The epidemiology of plant virus diseases

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INTRODUCTION

Plant viruses are obligate parasites and few can survive for long outside living tissue. The associations between viruses and their hosts are therefore particularly complex and this is evident from work on many aspects of virus spread. Nevertheless, only a small proportion of current research is devoted to epidemiological studies, despite their intrinsic interest and their relevance in developing measures for controlling the many virus diseases of major economic importance.

General features of the epidemiology of plant virus diseases are considered in this paper within a broad ecological context. The emphasis is on interactions between viruses and their hosts in which fungal or animal vectors are frequently involved. Such an approach is justified despite the limitations and deficiencies of the available information.

Virus diseases of potato, sugar-beet and a few other important crops of countries in temperate regions with well-developed systems of agriculture have received disproportionate attention. Until recently the main work in the tropics and sub-tropics has been on export crops such as cacao, citrus and groundnut. In all areas there is only limited information on viruses of natural vegetation and on the spread of those that do not cause obvious symptoms. Moreover, clover phyllody and several other important diseases for long attributed to viruses are now associated with mycoplasmas or other types of micro-organisms. The aetiology and/or means of spread of many other diseases are unknown. For these reasons the literature may be misleading and must be interpreted cautiously.

MEANS OF SPREAD

An effective means of spread is essential to ensure that the number of infected plants does not become so low that transmission to other susceptible hosts is unlikely. The critical level depends on the overall effectiveness of the transmission process and on the number and distribution of the host plants as well as their susceptibility, size, longevity and potency as sources of inoculum. As with other types of pathogen,

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Table 1. Some important viruses of crops and their mode of spread.

CONTACT (C)	
Potato spindle tuber viroid* (66)	Cucumber pale fruit viroid
Barley stripe mosaic* (68)	Chrysanthemum stunt viroid
Carnation mottle (7)	Tobacco mosaic (151)
Carnation ringspot (21)	Tomato mosaic* (156)
POLLEN (P)	
Black raspberry latent* (106)	Prunus necrotic ringspot* (5)
Prune dwarf* (19)	Raspberry bushy dwarf* (165)
FUNGI (Fu)	
Beet necrotic yellow vein (144)	Satellite (15)
Cucumber necrosis (82)	Tobacco necrosis (14)
Potato mop-top (138)	Wheat (soil-borne) mosaic (77)
NEMATODES (Nematoda: Ne)	
Arabis mosaic* (16)	Raspberry ringspot* (6)
Cherry rasp leaf* (159)	Tobacco rattle* (12)
Grapevine fanleaf* (28)	Tobacco ringspot* (17)
MITES (Acarina: Ac)	
Wheat streak mosaic (48)	Potato virus Y (37)
INSECTS	
Mealybugs (Coccidae: Cc)	
Cacao swollen shoot (10)	
Thrips (Thysanoptera: Th)	
Tomato spotted wilt* (39)	Tobacco ringspot* (17)
Beetles (Coleoptera: Cl)	
Bean pod mottle (108)	Turnip crinkle (109)
Broad bean stain* (29)	Rice yellow mottle (149)
Echtes Ackerbohnen mosaik* (20)	Squash mosaic* (43)
Aphids (Aphididae: Ap) non-persistent	
Alfalfa mosaic* (46)	Pea seed-borne mosaic* (146)
Bean yellow mosaic* (40)	Potato aucuba mosaic (98)
Beet mosaic (53)	Potato virus Y (37)
Cucumber mosaic* (1)	Plum pox (70)
Lettuce mosaic* (9)	Sugarcane mosaic* (88)
Peanut mottle* (141)	Watermelon mosaic (63)
Aphids (Aphididae: Ap) persistent/semi-persistent	
Barley yellow dwarf (32)	Citrus tristeza (33)
Beet western yellows (89)	Lettuce necrotic yellows (26)
Beet yellows (13)	Potato leafroll (36)
Cauliflower mosaic (24)	Sowthistle yellow vein (62)
Whiteflies (Aleyrodidae: Al)	
Cotton leaf curl	Bean golden yellow mosaic
Leafhoppers/planthoppers (Auchenorrhyncha: Au)	
Beet curly top	Potato yellow dwarf (35)
Maize rough dwarf (72)	Rice dwarf (102)
Maize streak (133)	Wheat striate mosaic (99)

*Also seed-borne (S) in at least some host species.

various strategies are exploited and the principal means of virus spread are by direct contact, through pollen and/or seed and by animal or fungal vectors. However, many important crop plants are grown as vegetatively propagated perennials and the extensive traffic in plant material provides an additional means of disseminating viruses within and between countries. Perpetuation in this way and by budding or other grafting operations provides a means of long-term survival that is completely independent of a continuing sequence of transmission between plants by natural means. Viruses are also transported by man into entirely new areas and over distances greater than achieved by vectors.

Spread by contact

The limited persistence of most viruses outside living cells restricts the opportunities for transmission by direct contact. Additional constraints are the immobility of rooted plants, their resistance to infection and the lack of suitable entry points. Nevertheless, several important viruses and viroids are spread by handling, pruning or by contact with infected plants, debris or contaminated implements (Table 1). The greatest opportunities for contact spread occur in glasshouse crops such as cucumber, tomato, chrysanthemum and carnation that are handled repeatedly in routine cultural operations. This facilitates the spread of stable, highly infectious viruses and viroids that are much less common in equivalent outdoor crops, where insect-borne viruses predominate.

Seed and pollen transmission

Seed transmission of viruses was initially considered to be a rare phenomenon, but it is now known to be an important feature of many viruses in at least some of their hosts. Transmission occurs by way of the ovule and sometimes by pollen (Shepherd, 1972). There are also instances of pollen infecting the female parent and this is the highly effective mode of spread of four important viruses in temperate fruit crops (Table 1).

All seed-borne viruses have additional means of spread and seed transmission has an important and sometimes crucial role in the epidemiology of viruses otherwise spread by contact, pollen or by certain arthropods (Table 1). Indeed, viruses can be carried further and survive longer in seeds than in pollen or in vectors. Seeds also provide an effective means of survival between growing seasons or throughout the sometimes prolonged interval between plantings of susceptible crops.

That pollen and seed transmission are not encountered more frequently suggests that there are strong selection pressures against these fea-

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The numbers in parentheses throughout this paper refer to the *C.M.I./A.A.B. Descriptions of Plant Viruses*, which provide further information on the viruses and viroids cited. Abbreviations for vectors, also in parentheses, are explained in Table 1.

tures. Certainly there are only limited opportunities for spread between species, or within species that are normally self-pollinated, and host fertility may be decreased. For example, prune dwarf and prunus necrotic ringspot viruses (P, S) have such drastic effects that this restricts spread by pollen and explains why infection does not occur even more widely in long-established varieties of susceptible *Prunus* spp. (Marenaud & Saunier, 1974).

Complex interactions between pathogen virulence and host response are apparent from studies on barley stripe mosaic virus (C, P, S) which is only perpetuated in seed stocks of varieties that are not so sensitive that few seeds are produced, or so resistant that few become infected (Timian, 1974).

Transmission by vectors

Certain root-infecting fungi and many phytophagous animals transmit viruses as they move between plants. This is the principal means of transmission and compensates for the immobility of rooted plants and the limited opportunities for spread between species by pollen or between sites by contact.

Transmission by nematodes and by fungi has been confirmed quite recently and further work may extend the range of known vectors to other taxonomic groups. There are mainly unconfirmed reports of a few viruses including potato virus Y (Ac, Ap) and tobacco ringspot (Th, Ne) being transmitted by vectors of more than one category (Table 1). Otherwise, vector-borne viruses are transmitted by one or more closely related species. Even within the same group of vectors there are different mechanisms of transmission with differing degrees of specificity. Some viruses are entirely dependent for their transmission (98), or multiplication (15), on the presence of unrelated viruses (Murant, this volume). With tobacco rattle (Ne) and many other viruses, particles of more than one type are necessary for infection (46, 108, 185). This increases the opportunities for variants to appear but places additional demands on the effectiveness of the transmission process (Harrison, 1977 and this volume).

The various mechanisms of transmission and their significance in epidemiology are reviewed elsewhere (Thresh, 1974a). Each system is effective for quite different reasons, having some features that facilitate spread and others that lead to unreliability. This is because rapid transmission in short probes and long persistence in the vector appear to be mutually exclusive attributes. An ability to multiply within vectors and pass to their progeny facilitates the persistence of some aphid-borne or hopper-borne viruses and enhances survival when plant hosts are not readily available. However, results obtained with maize rough dwarf (Au) and other viruses illustrate how the fertility and longevity of vectors may be impaired (Harpaz, 1972), and this may explain why multiplication in vectors seems to be uncommon. It may be restricted to viruses that primarily infect arthropods and which have only recently become adapted to plants (Tinsley, 1973). Other viruses including beet yellows (Ap) and beet mosaic (Ap) can have important direct or indirect effects on vectors in which they do not multiply. Various harmful and beneficial effects have been reported and this emphasizes the importance of considering the full implications of the often complex interactions between viruses, vectors and host plants (Kennedy, 1951).

Nematode and fungal vectors Viruses spread by nematodes or fungi are not usually dispersed quickly or far. However, an important compensating feature is that they are retained, sometimes for long intervals, between plantings of susceptible crops. The soil provides a stable environment and once vectors are established at a site they tend to persist, populations fluctuating less rapidly than those of arthropods above ground. Site, previous cropping history, soil moisture and soil temperature influence profoundly the incidence and spread of diseases. Hop nettlehead and other diseases of plantation crops caused by nematode-borne viruses often occur in slow-spreading patches that reappear at the same sites in successive plantings (Thresh & Pitcher, this volume).

There are differences in the behaviour of the two main types of virus with fungal vectors (Harrison, 1977). Tobacco necrosis and other viruses (15, 82) transported externally on the zoospores of chytrids are unusual in often being restricted to roots. New infections occur when virus particles that are released into the soil are introduced into roots after becoming attached to motile zoospores. Wheat (soil-borne) mosaic and other viruses (138, 144) that are carried within the resting spores of plasmodiophoromycetes tend to have a restricted host range. Nevertheless, they are widely distributed as they persist for long periods in durable resting spores that can be blown or carried in soil particles, debris or infested plants.

Arthropod vectors The arthropod vectors of plant viruses are a diverse assemblage (Table 1), with great differences between the various groups in life cycle, behaviour and activity. Whitefly larvae are virtually immobile and, apart from wind-borne mealybugs and perhaps mites, the immature forms of many other vectors seldom move far. Those able to transmit can only spread viruses to nearby plants reached by walking, crawling or hopping. Adults and especially winged forms also spread virus locally, but there is great variation in their ability to travel further. With aphids and several other types of vector there are numerous instances of alary or behavioural polymorphism with different forms of contrasting mobility (Table 2). The morphology, itinerant behaviour and delayed reproduction of specialized migrants are features that facilitate dispersal. Their main ecological role is to reach and colonize fresh habitats that are exploited by subsequent generations of less mobile forms that are well adapted for early and rapid reproduction (Johnson, 1969). Migratory behaviour is related to the type of habitat exploited and is a particular feature of arthropods that colonize short-lived hosts or otherwise ephemeral environments. This facilitates the periodic transfer of a portion of the population to fresh sites before the original ones disappear (Southwood, 1962). Migration also ensures that viruses are carried frequently and far by vectors that are infective on take-off, or that acquire viruses whilst dispersing (Thresh, 1974a).

Table 2. Arthropod vector groups with winged (W) or wingless (-) adult forms of contrasting mobility.

Group	Virus example	"Colonizers"	"Exploiters"
Mites*	Wheat streak mosaic	Wind-borne (-)	Sedentary (-)
Whiteflies	Cotton leaf curl	Active forms (W)	Less active (W)
Beetles	Squash mosaic	Active forms (W)	Less active (W)
Leafhoppers	Maize streak	Long fliers (W)	Short fliers (W)
Planthoppers	Maize rough dwarf	Macropterae (W)	Brachypterae (±W)
Aphids	Barley yellow dwarf	Alatae (W)	Apterae (-)

*The mealybug vectors of cacao swollen shoot virus behave similarly but the main dispersal is by first-instar nymphs

The rapidity and efficiency with which new plantings of short-lived arable crops are colonized by vectors determines the initial appearance and subsequent spread of viruses such as beet yellows (Ap) and groundnut rosette (Ap). Migrants usually appear at particular times, when populations are crowded or when seasonal or other conditions become unfavourable, especially when the infested source plants are maturing and beginning to senesce or die. There are many examples of spread by mites, aphids, beetles or hoppers moving to young plantings from ripening cereal crops or wild grasses (32, 48, 72, 133, 149).

An overall assessment

In assessing the overall effectiveness of the different methods of spread it is important to consider the distances involved and the likelihood of inoculum reaching suitable host plants. Local spread by contact or by vectors of limited mobility can lead to a thorough exploitation of the immediate habitat. There is little loss of inoculum, except to plants already infected. However, spread is circumscribed and occurs mainly to plants growing under the same conditions and in a similar phase of development. There are only limited opportunities for fresh outbreaks to be started in other plantings or in entirely new areas.

The dispersal of viruses over considerable distances by mobile vectors, pollen, or fungal spores has quite different consequences. There are ample opportunities for spread to new areas, but much of the inoculum may be carried to unfavourable habitats or beyond the range of susceptible hosts. A dual strategy of dispersal has obvious survival value. This accounts for the importance of specialized arthropod vectors that produce variable proportions of migrant and less active forms according to environmental or other circumstances and so provide an effective system by which viruses are spread locally and periodically over greater distances.

Many of the viruses with less versatile vectors have two distinct but complementary methods of spread that together produce an adequate distribution of inoculum. For example, wheat (soil-borne) mosaic and other viruses spread locally by fungal zoospores are also carried far in resting spores; dissemination in seeds is a feature of many nepoviruses (Ne) and non-persistent viruses (Ap) (Table 1) that are seldom dispersed far by animal vectors (Murant, 1970). Some of the complexities involved are apparent from recent studies on the divided genome of raspberry ringspot virus in which the two main mechanisms of survival and spread through seed and by nematodes are determined by different pieces of RNA that are subject to separate selection pressures in nature (Harrison, 1977 and this volume).

SOURCES OF INFECTION

The primary foci from which spread occurs can be similar or unrelated species of wild or cultivated plants occurring within or outside plantings. Long-lived hosts and others that survive between growing seasons are particularly important in maintaining the cycle of infection in annual crops. For example, perennial weeds in adjacent uncultivated land are the overwintering hosts of viruses causing prevalent diseases of legumes (146), cucurbits (63) and sweet pepper (37). In lettuce (9), brassica (24), cereal (48) and other crops (156) grown in overlapping sequence throughout the year there is extensive spread from old to new plantings. Aphids transmit peanut mottle virus to soybean (Demski, 1975) and there are many other examples of spread between unrelated crops.

Infected weeds and other sources that occur within crops are a major hazard because of the ease with which spread can occur to nearby plants. This explains the importance of infected material of sugar beet (13, 89), potato (36, 37) and many other crops surviving from previous plantings, especially after mild winters. There are also numerous instances of viruses being introduced in seeds (Table 1) or vegetative planting material and there are obvious agricultural advantages in using certified virus-free stocks. It may be more difficult to avoid common weeds such as *Stellaria media* (L.) Vill. in which cucumber mosaic and several nematode-borne viruses are seed-transmitted.

SPATIAL PATTERNS OF SPREAD

Virus diseases are seldom uniformly distributed in crops and tend to be concentrated in certain plantings or areas where conditions favour spread from primary foci. Observations on spatial patterns of spread are, therefore, important in identifying sources and in developing control measures. It is necessary to determine whether the initial spread is from inside or outside the crop and whether any influx is followed by secondary spread to neighbouring plants.

Spread from sources outside the crop

Spread into crops from outside sources is a feature of many virus diseases. A few, of which tomato spotted wilt (Th) and lettuce necrotic yellows (Ap) are the best-known examples, are spread exclusively in this way. Many others spread into and also within plantings, the relative importance of the two types of spread depending on the number and distribution of primary foci. Viruses such as beet curly top (Au) that persist in mobile vectors can be carried far to initiate new outbreaks, even at sites where local sources are avoided by suitable measures of crop hygiene (Fig. 1, Au). The spread of non-persistent viruses is much more circumscribed (Fig. 1, Ap) and this facilitates their control by isolation.

Infection due to incoming vectors tends to be greatest around the perimeter of plantings and many "edge" or "hedge" effects have been reported (Thresh, 1976). There is no single explanation and various physical and biological factors are involved. Wind-borne mite, aphid, whitefly or hopper vectors often alight or breed preferentially on the peripheral plants, especially to leeward of buildings, hedgerows, trees and windbreaks (Lewis, 1969). In many other instances spread is closely related to breeding or overwintering sites of the vector or to sources of infection in adjoining crops or natural vegetation.

Spread from sources inside the crop

Local spread from foci within crops accounts for the more or less discrete, expanding patches of infection encountered in studies on many diseases. Sources introduced in the planting material are likely to be randomly distributed unless the situation is complicated by the use of different batches. There is a less uniform distribution of primary foci where these are due to incoming inoculum or to weeds or crop residues surviving from previous plantings. For example, potato mop-top (Fu) and yellowing viruses of sugar beet (Ap) tend to be particularly prevalent at or alongside sites where crops are stored.

Disease gradients

The general tendency for the incidence of virus diseases to decrease with increasing distance from the source accounts for the gradients of infection that are such a common feature in many crops (Thresh, 1976). There are great differences between diseases in the amount and distance of spread, but many gradients have the same curvilinear shape (Fig. 1). The incidence of disease usually decreases steeply near the source and less steeply at greater distances to reach zero or a low background level. However, gradients may be flattened near the source due to multiple infection (Gregory, 1948). They also tend to become flatter with time as secondary spread occurs, or following an increase in the number or activity of vectors.

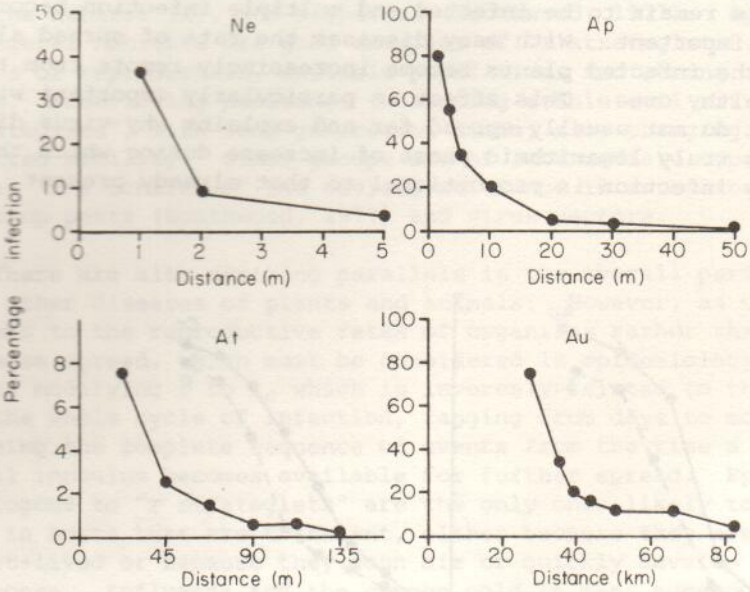

Figure 1. Disease gradients due to spread by nematodes (Ne) of the hop strain of arabis mosaic virus (Thresh, unpublished), aphids (Ap) of peanut mottle virus (Demski, 1975), aphids (At) of citrus tristeza virus (Bar-Joseph *et al.*, 1974), leafhoppers (Au) of beet curly top virus (Annand *et al.*, 1932).

The size and spatial configuration of the source contribute to the rate of dilution of inoculum with distance but gradients are mainly influenced by the behaviour of vectors. Nematodes seldom move far and this leads to steep gradients and distinct patches of disease that progress slowly as a solid advancing front of infection in crops such as hop (Fig. 1, Ne). Gradients due to winged aphids and other wind-borne vectors are much shallower, especially downwind, as recorded for the spread of bean yellow mosaic virus (40) from clover to adjacent bean plantings (Hampton, 1967). Persistent viruses such as barley yellow dwarf (Ap) can be carried far, leading to widely scattered primary foci from which secondary spread occurs by subsequent local movement of incoming migrants or their progeny.

TEMPORAL PATTERNS OF SPREAD

Curves of disease progress with time tend to be sigmoid (Fig. 2), although within the same overall pattern there are great differences between sites, seasons and diseases in the onset, rate, duration and total amounts of spread (Thresh, 1974b). After the first appearance of disease there is usually a period of rapid increase in the cumulative total of infected plants. The rate of increase then declines as weather or other conditions become unfavourable or because progressively fewer healthy plants remain to be infected and multiple infection becomes increasingly important. With many diseases the rate of spread also declines as the infected plants become increasingly remote from the remaining healthy ones. This effect is particularly important with diseases that do not usually spread far and explains why virus diseases seldom have a truly logarithmic phase of increase during which the amount of new infection is proportional to that already present.

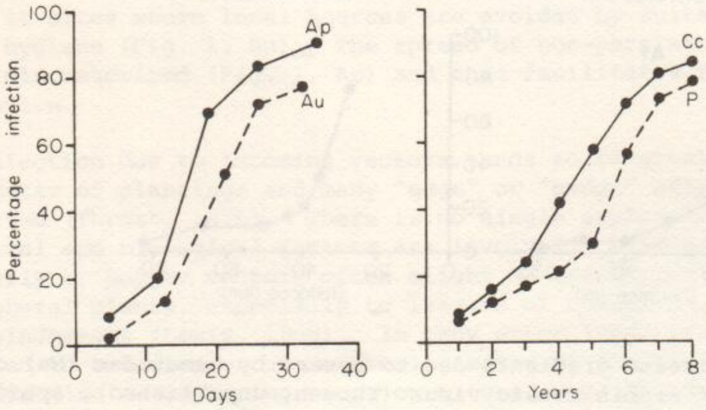

Figure 2. Progress of disease due to spread by aphids (Ap) of cucumber mosaic virus (Loebenstein *et al.*, 1966), leafhoppers (Au) of beet curly top virus (Annand *et al.*, 1932), mealybugs (Cc) of a Trinidad cacao virus (Dale, 1953), pollen (P) of prune dwarf and prunus necrotic ringspot viruses (Smith *et al.*, 1977).

A slow rate of spread will suffice to maintain infection in perennial crops, many of which are propagated vegetatively. Spread is usually slowest in woody perennials with a time-scale measured in years (Fig. 2, Cc, P). By contrast, virus diseases of market garden, arable and other short-lived crops often appear early and spread rapidly so that a substantial proportion of the stand is affected within a few weeks or months (Fig. 2, Ap, Au). Young plants are particularly vulnerable to infection and are usually those most seriously affected by viruses. There are thus advantages in delaying the onset of disease by using healthy planting material, adequate isolation or other measures of crop hygiene. The alternative and often complementary approach is to decrease rates of spread by using resistant varieties, pesticides or repellent materials that prevent vectors alighting.

STRATEGIES OF VIRUS SPREAD

Ecologists have recently been stressing the interrelationships between the maximum intrinsic rate of reproduction (r_{max}), the carrying capacity of the habitat (*K*), and dispersal (Southwood, 1977). Aphids exploiting ephemeral habitats are good examples of "*r* strategists", having high rates of reproduction, unstable populations and strong migratory tendencies. Free-living nematodes inhabiting stable soil situations are "*K* strategists", with long generation times, slow-changing populations and limited mobility. Other groups are of intermediate type and the concept of an "*r*-*K* continuum" has obvious relevance in assessing the behaviour of crop pests (Southwood, 1977) and virus vectors.

There are also striking parallels in the overall performance of virus and other diseases of plants and animals. However, as used previously *r* refers to the reproductive rates of organisms rather than to rates of disease spread, which must be considered in epidemiology. This necessitates modifying *r* to r_c which is inversely related to the mean duration of the whole cycle of infection, ranging from days to months and comprising the complete sequence of events from the time a host is infected until inoculum becomes available for further spread. Epidemic diseases analogous to "*r* strategists" are the only ones likely to become prevalent in hosts that are transient, either because they are inherently short-lived or because they soon die or quickly develop an immune response. Influenza and the common cold in man, numerous fungal diseases of foliage and many of the virus diseases of annual plants are examples of this type that spread rapidly from a few initial foci of infection. They operate on an opportunist strategy of high transmission rate and rapid invasion of hosts which become infectious within a few days. Infectious hepatitis, many soil-borne fungal diseases and nematode-borne virus diseases of fruit trees and other plantation (6, 16, 28, 159) are examples of diseases behaving as "*K* strategists"; they spread slowly but inexorably over limited distances between long-lived hosts. Intermediate types include citrus tristeza (Ap), cacao swollen shoot (Cc), blackcurrant reversion (Ac) and other diseases of woody plants caused by viruses with arthropod vectors.

The continuous gradation in behaviour from extreme "*r*" to "*K*" forms provides a further illustration of the diversity of plant viruses that makes them such widespread and consistently successful pathogens. Despite the enormous developments made recently in basic and applied virology there has been only limited progress in epidemiology and in devising adequate control measures. In many crops losses continue on an undiminished or even increasing scale with no immediate prospect of major improvement. The need for a broad-based ecological approach to epidemiological problems has long been recognized, but the necessary multi-disciplinary teams have seldom been established and maintained.

There is an urgent need to redress the present imbalance between field and laboratory studies. Additional resources and new initiatives are required if satisfactory progress is to be made.

REFERENCES

Annand P.N., Chamberlin J.C., Henderson C.F. & Waters H.A. (1932) Movements of the beet leafhopper in 1930 in Southern Idaho. *Circular, United States Department of Agriculture* Number 244, 24 pp.

Bar-Joseph M., Loebenstein G. & Oren Y. (1974) Use of electron microscopy in eradication of Tristeza sources recently found in Israel. *Proceedings of the 6th Conference of the Organization of Citrus Virologists* (Ed. by L.G. Weathers & M. Cohen), pp. 83-5. University of California, Berkeley.

Dale W.T. (1953) Further notes on the spread of virus in a field of clonal cacao in Trinidad. *Cocoa Research 1945-1951*, pp. 130-1. Imperial College of Tropical Agriculture, Trinidad.

Demski J.W. (1975) Source and spread of peanut mottle virus in soybean and peanut. *Phytopathology* 65, 917-20.

Gregory P.H. (1948) The multiple-infection transformation. *Annals of Applied Biology* 35, 412-7.

Hampton R.O. (1967) Natural spread of viruses infectious to beans. *Phytopathology* 57, 476-81.

Harpaz I. (1972) *Maize Rough Dwarf.* Israel Universities Press, Jerusalem.

Harrison B.D. (1977) Ecology and control of viruses with soil-inhabiting vectors. *Annual Review of Phytopathology* 15, 331-60.

Johnson C.G. (1969) *Migration and Dispersal of Insects by Flight.* Methuen, London.

Kennedy J.S. (1951) A biological approach to plant viruses. *Nature, London* 168, 890-4.

Lewis T. (1969) Factors affecting primary patterns of infestation. *Annals of Applied Biology* 63, 315-7.

Loebenstein G., Deutsch M., Frankel H. & Sabar Z. (1966) Field tests with oil sprays for the prevention of cucumber mosaic virus in cucumbers. *Phytopathology* 56, 512-6.

Marenaud C. & Saunier R. (1974) Action des virus du type ILAR sur le pollen de l'espèce *Prunus persica*. *Annales de l'Amélioration des Plantes* 24, 169-84.

Murant A.F. (1970) The importance of wild plants in the ecology of nematode-transmitted plant viruses. *Outlook on Agriculture* 6, 114-21.

Shepherd R.J. (1972) Transmission of viruses through seed and pollen. *Principles and Techniques in Plant Virology* (Ed. by C.I. Kado & H.O. Agrawal), pp. 267-92. Van Nostrand Reinhold, New York & London.

Smith P.R., Stubbs L.L. & Challen D.I. (1977) Studies on the epidemiology of peach rosette and decline disease in Victoria. *Australian Journal of Agricultural Research* 28, 103-13.

Southwood T.R.E. (1962) Migration of terrestrial arthropods in relation to habitat. *Biological Reviews* 37, 171-214.

Southwood T.R.E. (1977) The relevance of population dynamic theory to pest status. *Origins of Pest, Parasite, Disease and Weed Problems*

(Ed. by J.M. Cherrett & G.R. Sagar), pp. 35-54. Blackwell Scientific Publications, Oxford.

Thresh J.M. (1974a) Vector relationships and the development of epidemics: the epidemiology of plant viruses. *Phytopathology* 64, 1050-6.

Thresh J.M. (1974b) Temporal patterns of virus spread. *Annual Review of Phytopathology* 12, 111-28.

Thresh J.M. (1976) Gradients of plant virus diseases. *Annals of Applied Biology* 82, 381-406.

Timian R.G. (1974) The range of symbiosis of barley and barley stripe mosaic virus. *Phytopathology* 64, 342-5.

Tinsley T.W. (1973) Origins of vector-borne plant viruses. *Insect/Plant Relationships* (Ed. by H.F. Van Emden), pp. 43-50. Blackwell Scientific Publications, Oxford.