burkina Faso, Côte d'Ivoire, South Africa, Malawi, Tanzania and Uganda, rosette disease has been, and continues to be, responsible for substantial and sometimes devastating losses to groundnut production wherever it is grown in sub-Saharan Africa (SSA). Rosette epidemics are sporadic and unpredictable (Table 1), but when they occur, yield losses are severe and the impact on the rural economy is profound. For instance, the 1975 epidemic in northern Nigeria destroyed an estimated 0.7 million ha of groundnut, with estimated losses of *c.* US $250 million (Yayock, Rossel & Harkness, 1976; Yayock, 1977). The 1995 epidemic in the Eastern Province of Zambia affected *c.* 43 000 ha, causing losses of *c.* US $4.89 million (Anon., 1996). In much of semi-arid SSA, smallholder farmers grow groundnut for both subsistence and cash. When a disaster such as this strikes, they lose a valuable source of dietary protein and much of their income. Additionally, they face severe shortage of seed for the next year's planting. Due to the 1994/1995 rosette epidemic in Malawi, the area under groundnut fell by 23% from 89 000 ha in 1994/95 to 69 722 ha in 1995/96 (Anon., 1996). Such recurring epidemics of rosette disease have hampered the production and expansion of the groundnut crop and substantially changed cropping patterns in several countries in SSA.

Table 1. *Documented reports of groundnut rosette disease epidemics in sub-Saharan Africa*

Location	Year	Reference
Lindi region, Tanzania	1907	Zimmerman, 1907
Transvaal, S. Africa	1922-23	Storey and Bottomley, 1928
Lindi region, Tanzania	1924	Kirby, 1924
Senegal	1925	Réal, 1953
Gambia	1927	Réal, 1953
Zaire (Belgian Congo)	1939	Réal, 1953
Mokwa, Nigeria	1949-54	Harkness, 1977
Kontagora/Bida, Nigeria	1950's	Harkness, 1977
Senegal	1952	Catherinet *et al.*, 1955
Nigeria	1975	Yayock *et al.*, 1976
Niger	1975, 1987	Subrahmanyam *et al.*, 1988 Oumarou *et al.*, 1990
Nigeria	1983, '85, '88	Misari *et al.*, 1988*b*
Malawi, Zambia	1996	Subrahmanyam, unpublished data

Plant virus epidemics are multi-component systems resulting from complex interactions between the virus(es), vector(s) and host plant(s), influenced by their shared physical environment (Thresh, 1983*a*). Therefore, a full understanding of the epidemiology of rosette disease and the environmental factors (both biotic and abiotic) that influence the onset and progress of disease outbreaks is critical for the development of sustainable management strategies. This review summarises the available information on groundnut rosette and highlights the main unresolved issues concerning the disease and considers how they can be addressed by employing the latest techniques and approaches that have been used in understanding the epidemiology of other insect-borne viruses.

Rosette: a disease of complex aetiology

Symptoms

Rosette disease occurs as two predominant symptom forms: chlorotic rosette and green rosette (Figs 1*a* & 1*b*; Storey & Bottomley, 1928; Hayes, 1932; Réal, 1955; Gibbons, 1977; Murant & Kumar, 1990). Chlorotic rosette is widely distributed, whereas green rosette has so far been reported only from certain countries in West Africa and Uganda, and more recently in northern Malawi, Angola and Swaziland (Subrahmanyam & Mamba, 1993; Subrahmanyam &

Fig. 1. Rosette disease symptoms in groundnut, (*a*) chlorotic rosette and (*b*) green rosette. Note severe stunting of infected plants in both forms of the disease.

Chiyembekeza, 1995). In the latter countries, both chlorotic and green rosette sometimes occur in the same fields. A third form called mosaic rosette has been reported from East Africa (Storey & Ryland, 1957). Plants affected by either green or chlorotic rosette are severely stunted and of bushy appearance due to shortened internodes and reduced leaf size. Leaves of green rosette-affected plants appear dark green in colour when compared to unaffected plants. Some leaves show a light green and dark green mosaic. Leaves of chlorotic rosette-affected plants are curled and puckered and show a bright chlorosis, usually with a few green islands. Either the whole plant, or only some branches or parts of branches may show rosette symptoms depending on the stage of infection . Early infection with green or chlorotic rosette results in severe or total yield loss, whereas late infection causes less drastic decreases in the number and size of pods. For example, in semi-erect, long-maturing varieties of groundnut, rosette disease causes almost complete loss in pod yield if plants are infected within the first 6 wk of sowing and about 50% loss from infection 6–12 wk after sowing. The effects on yield after later infection seem negligible (de Berchoux, 1960). Infected groundnut leaves may also show symptoms other than the typical chlorotic or green rosette suggesting even wider variability in visible symptoms (Naidu *et al.*, 1997).

Causal agents

Groundnut rosette disease is caused by a complex of three agents, *viz.* groundnut rosette virus (GRV), satellite RNA (sat RNA) and groundnut rosette assistor virus (GRAV). GRAV is a member of the genus *Luteovirus* (Casper *et al.*, 1983; Murant, 1989), while GRV belongs to the genus *Umbravirus* (Murant, Robinson & Gibbs, 1995; Taliansky, Robinson & Murant, 1996). GRV is mechanically transmissible and replicates independently in plants, whereas the sat RNA depends entirely on GRV for its replication (Murant, Rajeshwari, Robinson & Raschke, 1988; Blok, Ziegler, Robinson & Murant, 1994). Either GRAV or GRV alone cause no obvious symptoms or only a mild transient mottle in groundnut. The sat RNA is largely responsible for the different types of rosette symptom. Variants of sat RNA were shown to cause the chlorotic and green forms of the disease, and other variants induce mild or no symptoms in groundnut (Murant *et al.*, 1988; Murant & Kumar, 1990). In nature, individual plants may contain a mixed population of sat RNA variants. Mosaic rosette is probably an example of such a dual infection with both chlorotic and mild forms of the sat RNA. Other atypical symptoms are caused by mixtures of sat RNA variants. However, there may be variation among isolates of GRV itself or an influence of other viruses.

The inter-dependence and interactions among the three agents of rosette disease are complex. GRAV replicates autonomously in plants and is transmitted only by aphids, mainly *Aphis craccivora* Koch (Homoptera: Aphididae). By contrast, sat RNA depends on GRV for replication and GRV depends on sat RNA for aphid transmission. Both, in turn, depend on GRAV for their packaging in GRAV coat protein and subsequent transmission by an aphid vector (Murant, 1990). In nature, all three agents must occur together for rosette disease to be transmitted successfully by the aphid vector. Thus GRV and its sat RNA are dependent for their survival not only on each other but also on GRAV. This strict dependence probably explains why the sat RNA has been found in all naturally occurring GRV isolates.

Diagnosis

The recently developed ability to detect each of the three agents of rosette disease both in plants and in aphid vectors will be crucial in gaining an improved understanding of the ecology and epidemiology of the disease, to develop infectivity-based forecasting systems, and to identify sources of resistance to the individual agents of the disease in groundnut. Until recently, rosette disease was identified in groundnut solely on the basis of visual symptoms,

but recent advances in diagnosis have been achieved through the development of accurate serological detection and nucleic acid hybridisation assays. GRAV can be detected by serological tests such as enzyme-linked immunosorbent assay (ELISA) (Casper *et al.*, 1983; Rajeshwari, Murant & Massalski, 1987; Scott *et al.*, 1996). Since GRV and its sat RNA lack coat protein, they cannot be detected by such tests but GRV can be detected by manual inoculation to suitable test plants (*Nicotiana benthamiana* Domin. or *Chenopodium amaranticolor* Coste and Reyn.). Both agents can be detected in dot-blot hybridisation assays by using nucleic acid probes specific to either GRV or sat RNA (Blok *et al.*, 1995), or by reverse transcription-polymerase chain reaction (RT-PCR) with specific primers (R A Naidu, D J Robinson and F M Kimmins, unpublished data). Detection and diagnosis of rosette disease based on symptoms in herbaceous plants and aphid transmission procedures are time-consuming and labour intensive, whereas a combination of ELISA and dot-blot hybridisation or RT-PCR assays offers several advantages. These include sensitivity, speed (the entire test takes only 2 to 3 days) and the ability to assay many samples concurrently.

Virus-vector-host plant interactions

Transmission

Rosette disease is transmitted efficiently in nature by the aphid, *Aphis craccivora* in a persistent and circulative manner (Storey & Bottomley, 1928; Storey & Ryland, 1955; Hull, 1964). Another aphid species, *Aphis gossypii* Glov., was reported to be an inefficient vector (Adams, 1967), but this has not been confirmed. Okusanya & Watson (1966) first reported that aphids failed to transmit groundnut rosette disease from plants that had been infected mechanically by sap inoculation. This is now known to be because GRAV is not mechanically transmissible and only GRV and its sat RNA establish and multiply in manually inoculated plants (Murant *et al.*, 1988). It was subsequently suggested that the ability of GRAV to assist aphid transmission of GRV and its sat RNA results from the packaging of GRV RNA and sat RNA in the coat protein of GRAV (Murant, 1990). Aphids can acquire and transmit GRAV irrespective of the presence of GRV and its sat RNA. Thus rosette is of particular interest in that the transmission characteristics of the disease are influenced by GRAV, but not by either GRV or sat RNA as they are encapsidated within the GRAV particles. Through the ability to utilize the coat protein of GRAV, GRV and its sat RNA gain epidemiologically by acquiring a persistent relationship with the aphid vector. Like other persistently transmitted viruses, GRAV is retained by the vector for periods of up to 14 days and possibly for life (Storey & Ryland, 1955; Watson & Okusanya, 1967; Misari *et al.*, 1988*a*). The virus is not lost during ecdysis, and all stages of the insect may acquire and transmit the virus. With these transmission characteristics, spatial spread of a persistently-transmitted disease such as rosette can be expected to be much more general and widespread than the more localised distribution of diseases caused by non-persistently or semi-persistently transmitted viruses (Duffus, 1973).

Two detailed studies have determined rosette disease transmission efficiencies of different clones of *A. craccivora* collected from groundnut. Dubern (1980), using chlorotic rosette from Côte d'Ivoire and virus- and vector-susceptible genotypes of groundnut reported minimum acquisition access and inoculation access periods of 4.5 h and 3 min, respectively. The latent period was 18 h and 2 h following acquisition feeding periods of 4.5 h and 24 h, respectively. Misari *et al.* (1988*a*) reported minimum acquisition feeding periods of 4 h and 8 h, respectively, for chlorotic and green rosette isolates from Nigeria. The minimum inoculation access period, however, was less than 10 min for both forms of rosette. The median latent

periods were 26.4 h and 38.4 h, respectively, for chlorotic and green rosette. These results suggest that chlorotic rosette is transmitted more efficiently than green rosette, but no explanation has been offered for this.

There are many reasons for caution when assessing the implications of the different transmission efficiencies of chlorotic and green rosette by aphids in the epidemiology of the disease and in extrapolating the efficiencies to the field situation. The first is that in all previous vector studies, successful transmission has been assessed by the appearance of rosette symptoms in the inoculated plants, so that the data strictly refer only to transmission of GRV *and* its sat RNA (since GRAV causes no obvious symptoms). During penetration of the host plant by the vector, it is possible that the brief punctures made into mesophyll cells (Tjallingii, 1985) may suffice to inoculate GRAV, GRV and its sat RNA. If so, only the last two will replicate and multiply since GRAV, like other luteoviruses, multiplies only in phloem parenchyma cells (Francki, Milne & Hatta, 1985). Rosette symptoms may appear following the establishment of GRV and its sat RNA in the absence of GRAV, but such plants will not serve as sources of inoculum for subsequent spread by aphids. Although it seems likely that the minimum acquisition access feeding and latent periods for GRAV alone are similar to those of the complex (since GRV RNA and sat RNA are within GRAV coat protein), the minimum inoculation access period (IAP) for GRAV may exceed that for GRV and its sat RNA because GRAV must be inoculated into the phloem. Thus long acquisition access feeding is essential to acquire GRAV, but the ability of the vector to inoculate all three agents may depend on the inoculation period. Recent studies indicated that some field samples from plants having typical green or chlorotic rosette symptoms did not contain GRAV, whereas some apparently symptomless plants contained GRAV (assayed by TAS-ELISA and RT-PCR) and similar results were obtained with plants inoculated by single viruliferous aphids under laboratory conditions (R A Naidu *et al.*, unpublished data). The laboratory results with chlorotic rosette indicated that following relatively short IAPs (< 120 min), during which only some aphids reached phloem tissues, 40% of the plants developed chlorotic rosette symptoms, but these plants did not contain GRAV. A similar experiment using an IAP of 48 h resulted in 95% of the plants showing clear rosette symptoms, of which only 50% were positive for GRAV. This indicated that the transmission efficiency of the three agents increases with an increase in IAP.

The above observations show clearly that, from source plants containing both GRV and GRAV, some plants inoculated by aphids become infected with GRV and sat RNA only. In the source plants, particles consisting of GRAV coat protein and either GRV RNA (and sat RNA) or GRAV RNA alone are available for acquisition by aphids, but sometimes only the former are transmitted in sufficient quantity to cause infection. In experiments the converse situation is also frequently observed, in which only particles with GRAV RNA are transmitted by aphids from doubly-infected source plants. However, because plants infected with GRAV alone are virtually symptomless, it is unclear how frequently they occur in nature. A separation of the two infective agents could result either from the aphids acquiring or transmitting only one kind of particle from the source plant or from their inoculating a sufficient dose of only one kind of particle to the recipient plant. Because both kinds of transmissible particle contain GRAV coat protein, acquisition feeding must presumably be on tissue in which GRAV is actively replicating. However, GRV and its sat RNA may not always occur in the same tissue together with GRAV, and this could explain the transmissions of GRAV alone. Aphids are thought to inoculate phloem-limited viruses immediately after sieve element penetration through a brief period of salivation in the sieve elements (Prado & Tjallingii, 1994), which is followed by phloem ingestion. On a susceptible host, ingestion may be sustained and uninterrupted for several hours. In this situation, a single aphid may not release enough virus particles containing GRAV RNA into the plant to initiate GRAV

replication. However, sufficient particles containing GRV RNA and its sat RNA may have been deposited in mesophyll tissue during exploratory probes to establish these agents. In order to determine the GRAV-vector relationships more precisely, the transmission characteristics of rosette disease are now being studied using diagnostic tools and a precise Electrical Penetration Graph (EPG) method (Prado & Tjallingii, 1994)

Whatever the mechanism involved, it is clear that expression of rosette symptoms does not necessarily indicate the presence of the aphid-transmissible GRAV in the plant. This has important implications for studying the factors involved in rosette disease spread and those contributing to rosette epidemics. Furthermore, detection of GRAV coat protein in aphids by TAS-ELISA provides no information about which of the infective RNA components they carry. Studies on disease spread must use the diagnostic tools available to determine which components of the pathosystem occur in both plants and insects and where they occur.

A second reason for caution when interpreting transmission efficiency data is that they are usually measures of competence made under laboratory conditions (Gold, 1979). The term, vector propensity (Irwin & Ruesink, 1986) has been proposed as a more accurate method of measuring vector importance because it takes into account both aphid movement and feeding behaviour, as assessed in conditions where the aphid can move and feed freely. It is considered to quantify the natural ability of a vector species or clone to inoculate a virus into a plant (Yuan & Ullman, 1996). The propensity of different clones of *A. craccivora* to transmit rosette disease is now being studied using a range of groundnut genotypes selected by ICRISAT at Chitedze Agricultural Research Station in Malawi and which differ in susceptibility to both the virus and to the aphid vector (F M Kimmins *et al.*, unpublished data).

Biotypes

The term biotype is a relatively non-specific term which has been applied to any population of aphids that differs from previously studied populations of that species (Eastop, 1973). Storey & Ryland (1955) reported that not all cultures of *A. craccivora* could transmit rosette disease. Nutman, Roberts & Williamson (1964) found that colonies of *A. craccivora* collected from beans (*Vicia faba* L. and *Phaseolus multiflorus* Lam.) did not transmit rosette, and rosette disease was transmitted only with aphid colonies collected from and then maintained on groundnut. It has also been shown that a Nigerian race of *A. craccivora* transmitted rosette isolates from Nigeria, Kenya, and Uganda, whereas races of *A. craccivora* from Kenya and Uganda transmitted East African isolates but not those from West Africa (Watson & Okusanya, 1967). Jones (1967) regarded the different abilities of these two races to transmit rosette as evidence of distinct biotypes. Body and leg measurements confirmed that the two biotypes differed, but overlapping distributions of the measurements made it impossible to distinguish field colonies by size alone. It is likely that this method has limited usefulness in identifying biotypes with different feeding preferences since it is difficult to understand why body size should be related to transmission efficiencies or host preferences unless stylet length and a consequent change in ability to locate the phloem are involved. Biochemical techniques for examining aphid genomes may prove to be more informative.

Very large areas of cowpea and beans are planted annually in some SSA countries and *A. craccivora* also colonises many other legume and non-legume hosts during the cropping and off-seasons (Adams, 1967). In general, aphids prefer to feed and reproduce faster on young than on mature leaves (Kennedy & Booth, 1951*a,b*), especially if they are not well adapted to feed on the host species. In Nigeria, cowpea germplasm exposed to *A. craccivora* from groundnut showed a different pattern of varietal responses from germplasm exposed to aphids reared on cowpea (Anon., 1994). Differences in host response among populations of *A. craccivora* were also observed in the Indian sub-continent (Hamid, Shah & Anwar, 1977;

Srikanth & Lakkundi, 1988). Recent studies have shown that groundnut genotypes that were previously reported as highly resistant to *A. craccivora* in India were heavily infested with the aphid in Nigeria (J A Wightman, unpublished data). Our recent observations in Malawi revealed large numbers of *A. craccivora* on cowpea in farmers' fields but not on neighbouring groundnut, either in young or mature crops, both during and outside the main growing season.

Virus host range

Since GRV is mechanically transmissible and GRAV is not, many host range studies relate to GRV and not to GRAV. GRV has been transmitted to a limited range of species in the Leguminosae, Chenopodiaceae or Solanaceae (Okusanya & Watson, 1966; Adams, 1967; Hull & Adams, 1968; Dubern, 1980; Reddy *et al.*, 1985). *Chenopodium amaranticolor* is a good local lesion assay host, whereas *Nicotiana benthamiana* is a systemic host suitable for virus propagation. Sat RNA multiplies in all hosts of GRV that have been tested.

Under experimental conditions, GRAV has been transmitted to: *Pisum sativum* L., *Stylosanthes gracilis* Taub., *S. hamata* (L.) Taub., *S. mucronata* Willd., *S. sundaica* Taub., *Trifolium incarnatum* L., *T. pratense* L., *Capsella bursa-pastoris* (L.) Medicus, *Gomphrena globosa* L., *Montia perfoliata* L. and *Spinacia oleracea* L. (Adams, 1967; Hull & Adams, 1968; Okusanya & Watson, 1966; Murant, 1989). Of these, only *C. bursa-pastoris* showed symptoms (generalised chlorosis) when infected.

Adams (1967) and Hull & Adams (1968) reported *T. incarnatum*, *S. mucronata* and *S. sundaica* as hosts for the rosette complex under experimental conditions. However, groundnut is the only naturally infected host yet known for the entire rosette disease complex. The crop was introduced into Africa from South America sometime during the 16th century by the Portuguese, yet the pathogens causing rosette appear to be indigenous to Africa, as they have not been recorded elsewhere (Reddy, 1991). Thus groundnut became an accidental host of rosette disease and is an example of the new-encounter phenomenon (Buddenhagen & de Ponti, 1984).

Aphid host range

The host range of *A. craccivora* has been studied extensively in efforts to explain the seasonal survival of aphid populations. *A. craccivora* infests many plant species in many families, but has a strong preference for members of the Leguminosae, which account for *c.* 47% of the known host species (Eastop, 1981). Millar (1994), in a catalogue of the aphids of SSA, listed 142 plant species in 23 families as hosts of *A. craccivora*, of which 83 are in the Leguminosae. Adams (1967) listed 31 leguminous and 9 non-leguminous species as hosts in Malawi. Groundnut was the main host in the growing season from February until the end of May, while during the mainly dry months from September to December *Dolichos malosanus* Bak., *Eriosema affine* De Wild, *Eriosema psoraleoides* (Lam.) G Don, *Eminia antennulifera* Baker (Taub.) and *Adenodolichos punctatus* (Micheli) Harms (all Leguminosae) were the most prominent hosts. *Euphorbia hirta* L. was found to be the main dry season host in Nigeria (Booker, 1963). In Tanzania, Evans (1954) found that *Vigna* spp., *Millettia* spp. and *Lonchocarpus* spp. were the major dry season hosts, but considered self-sown groundnut 'volunteers' to be important in the seasonal survival of aphids. In the Central Province of Malawi, Nutman *et al.* (1964) and Hildebrand, Bock & Nigam (1991) found no evidence that volunteer groundnuts play any role in vector and/or virus survival. It was suggested that aphids survive on a succession of dry season hosts, particularly shrub and tree species that are common in the groundnut growing regions of Africa and that produce flushes of new growth before the onset of the rains (Adams & Farrell, 1967; Bock, 1985).

Host plants that support aphid colonisation and multiplication may also be reservoir hosts for rosette disease. Because several hours are needed for virus acquisition, the source must also be a good host plant for the aphid. However, as successful inoculation can occur within minutes, plant species that are not preferred by the aphids may still become infected during brief penetrations, provided they are hosts for components of the rosette disease complex. Such plants may support replication only of GRV and its sat RNA and would not be sources for further transmission. Alternatively, all three components may replicate, but the plants may be poor sources for subsequent spread because of the need for extended acquisition feeding. Thus it is critical to understand the significance and importance of plants that are preferred hosts for the aphids in the ecology and epidemiology of rosette disease. It is also unclear whether *A. craccivora* found on different plant species are of different biotypes and whether all of them transmit rosette.

Factors that influence epidemics

Aphid development and population dynamics

Aphis craccivora is polyphagous and has a world-wide distribution (Anon., 1983). Only winged and wingless viviparous forms (female adults producing live offspring) have been described in the tropics, and these reproduce parthenogenetically throughout the year. As with other aphid species, *A. craccivora* possesses needle-like mouth parts which enable the aphid to pierce plant tissues and feed on plant sap, predominantly that of phloem sieve elements (Pollard, 1973). In this way, GRAV is inoculated directly into phloem sieve elements. Reports on the aphid's biology (Réal, 1953; Mayeux, 1984; Ansari, Singh & van Emden, 1987) indicate that it has a rapid development rate and a high reproductive potential compared with many other aphid species. It was also documented that *A. craccivora* populations develop faster and that increased numbers of winged adults (alates) are produced on rosette diseased groundnut plants than on healthy ones (Réal, 1955). These characteristics and the ready ability of populations to produce alates in response to crowding (tactile stimulation), quality of the host plant (age and decreasing nutritional quality), and climatic conditions (temperature, rainfall, humidity and sunshine) show their striking adaptability to exploit the habitats in which they thrive (Dixon, 1985). In the field, aphid populations undergo a succession of developmental stages. Mayeux (1984) distinguished an initial stage, initiated by small numbers of immigrant aphids; a subsequent explosive stage of massive population buildup; and a decreasing stage in which the population declines due to factors such as natural enemies and host plant quality.

Two basic studies on the biology of *A. craccivora* in Africa have been published, one relating to groundnut (Réal, 1953) and the other to cowpea (Ansari, Singh & van Emden, 1987). Population levels vary with season and depend on fecundity, mortality and migration rates which are controlled by abiotic (e.g. weather, soil fertility and moisture) and biotic (e.g. host plant, natural enemies, crowding) factors. *A. craccivora* thrives best between 24.0°C and 28.5°C and *c.* 65% relative humidity (Réal, 1953). Under these conditions, an adult can produce larvae for 6–7 days, at the rate of two to three per day or a total fertility of 13–14 descendants and each larval generation takes 6–8 days to develop into adults (Mayeux, 1984). Fecundity is low compared to data from India which range from 15 to 124 nymphs per adult depending on environment (Bakhetia and Sidhu, 1976).

The population dynamics of *A. craccivora* are also influenced by the spacing and age of groundnut plants (Farrell, 1971; 1976*b*). The number of incoming alate vectors was

significantly greater on young (late-sown) or open spaced than on old (early-sown) or close spaced plants. Consequently, the aphid density per infested plant was greater at wide spacings although the number of aphid-infested plants per unit area was less. Initially, the aphid numbers per unit area increased similarly in either open or closed spacing until the crop was 40–50 days old and afterwards the potential rate of increase of aphid populations, as well as size and density of the colonies, was lower at high plant densities. This was due to reduced aphid fecundity on the slower-growing and less nutritious plants in the dense crop.

Population levels are regulated by natural enemies and possibly rainfall. Natural enemies in the field include predators (Syrphidae, Coccinellidae), parasitoids (Braconidae, Encyrtidae), and pathogens (entomophagous fungi) (Evans, 1954; Booker, 1963; Farrell, 1976*b*). Farrell (1976*b*) concluded that the natural enemies reduced population densities only after the aphid populations started to decline toward the end of the season. Persistent rain dislodges aphids from plants onto the soil surface where they are exposed to predation (Wightman *et al.*, 1990), or they may be killed directly by soil particles splashed onto their colonies. Rainfall at frequent intervals increases the relative humidity around the plants, thereby promoting the development of entomophagous fungi. Nevertheless, accurate data on the effects of rain and predators on *A. craccivora* population dynamics are lacking. Such information would help to exploit natural enemies as control agents of *A. craccivora.*

Migration and climate

A. craccivora is an opportunistic coloniser that can thrive in a sequence of transient habitats due to its ability to reproduce rapidly and develop winged forms at relatively low population densities (Réal, 1953; Mayeux, 1984; Gutierrez, Nix, Havenstain & Moores, 1974; Thresh, 1983*c*). In temperate regions of south-east Australia, climate plays a major role in the development of *A. craccivora* populations (Campbell *et al.*, 1974) and the most favourable periods are early spring when pasture legumes germinate and late autumn before the plants mature and die. In south-east Australia, *A. craccivora* has been described as a super migrant because of its ability to migrate long distances between ephemeral habits (Gutierrez, Morgan & Havenstein, 1971; Gutierrez *et al.*, 1974). Whether long distance movement of *A. craccivora* occurs in Africa, however, is uncertain. Monitoring of seasonal flight activity (Eastop, 1957; Adams & Farrell, 1967; Davies, 1972; Farrell, 1971) by trapping alatae with water pan and suction traps has been done in a few places in SSA, although for only short periods. These studies showed that alatae occur throughout the year, but numbers peak during the early part of the wet season. Alatae can infest groundnut crops within a week of emergence and rosette symptoms appear about 2 wk later (Bock & Nigam, 1988). The origin of the alatae and rosette inoculum and the distance of migration cannot be deduced from such studies. However, data obtained from high-altitude sticky traps in Nigeria (Bottenberg *et al.*, 1997) and aircraft netting in Sudan (R C Rainey in Thresh, 1983*c*) suggest that alates of *A. craccivora* could be transported considerable distances by upper-level winds once they leave the surface boundary layer (Johnson, 1957). The prevailing wind direction changes seasonally in Africa according to the position of the Inter-tropical Convergence Zone. At the onset of the rainy season, rain-bearing winds come predominantly from the south (Griffiths, 1972; Grove, 1989; Udo, 1982) in the northern hemisphere and from the north in the southern hemisphere (Pike & Rimmington, 1965). Thus it is possible that aphids, originating from areas that receive rains and are planted earlier, are carried on these winds and are deposited along a zone of wind convergence in areas where the rains and planting have just started. Such depositions occurs in Africa with migratory pests such as the desert locust (*Schistocerca gregaria* Forsk.) and African army worm (*Spodoptera exempta* Wlk.) (Betts, 1975), but it is not clear from the available information whether it is also true for *A. craccivora* (Thresh, 1983*c*).

Field spread of the disease

Since groundnut rosette is transmitted by a mobile insect vector and is not seed-borne, spread in groundnut crops does not occur until inoculum is introduced from other sources. This is largely determined firstly, by the nature and distribution of the initial sources of infection from which spread occurs and secondly, by the extent to which the incoming aphids and their progeny survive and multiply on such plants. The initial sources might be different host plant species in areas where the cropping season is followed by a long dry season, or may be volunteer and/or overlapping groundnut crops in areas of bimodal annual rainfall. Primary infection is determined by the number, timing and infectivity of aphids moving into the groundnut crop. The infectivity of such aphids is in turn determined largely by the number, proximity and potency of source plants on which they survive during the off-season.

Once groundnut seedlings emerge, viruliferous winged immigrants alight within the field and establish randomly distributed primary inoculum sources. The mainly apterous progeny of the immigrant aphids then spread the disease within the field by crawling from plant to plant (Storey & Bottomley, 1928; Réal, 1953; Evans, 1954; Storey & Ryland, 1955; Davies, 1976; Farrell, 1971; 1976*a*). Previous studies have shown that greater numbers of these immigrants land on a young crop at wide spacing due to their differential alighting response or "chequer-board" effect (Kennedy, Booth & Kershaw, 1961). Under these conditions many primary infection sites would be created, and subsequent secondary spread would lead rapidly to almost total infection. In contrast, relatively few immigrants land in either early sown or close spaced crops due to groundcover which inhibits aphid landing response and thus less disease occurs in such a situation. In addition, plants tend to become decreasingly vulnerable to infection with age (Gibbons & Farrell, 1966; Bottenberg & Subrahmanyam, 1997) even though they come into more contact with neighbouring plants and present an increasing catchment area to the aphids. Hayes (1932) observed that a cover of grass or weeds between groundnut plants also reduced the number of diseased plants further suggesting that groundcover must have inhibited landing of alates. A crucial factor, therefore, in the development of epidemics is the number of viruliferous immigrant aphids that colonise the crop at an early stage when plants are most vulnerable to infection (Thresh, 1983*b,c*).

Rosette is regarded as a typical polycyclic disease because it spreads from sources of inoculum whose number increases during the growing season as progressively increasing numbers of plants become infectious (Thresh, 1983*b*). Sequential observations on disease progress have shown that secondary spread often occurs within the crop around these initial foci of infection to give clusters of infected plants. This leads to a change over time from an initial random to a clumped distribution of infection (Farrell, 1976*a*). Cluster size, their position within the crop, density of diseased plants and crop variety can provide information about the rate of disease spread, the underlying dynamics of virus transmission, vector dispersal, and the effects on those of cultural practices.

Disease progress curves based on symptom development are typically sigmoid and reach maxima 45–55 days after primary infection occurs (Sauger & Catherinet, 1954*a,b*; Catherinet, Sauger & Tardieu, 1955). However, detailed information is not available on the time taken for symptoms to develop in relation to environmental conditions, and the age and variety of groundnut. The reproduction, survival and behaviour of the aphid species are important factors in determining the amount and extent of secondary spread of the disease. The influence of resistant varieties (to the aphid and/or virus complex), the transmission efficiency of different biotypes, the number of diseased plants containing all three agents of rosette disease together with environmental factors including wind, rain, humidity, temperature and the effects of natural enemies on aphid build up and dispersal require further study in order to understand their relative contribution to the secondary spread of the disease within the crop.

Management of groundnut rosette disease epidemics

Chemical control

Insecticides have been used to control *A. craccivora* and to minimise or prevent spread of rosette disease in field trials and they have also featured in recommendations to farmers (Panse, 1937; Soyer, 1939; Evans, 1954; Davies & Kasule, 1964; Davies, 1975*a*,*b*). Davies (1975*b*) found that application of menazon at least four times during the growing season, with the first spray applied early and before symptoms appeared in the field, decreased rosette incidence. Ideally, accurate forecasts of disease incidence are required in order to decide on the need for and time of application of insecticide. Additionally, it is important that farmers who have access to and can afford pesticides are taught to monitor vector populations, especially at the early stages of crop growth (i.e. up to 12 wk after sowing), so that appropriate insecticides are applied *only* when required. For these farmers, integrating chemical sprays with other disease management strategies should be recommended so that the development of insecticide-resistant biotypes is minimised and the impact of natural enemies in controlling aphid populations is unaffected. Insecticides, however, are unlikely to be a realistic control measure for the mainly smallholder farmers in SSA and considering the toxicity and potentially detrimental effect of insecticides on the environment, other low-input integrated methods of aphid and disease management such as improved cultural practices and resistant varieties are preferable.

Cropping practices

Information on the control of rosette disease by cultural practices has been obtained in different parts of SSA (Guillemin, 1952; Jameson & Thomas, 1954; Sauger, Catherinet & Durand, 1954; Smartt, 1961; Booker, 1963; A'Brook, 1964; Davies, 1976; Farrell, 1976*a*,*b*). Results have shown consistently that early sowing and maintaining uniform dense stands of groundnut greatly reduce the incidence of rosette disease. Early-sown crops largely escape infection and the greater virus incidence in later-sown crops is probably a function of the timing of vector flights and preference for the young crop. It is known that alate aphids of several species often alight preferentially on plants at wide spacing (A'Brook, 1968; Farrell, 1976*b*; Irwin & Kampmeier, 1989), leading to higher rates of spread in such crops. Moreover, a low plant density also means that a greater proportion of the total stand will be infected by a similar number of infective immigrants entering the field, thus increasing the ratio of infected to uninfected plants. In contrast, planting at high density has a dilution effect and may in part compensate for the losses due to a smaller proportion of infected plants.

Management by early planting and dense spacing continues to be satisfactory in the few parts of SSA where large-scale commercial farming is practised. However, widespread adoption of such practices by the small-holder farmers in SSA is seldom feasible because of the preferred sowing sequence of crops (many farmers sow cereal crops first), labour constraints and costs, and/or insufficient seed to allow even moderately dense populations. Farmers often delay weeding in groundnut fields because of their preoccupation with other food and cash crops. Although the impact of late weed control in providing camouflage and decreasing rosette was known (Hayes, 1932), it is hazardous and not feasible because of the likelihood of detrimental effects on plant growth and yield. Furthermore, early planting, in many areas, necessitates harvesting the crop during wet weather, causing problems of drying and predisposition to moulds.

In many parts of SSA, groundnut is grown as a mixed/inter crop with cereals such as maize, sorghum, finger millet or legumes e.g. beans, cowpea and soybean. Intercropping can be

expected to have marked effects on colonisation by insect vectors and on their movement and behaviour within crops. There have been few studies of these effects on the spread of rosette, but Farrell's (1976*c*) results suggested that intersowing with beans reduced the incidence of the disease. However, this reduction was less than with early-sown and high-density plantings of groundnut in monoculture and additional studies are required on the effectiveness, feasibility of and acceptability of this approach to rosette disease management.

Breeding for host plant resistance

Resistance to rosette was first found in groundnut germplasm originating from Burkina Faso and Côte d'Ivoire (Sauger & Catharinet, 1954*a,b*; de Berchoux, 1960) and material from this region of West Africa has been the source of resistance in all the rosette-resistant cultivars developed since (Dhéry & Gillier, 1971; Gillier, 1978). Resistance in these genotypes is determined by two independent recessive genes (de Berchoux, 1960; Nigam & Bock, 1990; Olorunju *et al.*, 1992) and is effective against both chlorotic (de Berchoux, 1960) and green rosette (Harkness, 1977). The resistance appears to be against GRV and *not* GRAV (Bock, Murant & Rajeshwari, 1990; Olorunju *et al.*, 1991). Resistance to GRV also confers resistance to its sat RNA so that these genotypes do not express rosette symptoms. However, the resistance to GRV is not immunity and seems to be overcome under high inoculum pressure and in adverse environmental conditions (Bock *et al.*,1990). As mentioned earlier, GRAV alone and sat RNA-free isolates of GRV cause either symptomless infection or a transient mottle. Moreover, some of the sat RNA variants induce only mild symptoms in groundnut. Hence plants that show no symptoms may be infected by one or more agents of the disease complex. It is therefore important to use appropriate diagnostic tools in breeding programmes to test the resistant materials in order to determine against which of the three disease agents the resistance is directed.

Unfortunately, the genotypes released so far (including RMP 12, RMP 91, RG 1 etc.) represent only a narrow genetic base and are late-maturing Virginia types and so are not suitable for the many locations in SSA that have a short rainy season. In recent years, efforts to transfer rosette resistance to short-duration Spanish types with agronomically desirable characteristics have been successful (Reddy & Subrahmanyam, 1997). Consequently, several advanced groundnut breeding lines with high levels of resistance to rosette disease have been identified and they have the potential to be released as cultivars having the pod yield, pod and seed qualities and other agronomic characteristics that are desired (Chiyembekeza, Subrahmanyam & Hildebrand, 1997; van der Merwe & Subrahmanyam, 1997).

All rosette-resistant germplasm lines and genotypes so far identified are susceptible to GRAV (Olorunju *et al.*, 1991; Subrahmanyam *et al.*, 1998). Although GRAV alone causes no visible symptoms, it does appear to interact synergistically with the other two agents in rosette disease development because, in a mixed infection, stunting and yield reduction are greater than with single infection with GRV and its sat RNA (Ansa *et al.*, 1990). However, this needs to be substantiated further. New sources of resistance to GRAV in some wild *Arachis* accessions or species have been identified (Murant, Kumar & Robinson, 1991; R A Naidu *et al.*, unpublished data). These sources of resistance to GRAV could be exploited by incorporating them into cultivated groundnut by conventional breeding (if suitably compatible) or through biotechnological approaches.

Breeding for vector resistance

Evans (1954) first detected resistance to *A. craccivora* in the groundnut cultivar Asiriya Mwitunde from the northwest of Tanzania. Although primary spread of disease occurred into the crop, subsequent spread by immigrant alates or their progeny was restricted. Padgham,

Kimmins & Ranga Rao (1990) found such resistance in the groundnut genotype EC 36892, in which colony development was restricted. In field trials, this genotype showed a significantly reduced incidence of rosette disease (K R Bock and P Subrahmanyam, unpublished data). Under greenhouse conditions, wild *Arachis* species (*A. chacoense* Krap. et Greg. nom. nud., *A. villosa* Benth., *A. correntina* (Burkart) Krap. et Greg. nom. nud., and *A. glabrata* Benth.) exhibited high levels of resistance to *A. craccivora* (Amin, 1985). Such sources of resistance could be exploited further in breeding programmes.

Strategic Research Needs

An understanding of the most important processes, and how they combine to determine the dynamics of rosette epidemics is essential for the successful development of sustainable disease control strategies suitable for different agro-ecological zones.

Detection of rosette disease agents in aphid vector

The availability of sensitive diagnostic tools for detecting all three rosette disease agents in the aphid vectors, and knowledge of their molecular characteristics (Blok *et al.*, 1994; Scott *et al.*, 1996; Taliansky, Robinson & Murant, 1996) will facilitate more extensive and detailed studies of the ecology and epidemiology of rosette disease. These modern techniques have the advantage over aphid transmission tests in that aphids can be sampled in different regions and tested centrally within 2 days. Amplified triple antibody sandwich (TAS)-ELISA (Smith, Stevens & Hallsworth, 1991) can detect particles containing GRAV coat protein in individual aphids, and an RT-PCR-based assay (Singh, Kurz, Boiteau & Bernard, 1995) can detect GRAV RNA, GRV RNA and sat RNA (R A Naidu, D J Robinson and F M Kimmins, unpublished data). An advantage of RT-PCR is that it can detect each of the three agents concurrently in a single aphid or plant. Results from such research would facilitate estimates of the proportion of immigrant aphids carrying the rosette disease agents. It may also be possible to correlate presence of these agents in the aphid with the ability to transmit the disease and to distinguish strains/variants carried by aphids; this would facilitate the development of disease forecasting systems.

Symptom and virus variability

Recent observations show wide variability in visible symptoms of rosette disease. There is a need to document the whole range of symptoms encountered in SSA and to develop improved methods of rosette disease identification and monitoring. Ideally, any plants showing atypical or suspect rosette symptoms should be tested for the three agents of rosette and also for other viruses to provide unequivocal results.

The occurrence and significance of diversity among the three agents of rosette disease has not been studied. Tobacco yellow vein virus (TYVV), the putative umbravirus component of the tobacco yellow vein disease complex, multiplies and induces mild symptoms (leaves showed small pale green rings; chlorotic areas were frequent along the mid-rib or main lateral veins; there was no leaf distortion and infected plants were only slightly stunted) in groundnut when mechanically inoculated (Adams & Hull, 1972). Moreover, GRAV aided the transmission of GRV and of TYVV by *A. craccivora*, both separately and together. There are striking similarities between the sat RNAs associated with the groundnut rosette disease complex and pea enation mosaic virus (PEMV) and each can replicate in the presence of the other's helper virus (Demler *et al.*, 1996). The significance of these findings for the origin and evolution of novel variants of the three components of rosette disease requires study, and

information on the genome variation and population genetics of the three agents of rosette disease are essential for an understanding of the various control strategies. This would help to resolve uncertainties concerning rosette epidemiology including (i) whether different outbreaks in the same area have a common origin (ii) where the disease originated, in terms of season, region and plant species (iii) whether long-range migration of the aphid vector contributes to disease outbreaks (iv) whether the primary source(s) of green and chlorotic rosette are the same (v) does inter-mixing among the three agents occur and lead to new variants if both forms of rosette are inoculated into the same plant by the vector? (vi) do variants among GRAV, GRV and its sat RNA arise under selection pressure and, if so, could this contribute to the origin of new strains that differ in pathogenicity?

Aphid biotypes

The existence of biotypes of *A. craccivora* that differ in host-plant specificity and transmission efficiency has important implications for the epidemiology of rosette. Important unanswered questions include: (i) how do the immigrating alates from hosts other than groundnut behave on groundnut? (ii) are the alates and subsequent progeny from dry season groundnut volunteers more efficient colonisers and vectors than the immigrant alates from non-cultivated legume species? (iii) if so, does the secondary spread of rosette within crops by groundnut-feeding aphids have more influence than primary infection from outside sources on disease outbreaks? and (iv) is there any specificity between different biotypes of aphid vector and green and chlorotic forms of rosette? Other questions concern the stability of the host preferences of *A. craccivora* biotypes and its potential effect on rosette disease transmission. Studies are required to assess the role of *A. craccivora* biotypes originating from host plants other than groundnut and from different geographic locations in rosette epidemics. Genetic markers such as mitochondrial DNA variation can be utilised to investigate the degree of genetic heterogeneity among populations of *A. craccivora*, as shown with other aphid species (Black *et al.*, 1992; Martinez, Moya, Latorre & Fereres, 1992) and to develop criteria for differentiating biotypes. Such molecular markers may provide a useful tool to rapidly identify the presence of biotypes at the field level.

Separation of rosette disease agents in time and space

Studies have shown that not all groundnut plants showing rosette symptoms contain GRAV. Such plants cannot serve as sources of inoculum and thus remain dead ends of the disease. Vector transmission studies have also shown that a proportion of viruliferous aphids inoculate only either GRAV or GRV and its sat RNA. Further studies are required to understand the causes for such a separation of rosette disease agents in time and space and its epidemiological significance. It is also important to know how GRV and its sat RNA are packaged in GRAV coat protein to form particles which have the infectivity of GRV and are transmissible by the aphid vector of GRAV. Similarly, the intriguing role of sat RNA in aphid transmission and in the biological survival of GRV is currently unknown. A complete understanding of these processes might help to devise novel control strategies that would interfere with the packaging of GRV and its sat RNA in GRAV particles thereby preventing disease transmission and spread.

Alternative hosts

It is likely that there are native plants from which the rosette disease spreads to groundnut and there is a need to identify these. Attempts to identify alternative hosts using symptom development, aphid transmission studies etc. (Adams, 1967) or using the diagnostic tools mentioned earlier (D B Dangora and D J Robinson, unpublished data) have not revealed any

non-cultivated host for the rosette complex. Consequently, it is unclear whether the main spread of rosette disease is between groundnut crops and/or from other sources. However, many native African plants remain untested and further work on this aspect is required to determine whether alternative hosts are of continuing epidemiological importance as sources of infection in the range of agro-ecosystems in which the groundnut crop is grown.

Resistant cultivars on disease epidemics

The relative effects of resistant cultivars on disease distribution and progress over time, and their potential as one of the tools for management of rosette epidemics under a range of agro-ecological conditions need to be evaluated. Recent studies in Malawi (R A Naidu, P Subrahmanyam and P J A van der Merwe, unpublished data) revealed that improved rosette disease-resistant lines varied in their response to GRAV (as measured by GRAV titre in TAS-ELISA). This type of resistance to GRAV is advantageous because it is expressed throughout the life of an infected plant. Such plants are poor sources of virus for the aphid vector and in the field the amount of virus spread is less than that from plants having a higher viral content (Barker & Harrison, 1986; Gray, Smith & Sorrells, 1994). Benefits of this type of host gene-mediated resistance can be realised by combining with other desirable agronomic characteristics in the breeding programme.

Pathogen-derived resistance

In addition to conventional breeding efforts, opportunities also exist for utilising biotechnological approaches (Wilson, 1993) to incorporate pathogen-derived resistance to one or more of the rosette disease agents. Possible constructs could be based on the coat protein gene of GRAV (Scott *et al.*, 1996), the RNA-dependent RNA polymerase gene of GRV (Taliansky, Robinson & Murant, 1996), or sequences of a variant sat RNA that down-regulates GRV replication (Taliansky & Robinson, 1997). Progress in this area is currently hampered by the difficulty in transforming groundnuts, but there are prospects of success in the medium to long term. It would be advantageous to exploit a combination of virus resistance genes and genes for vector-resistance to broaden the genetic base of resistance to rosette disease and enhance its durability.

Disease spread in time and space

The relative importance of primary spread into and subsequent secondary spread within groundnut crops is not clearly established. It is essential to make detailed studies on the pattern and sequence of spread at a representative range of sites in different agro-ecologies and under different cropping systems over a period of several years. Experience with other arthropod-borne viruses (Harrington *et al.*, 1994) suggests that it will be important to monitor the numbers and infectivity of the vectors reaching crops, especially during the critical early stage of growth since young plants are most vulnerable to infection and early infection provides the greatest opportunity for secondary spread. Research is also needed on the factors affecting aphid movement and survival in the crop in order to better understand disease build-up and the initiation of outbreaks. Little is known about the relative importance of short and long-range dispersal of the vector and the influence of environmental factors including wind and frontal systems on such movements. There is no direct evidence that *A. craccivora* behaves in the same way in tropical SSA as it does in south-east Australia. Studies on potential migration may not be justified until circumstantial evidence of this is obtained since obtaining adequate information would be both difficult and expensive in Africa. Nevertheless, the recent development of rapid, microcomputer-based Geographic Information Systems

(GIS) with analytical capabilities provide a potential tool to investigate the range of climatic and ecological conditions under which the aphid thrives and disease outbreaks occur.

Modelling

Modelling and simulation have facilitated rapid advances in understanding other pathosystems due to their speed and ability to handle the more complex systems, especially in studies where an objective is disease forecasting. There are opportunities for making rapid advances in using mathematical techniques and indeed some of the models developed for insect-borne virus diseases such as cassava mosaic in SSA could be adapted for predicting and understanding rosette disease epidemics and making strategic disease management decisions (Thresh, 1983*b*; Jeger & van den Bosch, 1993; Fargette & Vié, 1994, 1995; Camann *et al.*, 1995; Madden & Nutter, 1995; Madden & Hughes, 1995; Holt, Jeger, Thresh & Otim-Nape, 1997).

Disease forecasting

An ability to forecast the incidence of rosette disease with reasonable precision, and ideally before or soon after crops are planted, could lead to improved control measures and management practices (Thresh, 1983*b*). Early studies of what would now be regarded as forecasting were made on rosette in South Africa during the early 1920s, when severe epidemics were associated with unusually high rainfall during the normally dry winter months before planting commences (Storey & Bottomley, 1928). Such studies have not been pursued further either in South Africa or elsewhere, although considerable progress has been made in forecasting the incidence of other aphid-borne viruses which have a persistent mode of transmission including potato leafroll and barley yellow dwarf luteoviruses (Harrington *et al.*, 1994). An important component of disease forecasting is the continuous monitoring of the number of viruliferous aphids. This can be done by a combination of sampling aphid populations using the Rothamsted 12 m suction trap (Macaulay, Tatchell & Taylor, 1988) and testing them for the presence of rosette disease agents. Such information, together with meteorological data, can provide key parameters for developing effective forecasting methods.

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