HOST-PLANT VIRAL INFECTION EFFECTS ON ARTHROPOD-VECTOR POPULATION GROWTH, DEVELOPMENT AND BEHAVIOUR: MANAGEMENT AND EPIDEMIOLOGICAL IMPLICATIONS

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Single factors, such as the introduction of a non-indigenous vector species and/or plant virus, are often invoked to explain the emergence and spread of plant virus epidemics. This has often been so when an epidemic has been associated with a noticeable increase in the vector population, as frequently occurs during the initial outbreaks and subsequent spread of arthropod vector-borne plant-virus epidemics. Such explanations, however, should be considered more appropriately in the wider context of the complex interactions that occur between the virus(es), host-plant species, the environment and the vector(s). Here, we review evidence for overall positive, negative and neutral effects on the relative population growth of several arthropod vectors when feeding on virus-infected and
uninfected host plants. The emphasis is on the whitefly, *Bemisia tabaci*, aphids, leafhoppers, mites and thrips. The *B. tabaci*-borne cassava mosaic disease pandemic in sub-Saharan Africa and the tomato leaf curl virus epidemics in the Indian subcontinent are then considered in more detail and experimental data are presented to show, for both of these cases, that a mutually beneficial relationship between the virus and the vector is an important component of the mechanism driving these epidemics. This mechanism is discussed in the context of associated changes in the behaviour of the whitefly vector as well as biochemical changes within the plant.

**I. Introduction**

The increased movement of plant material globally has resulted in the frequent introduction of plant viruses and vectors into new regions. This increase in global traffic has occurred together with a trend towards intensification of agriculture arising from the need to produce more food from a decreasing land area and with fewer resources. These factors have combined to alter ecosystems and favour the rapid spread of plant virus diseases, particularly those that are transmitted by arthropod vectors such as whiteflies, aphids, leafhoppers, thrips and eriophyid mites (Bos, 1992; Thresh, 1980).

Single causative factors, such as the arrival of a non-indigenous vector species and/or a plant virus, can sometimes provide an appealingly simple explanation for the appearance and rapid spread of plant virus epidemics. This has generally been so when epidemics are associated with an easily identifiable characteristic such as an increased disease severity and/or a marked increase in the vector population. More appropriately, however, the mechanisms driving arthropod-borne plant virus epidemics may be considered to encompass a suite of complex interactions of differing importance, which occur at several levels between the vector(s), the virus(es), the host-plant species and the environment.

Here, we review the evidence for positive, negative and neutral effects on the population growth of several arthropod vector species when feeding on virus-infected compared with uninfected host plants. The emphasis is on whiteflies and aphids as these have received the greatest attention. In part, this has been due to the dramatic increase, in the last 10–15 years, in agricultural and horticultural problems caused particularly by the whitefly, *Bemisia tabaci*, and the many viruses it transmits (Boulton, 2003; Jones, 2003; Morales and Anderson, 2001; Polston
and Anderson, 1997; Morales, this volume, pp. 127–162). Two important examples are the *B. tabaci*-borne cassava mosaic disease (CMD) pandemic, which continues to devastate cassava production in large areas of sub-Saharan Africa (SSA), and the tomato leaf curl disease epidemics in the Indian subcontinent, which have had equally serious implications for tomato production in this region. These problems are considered in detail and experimental data are included to show, for both pathosystems, that the *B. tabaci* colonising virus-infected host plants have significantly higher-population growth rates compared to those colonising virus-free hosts. This effect is discussed in the context of the significantly higher densities of *B. tabaci* present on symptomatic cassava and the behavioural changes associated with this effect. Data are also presented to show that the concentrations of four amino acids were significantly higher in the phloem sap of CMD-infected cassava plants. These interacting effects are considered in relation to probable mechanisms contributing to the rapid spread of these epidemics.

II. VECTOR–VIRUS–HOST PLANT INTERACTIONS

A. Aphids

Experiments on *Beet mosaic virus* (BtMV; Genus: *Potyvirus*; Family: *Potyviridae*) provided some of the first evidence that the population growth rate of an arthropod vector could be affected by the health status of the host plants (Kennedy, 1951). *Aphis fabae* reproduced approximately 1.5 times faster on virus-infected than on uninfected plants, which resulted in the aphid colonies on infected plants becoming overcrowded more rapidly. This, in turn, resulted in emigration of viruliferous adults beginning more quickly from the infected plants which, it was proposed, would lead to further virus spread.

Baker (1960) reported a similar phenomenon and showed that three aphid species bred more rapidly and lived longer on sugar beet leaves infected with virus yellows than on the green leaves of healthy plants. Other researchers also subsequently demonstrated that the English grain aphid, *Sitobion avenae* (formerly *Macrosiphum granarium*), had an increased rate of development, lived longer, had a longer reproductive period and, therefore, produced more progeny on *Barley yellow dwarf virus* (BYDV; Genus: *Luteovirus*; Family: *Luteoviridae*)-infected oats than on healthy plants (Miller and Coon, 1964). A significantly greater fecundity and reduced developmental period was also demonstrated for
S. avenae when feeding on BYDV-infected wheat compared to those feeding on healthy plants (Fereres et al., 1989).

The performance of the aphid, Myzus persicae, was also reported to improve on sugar beet plants infected with Beet yellows virus (BYV; Genus: Closterovirus; Family: Closteroviridae) compared to those colonising healthy plants. This effect was so rapid that nymphs born at the time of virus inoculation benefitted as much as those produced later (Williams, 1995).

The groundnut rosette virus disease complex (Naidu et al., 1998) provides another example in which symptomatic groundnut plants were more attractive to the aphid vector and populations colonising them developed more quickly. Also, higher numbers of winged adults were produced than on healthy plants (Réal, 1955). In other work, Aphis craccivora populations increased significantly faster on the infected compared to healthy plants of the groundnut varieties ICG12991 and JL24, which was due to an increase in aphid fecundity (Willekens, 2003).

As well as affecting the reproductive rate of vectors, virus-infected host plants have been shown to affect the physiological development of vectors. The cereal grain aphids, S. avenae and Rhopalosiphum padi, for instance, when reared from birth on BYDV-infected barley or oats, produced more than twice as many alates (winged adults) compared to those reared on otherwise comparable healthy plants. This effect was also apparent when field-collected first and second instar nymphs were transferred to BYDV-infected plants (Gildow, 1983; Montllor and Gildow, 1986). It was proposed that changes in host-plant physiology had affected aphid nutrition and development, leading to an increased ratio of winged to non-winged progeny. These interactions were considered adaptive in that they favoured viruliferous aphid dispersal by flight and, thus, the spread of viruses within and between crops (Gildow, 1983; Sohi and Swenson, 1964). Power (1992) later summarised the then available data for the BYDV pathosystem, and suggested that it involved an indirect form of mutualism between the vectors and virus mediated through the host plant.

The most recent work on this system involves the complex interactions that occurred when the bird cherry–oat aphid, R. padi, was allowed to colonise transgenic and untransformed wheat challenged with BYDV (Jiménez-Martínez et al., 2004a,b). The results indicated that transgenic virus resistance in wheat directly influenced R. padi settling preference, total fecundity, length of reproductive period and intrinsic rate of increase. Healthy transgenic plants were superior hosts for R. padi compared to the untransformed parental variety
Lambert. The situation was reversed, however, when Lambert plants were infected with BYDV and, in this situation, the transgenic plants became the inferior hosts.

For a different system, Markkula and Laurema (1964) found that the reproduction of the pea aphid, *Acyrthosiphon pisum*, increased on *Bean yellow mosaic virus* (BYMV; Genus: *Potyvirus*; Family: *Potyviridae*)-infected red clover (*Trifolium pratense*) expressing moderate viral symptoms compared with healthy plants, but that it decreased on plants with severe symptoms. They also found that the reproduction of *R. padi* increased with increasing concentration of the free amino acids on oats infected with BYDV but that the reproduction of two other aphid species remained unchanged, demonstrating that the effects of a virus on a particular host-plant species may not always result in an increased fecundity and survival of all virus vectors.

This point was also illustrated when *M. persicae* was reared on healthy and *Cucumber mosaic virus* (CMV; Genus: *Cucumovirus*; Family: *Bromoviridae*)-infected seedlings of *Nicotiana tabacum*, *Gomphrena globosa* and *Zinnia elegans*. After 8–12 days, populations were 2.0–4.8 times larger on the healthy than on diseased seedlings of each species (Lowe and Strong, 1962). In CMV-infected *N. tabacum*, the amount of glutamic acid had decreased after 182 h, which was proposed as the explanation for the poorer performance of aphids on CMV-infected plants.

The amino acid content of the phloem of diseased plants typically increases in response to virus infection (Matthews, 1981) and Selman et al. (1961) suggested that some viruses affect the biochemistry of host plants in a unique way. BYDV-infected spring wheat, for example, had an increased total amino acid content, and both alanine and glutamine occurred consistently in greater amounts than in healthy leaves. At the oldest growth stage, for instance, there was approximately 2.5 times more alanine in the infected plants’ phloem sap than in that of uninfected plants (Ajayi, 1986).

In BYV-infected sugar beet plants, aspartic and glutamic acids decreased, whereas other amino acids either increased, such as citrulline and threonine by three- and fourfold, respectively, or remained unchanged (Fife, 1961). These changes may account for the improved performance of *M. persicae* on BYV-infected plants compared to those colonising healthy sugar beet (Williams, 1995).

Much of the variability in the influence of virus-infected plants on vectors has been attributed to differences in the nitrogenous compounds present in these plants (Power, 1992), as these form an important constituent of the food of phloem-feeding insects (Auclair,
In most infections, the amount of virus protein synthesised within an infected plant is probably less than to be expected from the quantities of precursors available. Increased concentrations of free amino acids could potentially benefit both the virus and the vector, and further instances in which higher-nitrogen levels in the host plant led to increased vector populations are provided by Barker and Tauber (1951) and Coon (1959). However, there is a dearth of data on the specific amino acid requirements of vectors and so it is difficult to relate their performance to the effect of the virus infection on the concentration of individual amino acids within the host plant.

BYDV infection has also been shown to increase carbohydrate content of wheat tissues and it was suggested that this may account for the increased fecundity of aphids observed on such plants (Fereres et al., 1990).

Symptom expression, or otherwise, by virus-infected plants is subject to various selection pressures and, therefore, may also be a component of the mechanism that influences vector dispersal and thus virus spread. Many viruses produce a marked yellowing of infected tissue, yellow mosaics or yellow vein mosaics (Ajayi and Dewar, 1983; Muniyappa, 1980; Watson and Healy, 1953). Yellow is an attractive colour for many aphid, whitefly and other vector species (Dixon, 1985; Kennedy et al., 1961; Mound, 1962) and, in the field, this may increase the ‘contact rate’ between the virus and the vector, and may thus increase the proportion of viruliferous vectors in the population as a whole (Ajayi and Dewar, 1983).

The aphid M. persicae is the principal vector of Potato leaf roll virus (PLRV; Genus: Polerovirus; Family: Luteoviridae) and it transmits the virus in a persistent manner (Harrison, 1984). M. persicae grows faster, has higher fecundity and settles preferentially on cultivated potato, Solanum tuberosum, infected by PLRV than on uninfected potato plants (Castle and Berger, 1993; Castle et al., 1998). Eigenbrode et al. (2002) have since demonstrated that this preferential colonisation is influenced by volatile emissions from PLRV-infected plants.

Aphid species have plant physiological preferences which, combined with the tendency to move more frequently when feeding on a food source of poor quality, result in aphids colonising parts of plants where they can achieve the highest growth and developmental rates (Ibbotson and Kennedy, 1950; Kennedy et al., 1950). Other aphid species induce plant galls that provide them diverse benefits including a richer food supply than non-galled leaves, extension of the usual period for which high-quality food is available and protection from weather and predators (Forrest, 1971; Kennedy, 1951). Although they do not induce plant galls,
Aphid species including *Schizaphis graminum* and *Diuraphis noxia* induce chlorotic lesions and increase the amino acid concentrations of the phloem sap when feeding on grasses possibly in a nutritionally advantageous manner (Sandström *et al.*, 2000).

Aphids also demonstrate a strong preference for the best feeding sites on plants (Whitham, 1979), but the effect of viral disease symptoms on the suitability of these feeding sites is generally unknown.

**B. Leafhoppers, Thrips and Mites**

Information on the effects of plant viruses on their leafhopper vectors is relatively sparse and various authors have reported contradictory results. Several reviews, however, have concluded that plant viruses which multiply within their insect vector, such as those transmitted by delphacid leafhoppers, have not been shown unequivocally to harm the vectors (Nault, 1994; Nault and Ammar, 1989; Sinha, 1981). A general difficulty with experiments investigating this possibility, however, is the practical problem of separating the direct effects of the virus on the vector from those caused by the vector feeding on virus-infected host-plant tissue.

Fife (1956) reported that sugar beet plants infected with *Beet curly top virus* (BCTV; Genus: *Curtovirus*; Family: *Geminiviridae*) transmitted by the beet leafhopper, *Neoaliturus tenellus* (formerly *Circulifer tenellus*), contained twice the concentration of amino acids that occurred in equivalent healthy plants. Other work showed that sugar beet leafhopper populations were highest in fields where plant growth had been retarded in the early stages of development. These fields also had the highest incidence of BCTV and it was suggested that the disruption in leaf canopy produced sunnier and lower humidity conditions within the infected crop, which suited the beet leafhopper (Bennett, 1971). In the last decade, epidemics of BCTV have been occurring with increasing frequency in New Mexico, resulting in substantial losses (Creamer *et al.*, 2003). Although the plant–virus–vector interactions driving these epidemics have yet to be elucidated, it seems likely that there is an indirect positive effect of host-plant virus infection on *N. tenellus* population growth.

An example of the feeding behaviour of a leafhopper being affected by the virus it transmits is provided by *Cicadulina storeyi*, which spent more time ingesting from mesophyll and tissues other than the phloem when host plants were infected with *Maize streak virus* (MSV; Genus: *Mastrevirus*; Family: *Geminiviridae*) (Mesfin and Bosque-Pérez, 1998).
MSV virions are usually located in the nuclei of cells from the epidermis to the phloem parenchyma (Pinner et al., 1993) and so this altered behaviour increases the likelihood of virus acquisition.

One of the earliest reports of a plant virus affecting the population dynamics of its vector involved *Thrips tabaci*—the vector of yellow spot virus disease of pineapple in Hawaii (Carter, 1939). On its alternative weed host, *Emilia sonchifolia*, which is also susceptible to the virus, thrips populations were consistently higher on diseased than on healthy plants. It was suggested that the diseased plants also lived longer and that their mass of curled leaves provided better shelter to the thrips.

In a series of experiments, the direct and indirect (through the host plant) effects of *Tomato spotted wilt virus* (TSWV; Genus: Tospovirus; Family: Bunyaviridae) on its main vector, the western flower thrips, *Frankliniella occidentalis*, were quantified (Belliure et al., 2005; Maris et al., 2004). There was both a positive direct effect of TSWV on *F. occidentalis* and an indirect effect in which *F. occidentalis* juvenile survival and developmental rates were lower on healthy pepper plants than on those that had been either infected by thrips or by mechanical inoculation. It was suggested, therefore, that TSWV had evolved a mechanism to overcome the plant defences against the thrip vectors and thus assist its spread.

Another example of viral infection inducing changes in host-plant morphology is provided by *Blackcurrant reversion virus* that infects blackcurrant, *Ribes nigrum*, bushes. Infected plants have a reduced density of hairs on their leaves, flowers and stems and produce more shoots of a shorter length, which greatly increases the availability, accessibility and vulnerability of buds to the eriophyid gall mite vector, *Cecidophyopsis* (formerly *Phytopus*) *ribis* (Thresh, 1967).

In the pigeonpea sterility mosaic virus (PPSMV; ungrouped) pathosystem (Jones et al., 2004), a mutually beneficial interaction also occurs between the eriophyid mite, *Aceria cajani*, and the virus. This mite is highly host specific and is largely confined to pigeonpea and its wild relatives. The mites predominantly inhabit the lower surface of symptomatic leaves of PPSMV-infected plants. Multiplication of *A. cajani* on cultivated pigeonpea was much greater on PPSMV-infected plants than on healthy plants of the same genotype (Kulkarni et al., 2002; Muniyappa and Nangia, 1982; Reddy and Nene, 1980).

Another example of this effect is provided by the eriophyid mite, *Phyllocopetes fructiphylus*, the vector of rose rosette disease (RRD: causal agent yet to be determined) in multiflora rose, *Rosa multiflora*. From July to September, field populations of *P. fructiphylus* were up to
17 times higher on RRD-symptomatic R. multiflora than on non-symptomatic plants (Epstein and Hill, 1999).

C. The Whitefly, B. tabaci

The whitefly, B. tabaci, has become an extremely serious agricultural and horticultural pest both by causing direct damage and as a vector of more than 110 plant viruses (Jones, 2003; Polston and Anderson, 1997). Several studies have reported effects of virus infection of the host plants on B. tabaci reproduction rate and behaviour. Berlinger (1986), for instance, reported a significant preference by B. tabaci for healthy tomato plants compared to those infected with Tomato yellow leaf curl virus (TYLCV; Genus: Begomovirus; Family: Geminiviridae), and the effect was apparent even before the virus symptoms had developed. Negative effects of TYLCV on B. tabaci were also reported subsequently by Rubenstein and Czosnek (1997), where viruliferous B. tabaci exhibited a 17–23% reduction in adult life expectancy and produced 40–50% fewer eggs compared to TYLCV-free individuals. It was suggested that TYLCV, which had previously been considered to infect plants only, had features reminiscent of an insect pathogen.

In another study (Costa et al., 1991), adult B. tabaci from a colony that had been reared on a succession of squash plants for more than 5 years were exposed in clip cages to six plant species infected with one of four whitefly-transmitted plant viruses. Groups of 10 adult females were confined on them for 48 h and then the number of eggs was counted and their survival through to adulthood was assessed 3 to 4 weeks later. The mean number of eggs oviposited on healthy pumpkin compared to pumpkin infected with the watermelon curly mottle strain of Squash leaf curl virus (WCMoV/SLCV; Genus: Begomovirus; Family: Geminiviridae) was not significantly different. Survival to adulthood, however, was significantly greater on the diseased than on the healthy pumpkin and total free amino acid concentrations were significantly higher in virus-infected pumpkin, lettuce, tomato, zucchini squash, cotton and cantaloupe melon than in healthy plants of the same species. No simple relationship could be detected between total free amino acid levels and the oviposition or survival rates of B. tabaci, however, which suggested that B. tabaci did not assess host suitability for offspring survival and that, for any given host plant/virus/B. tabaci combination, there was not necessarily a positive effect on vector oviposition or survival (Costa et al., 1991).

More work has demonstrated clearly that the performance of B. tabaci populations can be affected positively by the health status
of the plants they feed on. Colvin et al. (1999) reported that the fecundity of cassava B. tabaci was greater on cassava infected with East African cassava mosaic virus-Uganda [Namulonge] (EACMV-UG [NAM]; Genus: Begomovirus; Family: Geminiviridae) than on otherwise comparable healthy plants. Mayer et al. (2002) and McKenzie et al. (2002) also reported a similar effect with Tomato mottle virus (ToMoV; Genus: Begomovirus; Family: Geminiviridae), where a B. tabaci biotype B population produced 2.5-fold more eggs on infected plants than those on healthy plants. This study is particularly interesting as the B. tabaci biotype B is continuously expanding its geographic range and has arrived in South India (Banks et al., 2001).

III. Studies on the Tomato Leaf Curl Pathosystem in India

Tomato leaf curl viruses (ToLCVs) are the most important viral pathogens of tomato in India and infected plants show a variety of symptoms including leaf curling, stunting and partial or complete sterility (Chowda Reddy et al., 2005; Ramappa et al., 1998; Saikia and Muniyappa, 1989). B. tabaci is the only known vector of ToLCVs (Vasudeva and Sam Raj, 1948) and both the vector and the viruses have a wide host-plant range, and many species act as a reservoir for both the viruses and the vectors throughout the year (Chowda Reddy et al., 2005; Ramappa et al., 1998).

Populations of B. tabaci fluctuate significantly each year in India and in the south they are highest during the hot season, which lasts from February to May (Ramappa et al., 1998; Saikia and Muniyappa, 1989). ToLCV disease is highly positively correlated with the size of the B. tabaci population and, in ToLCV-susceptible tomato not sprayed with insecticide, the disease incidence frequently reaches 100%, resulting in complete yield loss if plants are infected as seedlings (Holt et al., 1999; Ramappa et al., 1998; Saikia and Muniyappa, 1989).

A. B. tabaci Population Growth on Healthy and ToLCV-Infected Plants

In order to investigate possible reasons for the positive correlation between the size of the B. tabaci population and ToLCV-disease incidence, the common weed hosts Euphorbia geniculata, Parthenium hysterophorus, Acanthospermum hispidum, Ageratum conyzoides and tomato (Lycopersicon esculentum cv. Arka Vikas) were selected as experimental species as these are important sources of both B. tabaci

Seeds of the five plant species were collected, planted and seedlings were transplanted individually into earthenware pots at 7 days after germination. Plants were kept for a further 7 days after transplanting to allow establishment and further growth before use. The transplanted seedlings were maintained under *B. tabaci*-proof nylon netting to ensure they remained free of whiteflies and were not infected accidentally with ToLCVs.

The *B. tabaci* colony was an indigenous strain (analysed by sequencing of the mitochondrial cytochrome oxidase I gene (mtCOI), GenBank no. AM/4C595) that had been maintained on cotton at the Hebbal Campus of the University of Agricultural Sciences, Bangalore. The virus isolate used in the experiment was ToLCBV-[Ban4]. Cohorts of newly emerged adults were collected as follows. A single cotton plant was selected that had a high proportion of *B. tabaci* ‘pupae’ present on the leaves. All the adult insects were removed from the plant at 16:00 h on a particular day, and the plant was then left undisturbed overnight in a separate cage. The following day at 11:00 h, newly emerged adults were collected and approximately equal numbers were allowed to feed for 24 h on healthy or ToLCV-[Ban4]-infected tomato seedlings. The virus-free and the potentially viruliferous groups of *B. tabaci* were then collected separately and sexed. Initial populations of 10 male and 10 female insects were used to initiate either ToLCV-[Ban4]-free or ToLCV-[Ban4]-infected colonies on the host-plant seedlings. The seedlings with *B. tabaci* adults were enclosed in netting bags that prevented the adult *B. tabaci* from escaping, without allowing the build-up of condensation that might otherwise trap insects in water droplets or affect behaviour due to the possible impact of high humidity. Eighteen days after the release of the colonising *B. tabaci*, any adults present were collected and counted. The leaves of each plant were then examined under a microscope, and the numbers of eggs and nymphs were recorded. The adults were then released back onto the same plant and the process was repeated after a further 18 days. Data were analysed using a repeated measurements analysis of variance (GenStat 8.1, 2005).

The *B. tabaci* population growth on the ToLCV-[Ban4]-infected plants was significantly higher for all host-plant species, although the extent of the effect depended on the plant species (Fig. 1 and Table I). *B. tabaci* successfully colonised all of the plant species and populations increased significantly over time regardless of plant health status (Fig. 1 and Table I). The *B. tabaci* population increase was lowest on healthy
FIG 1. The population (adults, nymphs and eggs) growth of Indian \textit{B. tabaci} on ToLCBV-[Ban4]-infected and uninfected host plants of tomato (\textit{Lycopersicon esculentum} cv. Arka Vikas) and the common weed hosts \textit{E. geniculata}, \textit{Parthenium hysterophorus}, \textit{Acanthospermum hispidum}, \textit{Ageratum conyzoides}.

\begin{table}[h]
\centering
\caption{Repeated Measurements Analysis of Variance for Ln \textit{(B. tabaci Population Count)} at 18 and 36 Days After Groups of 10 Male and 10 Female Newly Emerged Adults Per Plant Were Allowed to Colonise Five Different Plant Species.}
\begin{tabular}{lcccc}
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Source of variation & Wald statistic & d.f. & Wald/d.f. & Chi ($P$) \\
\hline
Time & 21 929.24 & 2 & 10 964.62 & $<0.001$ \\
Plant species & 63.27 & 4 & 15.82 & $<0.001$ \\
Plant health$^a$ & 353.01 & 1 & 353.01 & $<0.001$ \\
Time $\times$ plant species & 67.05 & 8 & 8.38 & $<0.001$ \\
Time $\times$ plant health & 287.56 & 2 & 143.78 & $<0.001$ \\
Plant species $\times$ plant health & 38.68 & 4 & 9.67 & $<0.001$ \\
Time $\times$ plant species $\times$ plant health & 32.98 & 8 & 4.12 & $<0.001$ \\
\hline
\end{tabular}
\end{table}

$^a$ Plants either remained healthy or became infected with ToLCBV-[Ban4].
tomato plants and highest on infected *E. geniculata*, which was consistent with the observation that tomato is not considered a preferred host plant of South Indian *B. tabaci* (Ramappa *et al.*, 1998).

IV. STUDIES ON THE CASSAVA MOSAIC PATHOSYSTEM IN AFRICA

A. Current Cassava Mosaic Pandemic in Africa

Cassava is grown widely in SSA as a staple food crop although, in the last decade, production has been affected severely by a pandemic of an unusually severe form of CMD (Otim-Nape *et al.*, 2000; Thresh *et al.*, 1997). This disease is caused by whitefly-borne cassava mosaic viruses (Genus: *Begomovirus*; Family: *Geminiviridae*). Data collected as the CMD pandemic spread into new regions of southern Uganda were consistent with the rapid spread of the novel EACMV-UG (Zhou *et al.*, 1997) into areas where formerly only African cassava mosaic virus (ACMV) occurred. This resulted in a predominance of cassava plants with dual infections that expressed symptoms that were more severe than those caused by either virus alone (Colvin *et al.*, 2004; Harrison *et al.*, 1997). The spread of the pandemic was associated with significantly increased *B. tabaci* populations, which migrated into new areas ahead of a CMD ‘front’, thus causing its rapid spread (Colvin *et al.*, 2004; Legg and Ogwal, 1998; Otim-Nape *et al.*, 2000).

One hypothesis to explain the increased *B. tabaci* numbers proposed that a putative ‘invader’ *B. tabaci* genotype cluster, identified by a single mtCOI gene sequence present in *B. tabaci* adults collected from cassava in 1997 and 1999, was spreading southwards with EACMV-UG. It was proposed that this invader *B. tabaci* population might be better adapted to cassava, more highly fecund and able to transmit EACMV-UG with greater efficiency than the ‘local’ Ugandan *B. tabaci* genotypes that were present previously (Legg *et al.*, 2002). Various experiments designed to test for associations between fitness traits of the ‘invader biotype’ and the presence of the pandemic, however, proved unsuccessful (Colvin *et al.*, 2004; Maruthi *et al.*, 2001, 2002, 2004a). Moreover, in a separate analysis of *B. tabaci* samples collected from within and outside the CMD-affected zone in 1997, there was no apparent association between the presence of EACMV-UG and the two mtCOI haplotypes (Fig. 2 and Table II). Furthermore, in the separate study reported here, *B. tabaci* populations (UgCas-Ss1 and UgCas-Ss2) collected in 1997 with the so called invader mtCOI sequence were found in areas of Ssanji, southern Uganda, which were, at that time,
FIG 2. Clustering of partial mtCOI sequences of *B. tabaci* associated with CMD in Africa and ToLCVD in India. Samples in red and blue originate from the CMD epidemic and pre-epidemic zones in Uganda, respectively.

Red text = Epidemic site
Blue text = Non-epidemic site
Black text = Control populations

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Fig 2. Clustering of partial mtCOI sequences of *B. tabaci* associated with CMD in Africa and ToLCVD in India. Samples in red and blue originate from the CMD epidemic and pre-epidemic zones in Uganda, respectively.
<table>
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<th>Source plant, country</th>
<th>Location</th>
<th>Abbreviation</th>
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<td>AY057158</td>
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<td>UgCas-Mk6</td>
<td>AM040598</td>
</tr>
<tr>
<td></td>
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<td>AM040599</td>
</tr>
<tr>
<td></td>
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<td>UgCas-Mit1</td>
<td>AM040600</td>
</tr>
<tr>
<td></td>
<td>Mityana site 2</td>
<td>UgCas-Mit2</td>
<td>AM040601</td>
</tr>
<tr>
<td></td>
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<td>UgCas-Nk1</td>
<td>AM040602</td>
</tr>
<tr>
<td></td>
<td>Nkosi site 2</td>
<td>UgCas-Nk2.1</td>
<td>AM040603</td>
</tr>
<tr>
<td></td>
<td>Nkosi site 2</td>
<td>UgCas-Nk2.2</td>
<td>AM040604</td>
</tr>
<tr>
<td></td>
<td>Namulonge</td>
<td>UgCas-Nam2</td>
<td>AM040605</td>
</tr>
<tr>
<td></td>
<td>Ssanji</td>
<td>UgCas-Ss1</td>
<td>AM040606</td>
</tr>
<tr>
<td></td>
<td>Ssanji</td>
<td>UgCas-Ss2</td>
<td>AM040607</td>
</tr>
<tr>
<td>Cassava, Ghana</td>
<td>Tamale site 1</td>
<td>GhCas-Tam1</td>
<td>AM040608</td>
</tr>
<tr>
<td></td>
<td>Tamale site 2</td>
<td>GhCas-Tam2</td>
<td>AM040609</td>
</tr>
<tr>
<td></td>
<td>Accra</td>
<td>GhCas-Acc</td>
<td>AF418668</td>
</tr>
<tr>
<td>Cassava, Mozambique</td>
<td>82Moz</td>
<td>AF344278</td>
<td></td>
</tr>
<tr>
<td>Cassava, Malawi</td>
<td>21Malawi</td>
<td>AY057162</td>
<td></td>
</tr>
<tr>
<td>Cassava, Tanzania</td>
<td>Mtwarra</td>
<td>TzCas-Mtw</td>
<td>AF418667</td>
</tr>
<tr>
<td>Cassava, South Africa</td>
<td>St Lucia</td>
<td>81SA Lucia</td>
<td>AF344259</td>
</tr>
<tr>
<td>Cassava, Cameroon</td>
<td>76Cam</td>
<td>AF344247</td>
<td></td>
</tr>
<tr>
<td>Zimbabwe</td>
<td>83Zim</td>
<td>AF344285</td>
<td></td>
</tr>
<tr>
<td>Sweet potato, Uganda</td>
<td>Namulonge</td>
<td>UgSp-Nam</td>
<td>AF418665</td>
</tr>
<tr>
<td>Cassava, India</td>
<td>Bangalore</td>
<td>IndCas-Ban</td>
<td>AF418666</td>
</tr>
<tr>
<td></td>
<td>Trivandrum</td>
<td>IndCas-Tri</td>
<td>AF418670</td>
</tr>
<tr>
<td>Tomato</td>
<td>Kolar</td>
<td>IndTom-Kol</td>
<td>AF321928</td>
</tr>
<tr>
<td>E. geniculata</td>
<td>Bangalore</td>
<td>IndEg-Ban</td>
<td>AF418664</td>
</tr>
<tr>
<td>Cotton</td>
<td>Bangalore</td>
<td>IndCot-Ban</td>
<td>AM040595</td>
</tr>
<tr>
<td>Egg plant</td>
<td>Coimbatore</td>
<td>IndEgg-Coi</td>
<td>AM040596</td>
</tr>
<tr>
<td>Bangladesh</td>
<td>Joydebpur</td>
<td>BdEg-Joy</td>
<td>AJ748400</td>
</tr>
</tbody>
</table>

(continues)
unaffected by the pandemic (Fig. 2). A further problem with the invader hypothesis arises from a survey of the \textit{B. tabaci} cassava populations in post-epidemic areas of Uganda, which failed to detect invader nymphs developing on cassava (Sseruwagi, 2005), although several other known biotypes were identified to be present for the first time (Sseruwagi et al., 2005). Thus, there is presently no evidence of a link between the spread of the pandemic and the presence of a \textit{B. tabaci} invader population with innately superior fitness attributes.

\textbf{B. Increased} \textit{B. tabaci} Populations on Cassava Mosaic Disease-Affected Plants

A possible alternative explanation for the high-\textit{B. tabaci} populations associated with the epidemic was investigated by an experiment to determine whether plant health status influenced the population growth rate of cassava \textit{B. tabaci}. Thirty-five, 3 weeks old, healthy cassava plants (var. Ebwanateraka) were inoculated using either virus-free or EACMV-UG-infective, \textit{B. tabaci} collected from the pre-epidemic area. Ten days after inoculation, any \textit{B. tabaci} nymphs were removed mechanically from the plants. Five male and five female, 1-day-old, non-viruliferous, pre-epidemic, \textit{B. tabaci} adults were introduced onto each plant, which was then covered with a perforated plastic bag. Nymph and adult numbers were recorded both 3 and 6 weeks later as was the presence of any CMD symptoms. On the latter

\begin{table}[h]
\centering
\caption{continued)}
\begin{tabular}{llll}
\hline
Source plant, country & Location & Abbreviation & Accession numbers \\
\hline
China & 15China & & AF342777 \\
Nepal & 22Nepal & & AF342779 \\
Pakistan & 23Pakis & & AF342778 \\
Tel Aviv, Israel & Cabbage & IsCab-Tel & AF418671 \\
Arizona, USA & USAZ-B & & AY057140 \\
Puerto Rico & \textit{Sida} spp. & 27PRSida & AY057134 \\
Arizona, USA & & 4USAZ-A & AY057122 \\
Argentina & 2Arg & & AF340213 \\
Namulonge, Uganda & Sweet potato & 67NamSp & AY057207 \\
Entebbe, Uganda & Cassava & \textit{B. afer} & AF418673 \\
Bangalore, India & Phyllanthus emblica L. & T. vaporariorum & AF418672 \\
\hline
\end{tabular}
\end{table}
date, the top three leaves from each plant were removed for polymerase chain reaction (PCR) diagnostic analysis for geminivirus. Amino acids were also extracted from a randomly selected sub-sample of freeze-dried leaves that were placed in 50% methanol and fractionated by Dowex 50 ion-exchange chromatography (BDH Ltd, UK) before derivatisation with FMOC-Cl and subsequent analysis by high-performance liquid chromatography (Carratu et al., 1995; Worthen and Liu, 1992). Amino acids were identified and quantified by comparison of retention times and peak area with authentic standards (Sigma Ltd). Control plants for the biochemical analysis were EACMV-UG–infected and healthy plants that were free of B. tabaci. PCR and biochemical work was conducted using a blind-analysis protocol.

The B. tabaci populations increased significantly more rapidly on EACMV-UG–infected than on equivalent healthy plants (Fig. 3 and Table III) and the concentrations of asparagine, glutamine, tryptophan and tyrosine increased significantly in the phloem sap of the diseased compared with healthy plants (Table IV).

C. Increased Field-Population Densities of B. tabaci on CMD-Symptomatic Plants

Many arthropod vector species have been reported to aggregate on plants infected with the viruses that they transmit (Bautista et al., 1995; Eigenbrode et al., 2002; Maris et al., 2004) and it has been proposed that this can be explained if plant pathogens in general have evolved mechanisms to overcome plant defences against their vectors, thus promoting pathogen spread (Belliure et al., 2005).

The severe chlorosis that is produced by cassava plants dually infected with both EACMV-UG and ACMV causes a big reduction in plant size, total leaf area and distribution of green leaf tissue (Maruthi et al., 2002; Omongo, 2003). When total B. tabaci numbers per plant were counted, no significant differences were found between populations on CMD-symptomatic and healthy plants (Gibson et al., 1996). This assessment, however, failed to capture the complexity of the situation, as it did not consider the preference of B. tabaci to feed and oviposit on the non-chlorotic areas of symptomatic leaves (Gibson et al., 1996; Omongo, 2003).

Measurements of leaf area of the top five leaves of representative plants showed that the total leaf area of mildly and severely affected plants was reduced to 71% and 30% of the leaf area of symptomless plants, respectively. For plants exhibiting mild symptoms, 80% of the total leaf area was green tissue compared with only 30% for plants...
exhibiting severe symptoms. These factors were considered when investigating the densities of adult and immature stages of *B. tabaci* colonising blocks of cassava (cv. Bao) grown at the Namulonge Research Station, near Kampala, in Uganda. These data show clearly that the densities of *B. tabaci* adults and nymphs breeding on symptomatic diseased cassava leaves are significantly greater than on the

![Graph showing population growth of *B. tabaci* on virus-infected and uninfected host plant species.](image)

Fig 3. Population growth of African cassava *B. tabaci* on virus-infected and uninfected host plant species.
leaves of healthy plants (Fig. 4), reflecting the greater attractiveness and suitability of these plants to whitefly. In addition, virus content of leaves has also been shown to increase with increasing symptom severity (Fargette et al., 1987), which suggests that these viruses have probably evolved mechanisms to overcome the cassava plant’s defences against B. tabaci, thus, aiding their acquisition and subsequent inoculation.

### TABLE III

**Repeated Measurements Analysis of Variance for Ln (B. tabaci Nymph Count + 1) and Ln (B. tabaci Adult Count + 1) at 21 and 42 Days after Groups of Five Male and Five Female Newly Emerged Adults Per Plant Were Allowed to Colonise Cassava Plants**

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Wald statistic</th>
<th>d.f.</th>
<th>Wald/d.f.</th>
<th>Chi (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nymphs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>1301.89</td>
<td>2</td>
<td>650.95</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Plant health&lt;sup&gt;a&lt;/sup&gt;</td>
<td>44.53</td>
<td>1</td>
<td>44.53</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time × plant health</td>
<td>65.84</td>
<td>2</td>
<td>32.92</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Adults</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>83.70</td>
<td>2</td>
<td>41.85</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Plant health&lt;sup&gt;a&lt;/sup&gt;</td>
<td>32.53</td>
<td>1</td>
<td>32.53</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time × plant health</td>
<td>21.79</td>
<td>2</td>
<td>10.89</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<sup>a</sup> Plants either remained healthy or became infected with EACMV-UG[Nam].

### TABLE IV

**The Quantities of Four Amino Acids in Healthy (n = 26) and Infected Cassava (n = 13)**

<table>
<thead>
<tr>
<th>Amino acid&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Healthy cassava mg/g dry leaf weight ± SE</th>
<th>CMD-affected cassava mg/g dry leaf weight ± SE</th>
<th>d.f.</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asparagine</td>
<td>0.028 ± 0.003</td>
<td>0.136 ± 0.045</td>
<td>37</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Glutamine</td>
<td>0.135 ± 0.021</td>
<td>0.293 ± 0.101</td>
<td>37</td>
<td>=0.045</td>
</tr>
<tr>
<td>Tryptophan</td>
<td>0.037 ± 0.005</td>
<td>0.061 ± 0.009</td>
<td>37</td>
<td>=0.018</td>
</tr>
<tr>
<td>Tyrosine</td>
<td>0.039 ± 0.007</td>
<td>0.070 ± 0.016</td>
<td>37</td>
<td>=0.043</td>
</tr>
</tbody>
</table>

<sup>a</sup> Quantities of the 16 other amino acids tested were not significantly different. Data for each amino acid were analysed separately as a two factor ANOVA (healthy or infected, with or without B. tabaci). In the above four cases, both the effect of B. tabaci and the interaction effect (B. tabaci × plant-health status) were not significant and therefore data for plant-health status treatments were pooled.
Fig 4. Densities of cassava B. tabaci on plants in the field expressing different levels of severity in CMD symptoms.
V. CONCLUDING REMARKS

Previous publications and the new experimental data presented here provide an increasing body of evidence to suggest that vector–virus–host plant interactions are both widespread and complex, and that they play an important role in the mechanisms driving many important arthropod-borne plant virus disease epidemics. Of the large range of potential interactions, the one that best explains the increased populations of viruliferous vectors that are associated with some plant virus epidemics is the greater population growth rate of vectors colonising and feeding on virus-infected compared with healthy host plants.

It has also often been assumed that an increase in vector numbers, at least initially, should result in an increased incidence of disease, although, for various reasons, this has not always been easy to demonstrate in the field. Nevertheless, there are several examples of significant correlations particularly between aphid numbers and virus spread within different pathosystems and from various parts of the world (Alper and Loebenstein, 1966; Dickenson et al., 1956; Schwarz, 1965; Watson and Healy, 1953).

For B. tabaci, field experiments by several researchers have shown that CMD incidence was correlated positively with the size of the population of adult vectors about one month earlier. This delay corresponds with the expected latent period between infection and CMD symptom expression (Fargette et al., 1990, 1994; Fishpool et al., 1995; Leuschner, 1978; Otim-Nape, 1993). After crops reached 5 months old, however, the relationship was less clear probably because the crop became less attractive to whiteflies and less susceptible to infection (Colvin et al., 1998; Fishpool et al., 1995; Robertson, 1987).

Fauquet et al. (1988) found less consistent relationships between the size of the B. tabaci population and subsequent disease spread and attributed this to differences in the potency, prevalence and distribution of nearby sources of infection. High-disease incidences occurred, however, even in fields several kilometres from infected cassava, which suggested movement of viruliferous vectors over considerable distances (Fauquet et al., 1988). In more recent studies in Uganda, the extent and proximity of sources of infection was shown to be important (Legg and Ogwal, 1998), and at the epidemic front there was a highly significant association between the size of the cassava B. tabaci population and CMD spread (Colvin et al., 2004).

Swenson (1968) noted that an effective relationship between a virus and its vector(s) necessitates a dependable means of transfer to new
susceptible hosts. For several of the pathosystems described here, this is achieved partially through the increased vector population growth rates on virus-infected plants which, in turn, leads to crowding and the production of migrant viruliferous vectors (Byrne and Blackmer, 1996; Lees, 1966; Zhang et al., 2000). This mechanism is clearly an important component driving the CMD pandemic in East and Central Africa, where cassava plants that expressed the most severe symptoms generally contained both ACMV and EACMV-UG and generated large populations of viruliferous B. tabaci adults (Colvin et al., 2004). We suggest that it is highly probable that the improved performance of the B. tabaci populations on infected cassava is the result of the higher-amino acid concentrations in the phloem sap, which is generally poor in these constituents that are essential for the growth of other phloem-feeding species such as aphids (Dixon, 1985).

The severe chlorosis associated with the top leaves of these diseased cassava plants also reduces the acceptable areas of green tissue available for B. tabaci feeding and development. These two effects result in significantly increased population densities on symptomatic plants that, in turn, promote the movement of viruliferous adults and thus enhance disease spread.

The combination of ACMV and EACMV-UG in cassava plants clearly produces a range of interrelated effects that are highly efficient at ensuring the continuing spread of both the viruses and the vector. This mechanism can explain how the pandemic is effectively ‘self-perpetuating’ and why physical barriers, such as rivers and mountains, or seasonal reductions in B. tabaci populations present no apparent obstacle to its continuing progress (Otim-Nape et al., 2000). All that is required is the arrival of adult cassava B. tabaci, that are carrying EACMV-UG, into a new area where susceptible cassava varieties are being grown and where ACMV is already endemic.

The concept that the cassava viruses act to overcome the plant’s defences against the B. tabaci may also explain the presence of the so called invader B. tabaci population on cassava during the CMD pandemic in Uganda from 1997 to 1999 (Legg et al., 2002). Rather than being a cassava B. tabaci population with inherently superior fitness attributes, the available data suggest that it is more likely to be an indigenous population whose normal niche probably includes alternative plant species to cassava as its preferred hosts. The breakdown in vector resistance in the severely diseased cassava plants may, therefore, have temporarily increased the acceptability of cassava to this population, thereby enabling colonisation to take place.
A mutually beneficial interaction between viruses and *B. tabaci* populations is clearly also present in the ToLCV-pathosystem in South India. Data presented here show that significantly greater *B. tabaci* populations were generated on several ToLCV-infected weed species, which occur extremely commonly throughout most of South India and often cover much of the open ground around tomato fields (Ramappa *et al.*, 1998). This mechanism explains the rapid build-up of a large reservoir of ToLCV and *B. tabaci* during the summer season and why, when tomato was planted in a new area where it had not been grown previously, ToLCV-infected plants still appeared very rapidly and the final incidence was extremely high (Ramappa *et al.*, 1998).

In 1999 in South India, an unusually severe epidemic of ToLCV disease occurred in Karnataka state, which resulted in the complete failure of the tomato crop. This was associated with strikingly high *B. tabaci* populations exceeding 1000 adults per tomato plant (Banks *et al.*, 2001). The population associated with the ToLCV epidemic was identified subsequently by mtCOI gene sequence analysis and by the squash silver leaf assay as biotype B of *B. tabaci* (Banks *et al.*, 2001; Rekha *et al.*, 2005). This population had probably been introduced into southern India through the importation of horticultural material. Since then, the B biotype has spread rapidly throughout Karnataka, and into the neighbouring states of Andhra Pradesh and Tamil Nadu, undoubtedly assisted by the activities of nurserymen raising tomato seedlings who transport these to considerable distances. The arrival of the B biotype into new areas has also been associated with the appearance of new epidemics of *B. tabaci*-borne viruses causing cotton leaf curl, okra yellow vein mosaic and pumpkin yellow vein mosaic diseases. Completely new diseases caused by geminiviruses, such as potato leaf curl, have also appeared (Maruthi *et al.*, 2003, 2004b; Rekha *et al.*, 2005). However, the virus–vector–host plant interactions driving these new epidemics remain unknown, and research in this area represents a considerable opportunity and challenge for the future.

Certain aphid species alight on both host and non-host plants and host selection occurs only after arrival (Kennedy *et al.*, 1959a,b). Migratory behaviour takes precedence over host-plant finding behaviour and alighting on a host plant does not usually terminate flight (see Reynolds *et al.*, this volume, pp. 453–517). The aphids stay longer on host plants, feed and reproduce, and so their accumulation on them is mostly a result of differential departure rates, rather than preferential alightment (Kennedy *et al.*, 1959a,b). The non-discriminatory alighting and probing of aphids, the initial dominance of migratory over host-finding behaviour, and the intensity and duration of aphid
dispersal are all activities that facilitate the spread of non-persistent viruses (Kennedy, 1960). These factors ensure a dependable means of virus spread even into crops that have no colonising vector species as occurs, for example, with BYMV (Swenson, 1957; Swenson and Nelson, 1959) and melon mosaic viruses (Dickenson et al., 1949). This phenomenon may also occur with other vectors which make transitory visits to the crop, for example, leafhoppers that transmit MSV in the forest regions of Nigeria were considered to come from infection sources located outside the field and further spread occurred as the result of the transient leafhoppers spreading the virus from plant-to-plant within the field (Asanzi, 1991).

More studies on the aphid R. padi, which along with 25 other aphid species in North America transmits BYDV in a persistent-circulative manner, showed that they respond to the volatile cues emitted by infected plants and settle preferentially on them (Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004b). Behavioural responses that influence vector distribution and movement are particularly important because of their potential effect on the spread of virus in the field. Such responses also include the enhanced visual attraction of alate vectors to the yellowed leaves of BYDV-infected cereals (Ajayi and Dewar, 1983).

For cassava B. tabaci, field data show clearly that adults colonise severely symptomatic cassava plants most rapidly (Omongo, 2003), although it remains to be discovered which cues are important in this effect. In addition, it is unknown whether the yellow mosaic symptoms caused by many geminiviruses increases the attractiveness of affected plants to the B. tabaci biotypes that transmit the associated geminiviruses.

The data presented here have implications for the management of cassava mosaic geminiviruses, which is the subject of a review by Thresh and Cooter (2005). For this pathosystem, the main reservoirs of both the vector and the viruses are the cassava crop (Burban et al., 1992) and, in addition, our results suggest that infected plants are attractive to B. tabaci for oviposition, growth and development. Area-wide phytosanitation methods that remove these plants as soon as they begin to express symptoms (roguing) should, therefore, reduce the inoculum pressure to a disproportionately large degree. In the previous CMD epidemics in Uganda in the 1940s and 1960s, removal of infected plants was enforced by law, which together with resistant varieties successfully reduced losses due to CMD (Jameson, 1964). Currently, the inoculum pressure at, and immediately after, the pandemic front is so high that a control policy of only roguing is impractical, as it would result in the removal of
all but the most resistant varieties. Any roguing policy, therefore, clearly needs to be combined with the supply to farmers of healthy stocks of CMD-resistant planting material.

The situation is further complicated because many of the new cassava varieties are tolerant to disease in the sense that, even though they become infected, they still produce what farmers consider to be a satisfactory yield. Farmers, therefore, are understandably unwilling to remove and destroy these plants, and so a reservoir of virus and *B. tabaci* is maintained. This situation is probably one of the main reasons that cassava *B. tabaci* populations have remained high in the post-epidemic regions of Uganda.

Other recommendations made in the 1960s (Jameson, 1964) still hold, in that varieties that keep for more than one year in the ground should probably be avoided as these become reservoirs of infection. However, varieties with this characteristic are also valued as an important food reserve in the event that the rains fail, and so farmers may be reluctant to stop growing them. The emphasis in future control methods, however, should remain on phytosanitary measures that reduce both virus incidence and vector numbers (Colvin et al., 2003). Strict quarantine regulations on the movement of plant material between countries should also be enforced.

As well as the dissemination and distribution of tolerant cassava varieties that may be highly susceptible to *B. tabaci* even when virus-free, a potentially valuable strategy for tackling the breakdown in *B. tabaci* resistance associated with the CMD pandemic in Africa is to breed cassava varieties that incorporate both virus and vector resistance originating from different sources. Parental material with high levels of *B. tabaci* resistance have been identified in Africa (Omongo and Colvin, unpublished data) and in South America (Bellotti, 2002; Bellotti and Arias, 2001), and an international project has been initiated to produce new varieties with agronomic traits that are acceptable to farmers.

The evidence from the literature and the data presented here for various *B. tabaci* populations and viruses provides an increasing body of evidence demonstrating that co-adaptation and co-evolution between viruses and their arthropod vectors is even more prevalent than thought previously and that it has created several mechanisms that drive arthropod-borne plant virus disease epidemics.

Research was carried out to investigate whether or not various viral infections affected aphid performance differently and, if so, whether any pattern was apparent according to the type of virus-vector relationship. Plants infected with PLRV, a circulative virus highly
dependent on *M. persicae* for dispersal and transmission, caused the greatest intrinsic rate of increase (*r_* m) in the aphid population. Plants infected with *Potato virus Y* (Genus: *Potyvirus*; Family: *Potyviridae*), a non-circulative virus less dependent on *M. persicae* for dispersal, caused intermediate *r_* m values and plants infected with *Potato virus X* (Genus: *Potexvirus*), a non-vectored virus independent of *M. persicae*, were least suitable hosts and these aphid colonies produced the lowest *r_* m values (Castle and Berger, 1993). This work and the additional examples described here suggest that improved vector fitness may be found most commonly where there is a close relationship between the virus and the vector, and the virus is transmitted in a persistent or semi-persistent manner. This concept is consistent with the cassava mosaic pathosystem in both Africa and India, where the cassava whitefly populations are specialised on cassava and do not interbreed with sympatric *B. tabaci* populations on other host species nearby (Maruthi *et al*., 2001, 2004a).

The continued survival of a vector-dependent plant virus requires that the number of infected plants should never fall so low that transfer of virus to another susceptible host becomes unlikely (Swenson, 1968). The characteristics that determine transfer are therefore undoubtedly subject to selective pressures and so vectors must have a consistent relationship with their host plant(s) even if only to the extent of alighting and probing by a non-colonising species during migration. In such cases, the virus may be transmitted by several host plant-specific aphid species, but the virus itself may not be host-plant specific. Also, the fitness and abundance of diseased plants can be severely reduced by infection and their elimination is obviously not advantageous to the vector. It has been suggested that this negative interaction may explain the lack of specificity in some systems (Barbosa, 1991).

The pathosystems described in this article all involve interactions between plant, viruses and their vector(s) that result in complex and varied disease cycles of huge economic significance. BYDV, for instance, is recognised as one of the most serious viral diseases of wheat, barley, oats, grasses and other cereal crops throughout the world (Jedlinski, 1981) and the CMD pandemic continues to contribute to the food insecurity in a large region of SSA (Legg *et al*., this volume, pp. 355–418). There is, therefore, a continuing need to study these interactions both to gain a better understanding of the level of co-adaptation and co-evolution in the pathosystems and, subsequently, to use this information to develop rational, practical, improved and sustainable pest and disease management strategies.
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