TEMPORAL PATTERNS OF VIRUS SPREAD

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There have been outstanding advances recently in work on the structure and replication of plant viruses, but studies on their spread and control have been relatively neglected, and progress has been limited. Nevertheless, there is an increasing awareness of the prevalence of virus diseases in diverse crops of many countries.

Cacao swollen shoot, cotton leaf curl, hop nettlehead, and many other virus diseases have long caused serious losses, and these continue. Other viruses including plum pox, citrus tristeza, and sugar cane mosaic have increased in importance because of spread into new areas or crops. As a result of detailed surveys or the introduction of new tests, newly detected and in some instances widespread and prevalent viruses have been discovered in maize, rice, apple, and many other hosts.

The well characterized plant viruses are of several distinct morphological types and there is great diversity in their mode of spread. Recent reviews are available on transmission and spread of viruses by pollen and seed (64) and by vectors (20, 52, 56), especially aphids (73, 90), leafhoppers (7, 92), white flies (27), thrips (62), beetles (87), mites (55), nematodes (17, 75), and fungi (76). This paper emphasizes general features in the epidemiology of plant viruses that lead to characteristic curves of disease progress with time.[1] Spatial patterns of infection into and within crops will be considered in a subsequent review.

RATE OF SPREAD

The rate at which a virus spreads between plants varies widely according to the type of virus, crop, environment, and mode of transmission. In extreme instances large plantings become almost totally infected within a few weeks. By contrast, the spread of many viruses among woody plants is relatively slow, or even imperceptible. Such

[1]It is uncertain whether blackcurrant reversion and the diseases transmitted by whiteflies are caused by viruses. Otherwise the review is restricted mainly to economically important virus diseases and excludes those now associated with mycoplasmas or other organisms.

infection may have originated as a result of rare instances of spread from other hosts. Once established in commercial clones, viruses do not have to spread rapidly to survive, because they are perpetuated inadvertently by vegetative propagation (58). Cucumber mosaic virus, for example, occurs throughout certain clones of blackcurrant, despite an apparent inability to spread between bushes and despite the low rate of infection by aphids entering the crop from elsewhere (79).

Some viruses spread solely from sources outside the crop, there being no plant-to-plant spread within the crop, at least during the first year. Other viruses spread both into and within crops, and newly infected plants soon become foci for secondary spread. It is convenient to distinguish between these two main types of spread and refer to "simple-" and "compound-interest" diseases (83). These terms come from the analogy between the increase of disease with time and the increase of capital by simple or compound interest. This concept facilitates the analysis of data, although a particular virus does not always spread in the same way in all crops, at all sites, or at all stages of the growing season.

Six features are particularly important in distinguishing complex biological systems from simple mathematical models:

1. Spread does not occur at a uniform rate and depends upon such factors as seasonal or other changes in the size and mobility of vector populations.
2. There are finite limits to the amount of disease that can develop at a particular site.
3. An increasing number of plants receive more than one infective dose as spread proceeds. The extent of this "multiple infection" can be calculated, and appropriate values have been tabulated for transforming percentage of infection into infection units (39).
4. Newly infected plants do not immediately contribute to further spread, and the plants infected first become increasingly remote from the remaining healthy ones.
5. Disease is seldom randomly distributed and tends to occur in localized areas of high intensity (often referred to as foci).
6. There are seasonal changes in the size and susceptibility of plants and in the virus content of infected tissue.

Simple-Interest Diseases

Few virus diseases are of the simple-interest type that spread into crops solely from outside sources. There are few data on the progress of such diseases; this subject is discussed by Van der Plank (83).

Capital increases at a fixed rate of simple interest to give a linear increase on plotting the accumulated sum invested against time, the slope of the gradient depending on rate of interest (r). No such simple relationships have been observed between the proportion or percentage of virus-infected plants (x) and time (t). Values of x increase in curvilinear or sigmoid fashion; the influx of infection tends to be low at the outset and to increase with time as seasonal or other conditions affecting spread become more favorable. The absolute rate at which new infections appear ($x_{t_2}-x_{t_1}/t_2-t_1$) later declines as the influx decreases or conditions deteriorate. An important distorting factor is that progressively fewer healthy plants remain to

be infected and multiple infection becomes increasingly important (39). Hence, the correction factor $(1-x)$ is used when obtaining values of r by plotting $\log_e [1/(1-x)]$ against t. Van der Plank has presented the detailed mathematics and tabulated appropriate values for each unit of x (83).

Several diseases caused by tomato spotted wilt virus are of the simple-interest type. Infection is carried into tobacco, tomato, and pineapple plantings by adult thrips that acquire virus as nymphs while feeding on infected weeds (6, 51, 84).

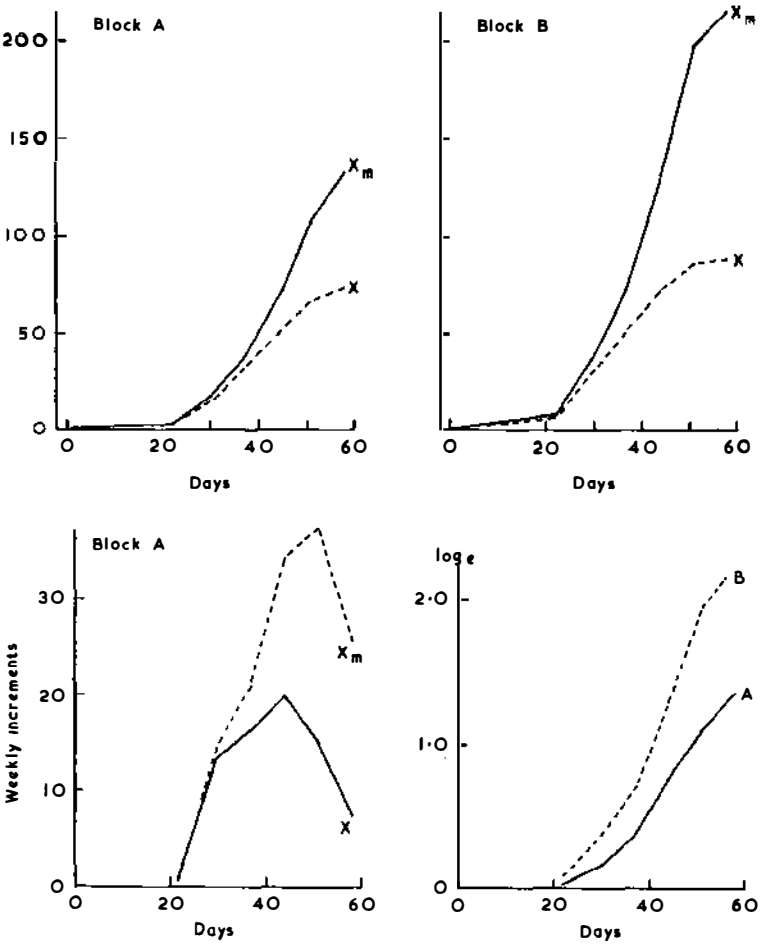

Figure 1 The spread of spotted wilt virus into two blocks (*A* and *B*) of tomato in Australia (6). (*top*) Weekly totals of infected plants in each block as a percentage (x) and after transformation (x_m) to allow for the progressively increasing amount of multiple infection (39). (*bottom*) Successive values of $\log_e [1/(1-x)]$ for each block (*right*) and weekly increments of x and x_m in block *A* (*left*).

Spread is influenced by weather conditions, principally temperature, which affects the development and influx of winged adults. Once spread begins it occurs in surges, and periods of high temperature are followed within days by the appearance of many new infections. These infections developed at random at sites in Australia, and statistical analyses revealed no evidence of the grouping that would have occurred as a result of spread between adjacent plants (6). A later method developed for the analysis of similar data from South Africa (81) involved a comparison between the observed number of pairs of infected plants and the number that would have been expected from a totally random distribution. This "doublet" test has been widely quoted and used to investigate the spread of other diseases, despite the limitations discussed by Freeman (36), who proposed an alternative method.

Apart from short-term variations in flight activity there are overall trends in thrip populations as the number of adults increases to a peak when the weed hosts mature, flower, and begin to senesce (Figure 2). Hence it is advantageous to increase the initial plant population and to delay thinning until the main influx of thrips is over (84).

Necrotic yellows is an aphid-borne virus of lettuce that is spread exclusively from sowthistle (*Sonchus oleraceus*), the primary host of virus and vector (60, 69, 70). Similarly, maize rough dwarf virus is carried into maize by planthoppers, which do not breed or acquire virus within the crop (41). Common mosaic of cotton in Brazil is an example of a virus transmitted by white flies and spread exclusively into crops from nearby malvaceous weeds (26).

In certain western states of the USA, sugar beet curly top virus is carried into bean, tomato, flax, melon, and other crops that are not breeding hosts of the leafhopper vector (*Circulifer tenellus*). The main influx is by hoppers from indigenous desert plants and introduced weeds that are the principal winter and spring hosts of virus and vector (4, 8).

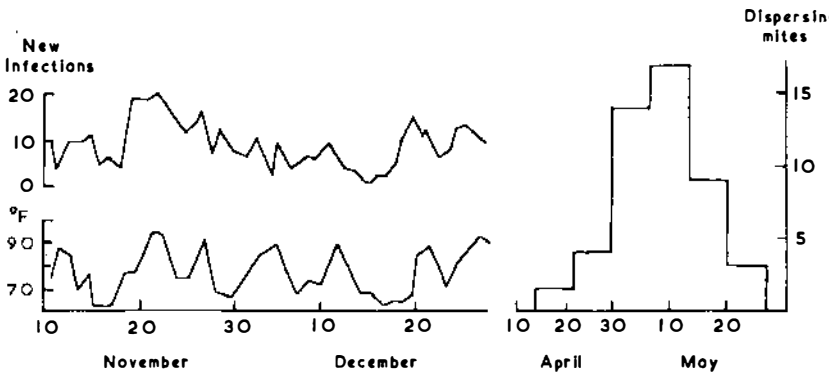

Figure 2 (*left*) Multiple peaks in the spread of spotted wilt virus in tomato (*top*) and maximum screen temperatures 12 days previously (*bottom*). (*right*) Single peak in the spread of the gall mite vector of blackcurrant reversion virus (79).

Curly top behaves like a compound-interest disease (21) when *C. tennelus* breeds and spreads virus within sugar beet crops. The spread of blackcurrant reversion virus by its mite vector is also complex and not wholly within a single category. In young plantations, spread is mainly from outside sources, and the newly infected bushes tend to be widely scattered and not obviously grouped around those present originally (72). Secondary spread occurs eventually, unless the early infections are removed before they have been invaded systematically and become particularly vulnerable to mite infestation (78).

Compound-interest diseases behave initially in the simple-interest manner when they first appear in crops and before secondary spread occurs. Thus cantaloup plantings were almost totally infected with watermelon mosaic virus 2 by an early and heavy influx of aphids from a nearby source (54). By contrast, there was little secondary spread of leaf roll of potatoes when aphid infestations occurred late in the season (18, 30).

Compound-Interest Diseases

Most viruses spread into and within crops and cause diseases of the compound-interest type. However, such diseases seldom spread for long in a manner closely analogous with the logarithmic increase of capital at compound rates of interest. With virus diseases the total amount of infection (x) usually increases in a sigmoid manner with time (t). Initially the increase of disease is limited by the few sources of infection present and/or by the lack of sufficient active vectors. There is a similarly low rate of increase when spread has continued until few uninfected plants remain. At intermediate values of x around 0.5 (50% infection) the absolute rate

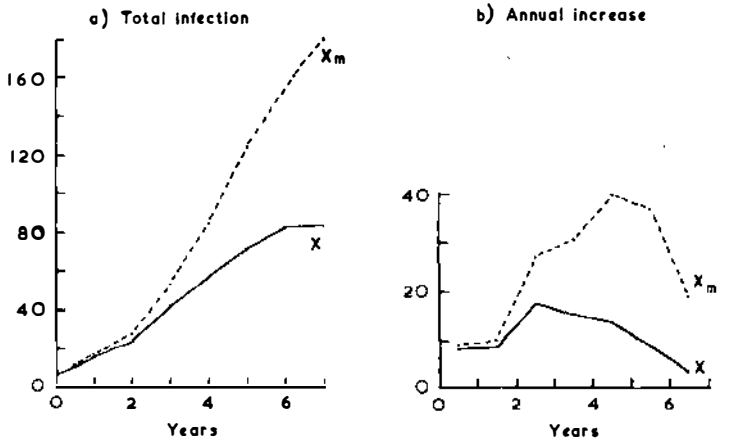

Figure 3 The spread of cacao virus in a Trinidad plantation (28). (*left*) The total number of infected trees at the end of each year as a percentage (x) of the total stand and after transformation (x_m) to allow for the progressively increasing amount of multiple infection. (*right*) The annual increments in x and x_m.

of increase is relatively fast because spread is not restricted by a lack of inoculum, vectors, or healthy plants.

The initial phase is truly logarithmic when the number of new infections that appear is directly proportional to the total infection already present. Hence, there is a linear relationship between $\log_{10} x$ and t, with a slope indicating infection rate (r). Any deviation from the line indicates that r has varied and the slope tends to flatten with time. (Such behavior distinguishes disease spread from the growth of fixed-interest investments and from autocatalytic chemical reactions proceeding at a fixed rate.) In calculating r it is again appropriate to use the correction factor $1-x$ to allow for the diminishing proportion of uninfected plants and plot $\log_{10}$ $[x/(1-x)]$ against t (Figure 4). Appropriate values have been tabulated for each unit of x (83).

Values of r are usually greatest during early spread when there is a progressively increasing number of infected plants from which further spread can occur and a corresponding decrease in the importance of outside sources. Spread is facilitated, especially in annuals, by an increase in plant size. This may be accompanied by an increase in the number and activity of the vectors. There is also decreased separation and sometimes increased contact between individual plants, together with a great increase in the amount of both infected and vulnerable tissue accessible to vectors.

As spread proceeds, the oldest infections become increasingly remote from the remaining healthy plants. Consequently there is an increase in the amount of infected tissue and in the number of vectors that do not contribute to spread. This effect is particularly pronounced for nematode and other vectors of limited mobility and for those that do not thrive on virus-infected plants. For example, cacao trees infected with virulent strains of swollen shoot virus become unfavorable hosts of the vector and eventually die (68). Spread is mainly by mealybugs that move onto the branches of adjacent trees from new infections on the periphery of outbreaks (25, 68, 77). Thus the annual spread in a plantation is directly proportional to the

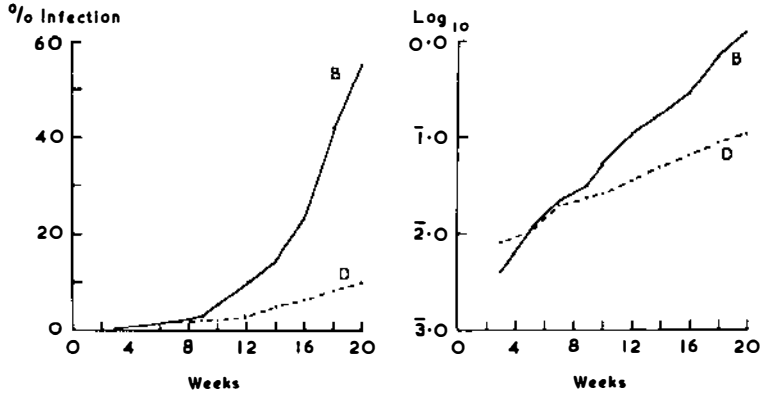

Figure 4 The spread of leaf curl virus in two varieties of cotton (*B* and *D*) in the Sudan (38). Infection plotted as percentage (x) of the total stand (*left*) and as $\log_{10}$ $[x/(1-x)]$ (*right*).

number of healthy trees in contact with infected ones at the beginning of the year and not to the total value of x (77).

With all diseases, the amount of multiple infection increases as progressively fewer healthy plants remain (Figure 3); but spread may be checked or even halted before this becomes important, especially in annual crops. These mature or otherwise become resistant to infection, as with leaf roll of potato (18, 74). There is often a tendency for the virus content of infected plants to decrease with age, and this decrease may be accompanied by a seasonal decline in vector populations or a decrease in their ability to acquire or transmit the virus (23). For example, the spread of maize rough dwarf is checked by high summer temperatures that decrease the virus content of the planthopper vector (41). Such factors contribute to the generally similar sigmoid shape of disease-progress curves, whether or not much spread occurs (Figure 5).

Vector populations may decline because of chemical control measures or seasonal trends. In some areas aphid numbers are decreased by high summer temperatures and, with irrigation, hot arid areas can be used to grow virus-free seed crops or planting material (71). Elsewhere the main check on the population comes with the onset of cool autumn conditions or as parasites and predators become numerous (10). Occasionally spread is halted by sudden storms (63) or by prolonged periods of unfavorable weather (33, 91).

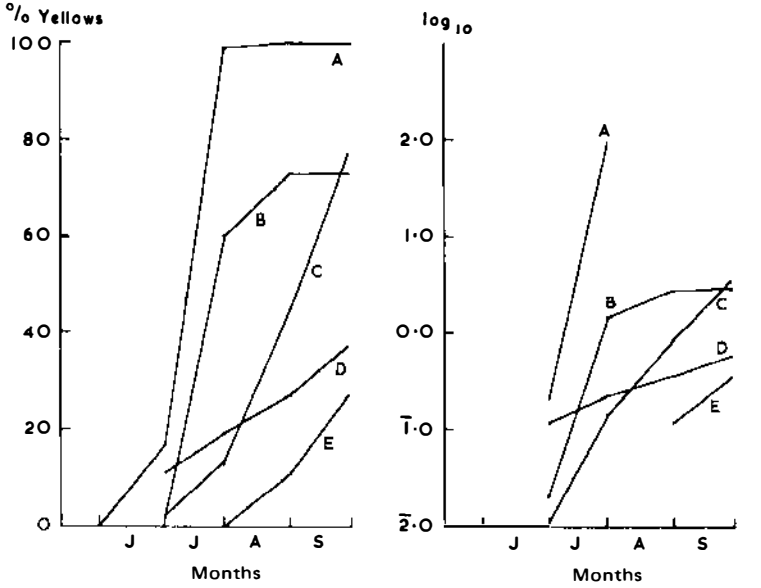

Figure 5 The spread of sugar beet yellows at five sites in England (45). Percentage (x) of plants infected at monthly intervals (*left*) and successive values of $\log_{10}[x/(1-x)]$ (*right*). Infection appeared early and spread rapidly at *A*, whereas spread was slow early or late in the season at *E* and *B* and throughout the season at *D*.

The ultimate proportion of infected plants and the rate at which new infections appear vary widely among different viruses and for different crops. There are also major differences between sites and seasons for particular diseases such as sugar beet yellows (31, 45, 65, 89) and plum pox (49).

Viruses that infect annual crops spread much more rapidly than those of trees and shrubs (82). This explains why eradication measures that control the spread of virus diseases of some tree crops are largely ineffective against those of herbaceous annuals. In a typical orchard in California, tristeza virus spread to an average of two citrus trees a year for each infected one already present (29). By contrast cauliflower mosaic virus spread from a single infected plant to as many as 131 others in one season (48). Often there is almost total infection of tobacco crops with mosaic (93) and of cotton with leaf curl virus transmitted by white flies (38). Similarly with the aphid-borne viruses of cucurbits (Figure 6), lettuce (15), groundnut (1, 10), pepper (50, 66), carrot (91), brassicas (13, 48), and other vegetables and cereals (12, 57, 67). The ability to spread rapidly and become established in new areas has obvious survival value for viruses and vectors that depend on exploiting ephemeral hosts and habitats. This is particularly important for viruses that are not seed-borne and for vectors that have no special drought-resistant or overwintering form.

There are relatively few papers on the spread of virus diseases among woody perennials. Compared with herbaceous plants they provide a stable long-lived substrate for virus and vector. Invariably such viruses as citrus tristeza (9), cacao swollen shoot (77), plum pox (49), peach mosaic (47), and blackcurrant reversion (3) take several years to spread throughout plantations (Figure 7). Nevertheless, they cause serious losses because individual trees are far larger and take longer to produce a crop and consequently are much more valuable than herbaceous plants. They are also slow growing and sometimes difficult or impossible to replace.

The actual rate of spread between trees depends on such factors as the number and proximity of the main sources of infection and the mode of spread (49). Tree size and age are also important. Prunus necrotic ringspot virus, which is pollen-borne, cannot spread until flowering commences (64). Similarly, young cacao trees support few mealybugs, and there is little spread of virus until the branches form a continuous interlocking canopy (25, 68).

Woody perennials tend to be difficult to infect, and the generally wide spacing between them hampers the movement of vectors and impedes virus spread. Moreover, virus is slow to become systemic and there is generally a long interval that may extend for many months before the virus becomes available to vectors. This delays spread compared with that in herbaceous hosts in which viruses may become systemic in a few days.

The date infection first appears within a crop is of crucial significance in epidemiology. The existence of viruses within crops from the outset poses a particular problem. These viruses may be due to the presence of weed hosts (32), to regeneration from the residues of previous crops, or to the use of infected stocks of seed (64) or other planting material. As a basic control measure such foci of infection should be eliminated; this is a major objective of the various seed and stock certification schemes being developed in many countries (44). These aim to defer the onset of

disease and delay or decrease the ultimate amount of infection, as with the use of mosaic-free lettuce seed (94). Such measures decrease crop losses, especially because the plants that are infected longest are usually the worst affected.

Soil-Borne Viruses

Diseases caused by viruses with nematode or fungal vectors behave unlike typical simple- or compound-interest diseases. Usually the roots of susceptible plants soon become infected when grown in soil containing infective populations of the vector, but there is some delay before virus reaches the aerial parts and causes symptoms (17, 75). The delay may extend to several months or more with fanleaf of grapevine or nettlehead disease of hop (80). These and other soil-borne diseases occur suddenly in patches that coincide with the distribution of infective vectors. The patches may extend over whole fields in the case of the wheat mosaic transmitted by the fungus *Polymyxa graminis* (Plasmodiophorales).

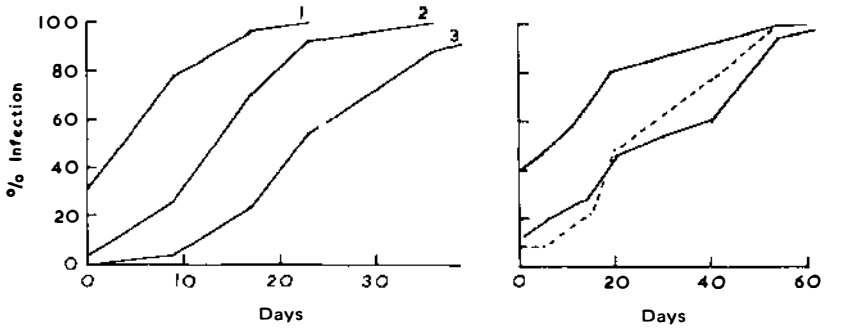

Figure 6 The spread of curly top virus into three sugar beet plantings in Idaho (*right*) and of watermelon mosaic virus 2 into a cantaloup field in Arizona (*left*). Sites 1–3 were at increasing distances from the original source of infection (54).

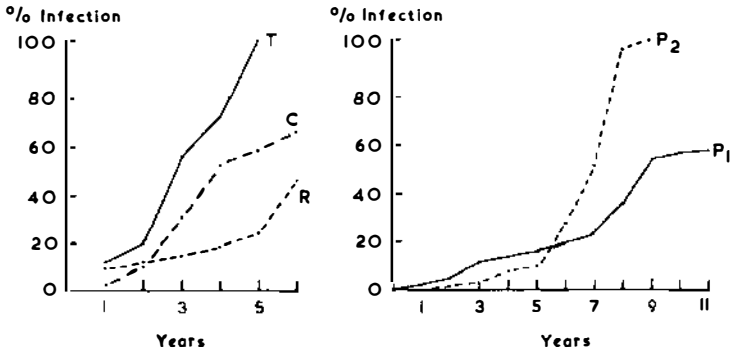

Figure 7 The spread of viruses of perennial crops. (*left*) C = cacao swollen shoot (77), R = blackcurrant reversion (3), T = citrus tristeza (9). (*right*) P_1 and P_2 = plum pox at different sites (49).

Soil-borne virus diseases tend to spread slowly after their initial appearance, due to the limited mobility of their vectors. There is no detailed information on fungal vectors, but it has been estimated that the nematode vector of arabis mosaic and strawberry latent ringspot viruses *(Xiphinema diversicaudatum)* moves through the soil only 30 cm per year (40). Viruses move more rapidly than this through the root systems of such perennials as grapevine, hop, and cherry. This explains the unexpectedly rapid spread that occurs when growers unintentionally plant infected stocks in soils containing noninfective nematode vectors of the virus. Increasing numbers of vectors have access to infected roots, from which they spread the virus to nearby healthy plants.

SEASON OF SPREAD

There is great diversity in the periodicity of virus spread, as may be expected from the different types of vector involved and the wide range of conditions under which crops are grown.

Weather conditions are most uniform in the humid tropics. Perennial crops grow almost continuously throughout the year in the forest areas of West Africa except during the short dry season and during the relatively cool conditions at the height of the rains. Hence there is at least some spread of viruses of sugar cane, cassava, pineapple, banana, abaca, cacao, and other crops at all times of year. Seasonal trends in the rate and pattern of spread of cacao swollen shoot virus are due to differences in the size and activity of populations of the mealybug vectors (25, 68, 77). These trends are not readily apparent because most new infections show during peak flushes of new growth after a long and variable latent period.

Elsewhere in the tropics and subtropics there are clearly defined seasons of growth separated by dry periods that may be intense and in some areas prolonged. The main rain-fed crops are annuals including many cereals and legumes. Many of these are of short duration, and yet they may become severely affected by virus diseases. Groundnut rosette virus has received particular attention in many African countries, because it is soon carried into and spread rapidly within plantings by aphids that originate from distant crops or from nearby perennials that survive the dry season (2). At Nigerian sites, the incidence of rosette was closely correlated with the proportion of plants that become infested with *Aphis craccivora.* Predators were numerous, and aphid populations fluctuated rapidly, with few colonies persisting more than a week (10).

There is a similar pattern of invasion of tobacco, cotton, pepper, cucurbit, maize, vegetable, and other crops with viruses transmitted by aphids, whiteflies, leafhoppers, or beetles.

Spread between crops is facilitated in regions with a bimodal distribution of rain and two overlapping growing seasons per year. This contributes to the prevalence of sugar cane mosaic and other viruses of maize in parts of Kenya. Similarly, the increasing use of irrigation to obtain crops during the dry season is likely to increase the overall incidence of disease, as noted already in rice and maize (5, 61, 85).

Drought provides the main check on growth in the tropics and subtropics, whereas cropping patterns in the temperate regions are mainly determined by winter temperatures. The principal crops are deciduous perennials or annuals with a restricted season of growth. The activity of virus vectors is similarly restricted, especially in areas with severe winters where the topsoil is frozen for long periods and where insects survive only as eggs or in diapause. In less extreme conditions there is spread of some viruses during the winter months, although the movement of vectors between plants is less than at higher temperatures and many crops are not planted until the spring or early summer. The increased survival of crop and weed hosts and of vectors in mild winters and at protected sites is important in the epidemiology of many viruses and leads to much spread early in the main growing season (13, 15, 46, 88, 91).

The spread of blackcurrant reversion virus is unusual in that it is restricted to a few weeks in late spring (Figure 2). At this time, the gall mite vector emerges from overwintered galls and disperses to young buds of the new season's growth (78, 79). Other mite vectors are less confined to buds, some being free living with a prolonged dispersal period (55).

Many insect vectors and especially aphids (73) have a complex life cycle that determines the main period of virus spread. This often coincides with the appearance of particularly active and in some instances specialized forms of the vector that are adapted for dispersing to new habitats. For example, the peak periods of spread of aphid-borne viruses of strawberry in southern England coincide with the appearance of alate forms, and apterae predominate at other times (59). Two main periods of spread were detected by exposing successive batches of potted plants at intervals throughout the growing season (Figure 8). Some plants were protected by sticky

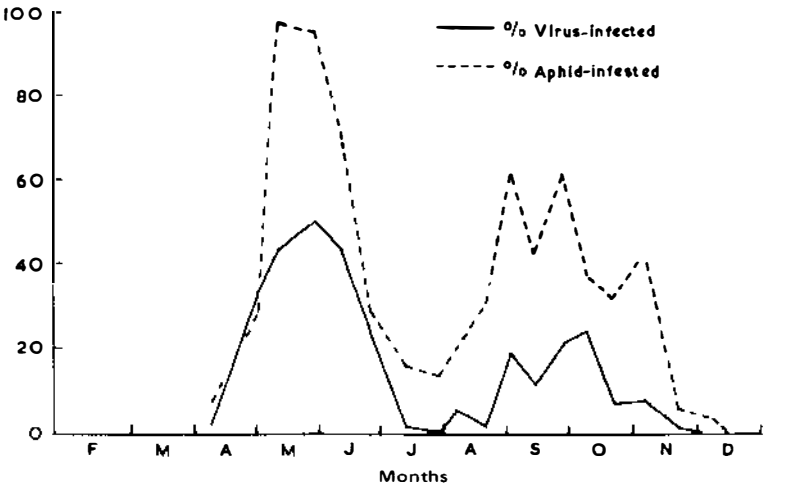

Figure 8 The spread of viruses and their aphid vector to successive batches of strawberry plants exposed at fortnightly intervals throughout the growing season in southern England (59).

surfaces, as in previous work with potato (14), in an attempt to distinguish spread due to alates and apterae from that due to alates alone.

A limitation of the exposure technique is that it is not always possible to ensure that the experimental plants are exactly comparable in size and susceptibility to those of the crop in which they are placed. Nevertheless, the method is more generally applicable than others used to investigate the spread of potato viruses. These involve either the sequential sampling of tubers (30), or the use of insecticides in attempts to halt spread at different stages of the season (16). Knowledge of the main period of spread is important in developing control measures and methods of forecasting the progress and prevalence of a disease.

FORECASTING

Many environmental and other factors influence the complex interactions between virus, host, and vector; hence forecasting the appearance and development of a disease is difficult. Attempts at forecasting are justified because success indicates an understanding of the main factors influencing epidemiology. There are also great potential benefits to growers, processors, and advisory officers who can anticipate losses and apply appropriate chemical or other control measures. Moreover it may be possible to operate an early warning system or to select growing seasons or areas for special crops where infection is unlikely.

There has been little progress in forecasting virus disease with many crops. A major problem has been the difficulty in getting the necessary sequence of data on disease incidence from representative sites over a sufficient number of years. Standard methods are required for assessing virus infection and vector abundance throughout the season, and it is desirable to have continuity of suitably trained personnel.

Few crops have received such detailed attention as sugar beet. In Idaho a correlation was soon established between severe winters and relative freedom from damaging outbreaks of the leafhopper vector of beet curly top (19). Winter and early spring temperatures affect the rate at which the vectors mature on their winter hosts and the main dispersal period was predicted from data on accumulated degree-days above 45°F (24).

Elsewhere, trap catches of the leafhopper vectors of maize streak virus in Rhodesia were correlated with the amount of rain at the end of the preceding wet summer season (61). More complex relationships have been used to predict the incidence of rice dwarf virus and its leafhopper vector in Japan. In this case, the amount of infection the previous season and counts of overwintering individuals, as amended by later population trends (42, 53), were used as the basis of prediction.

In Britain, aphids carry yellows viruses into the sugar-beet crop from outside sources. Further spread is mainly within or between crops until there is almost total infection or until growing conditions become unfavorable. Early infection increases in the compound-interest manner, and it is possible to predict the ultimate amount of infection from earlier counts of yellows and sticky trap catches of winged *Myzus persicae* (89) (Figure 9).

Aphid counts were weighted according to collection date, because infection and loss of crop are greatest when virus appears early. This occurs after mild winters, when many aphid colonies survive on herbaceous plants including some that are hosts of virus. Alates develop later and are least numerous after cold winters, when survival is mainly as eggs on primary woody hosts immune to virus. For this reason there is a good correlation between winter temperatures (46) or number of freezing days (88) and the later incidence of virus (Figure 10). Weather conditions are partially related to cycles of sunspot activity, which have also been associated with the incidence of yellows (37).

In concurrent work, yields of carrots were correlated with sticky-trap catches of the aphid vector of carrot motley dwarf virus (91). Similar factors affecting the overwintering of this aphid and *Myzus persicae* explain the association between the prevalence of motley dwarf and beet yellowing viruses (91).

Sticky-trap catches have also been used to predict the incidence of barley yellow dwarf virus in winter wheat in New Zealand. Growers are advised to spray when many cereal aphids are caught in the autumn. Insecticides decrease overwintering populations and check the secondary spread that otherwise leads to heavy crop losses (22).

Quite different conditions affect the appearance of barley yellow dwarf virus in Minnesota and adjacent states where the main vectors do not overwinter. Severe losses occur only in seasons permitting rapid increase and spread following an early and heavy influx of aphids from southern states (12). The particular conditions favoring such long distance dispersal have been established, and heavy infestations can be anticipated if suitable weather occurs in the critical April–May period (34, 43, 86).

Wheat streak mosaic virus has also received much attention in North America. The eventual prevalence of infection and loss of yield in autumn-sown crops was predicted with fair accuracy by assessing early infection. This assessment was done in winter when cold weather had virtually halted spread by the mite vector until

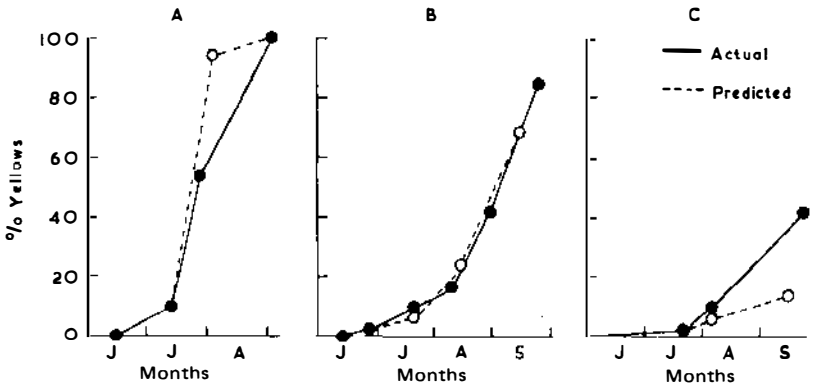

Figure 9 The spread of sugar beet yellows virus at three sites (*A*, *B*, and *C*) in England as observed and as predicted from previous observations and sticky trap catches of winged *Myzus persicae* (89).

the following spring. Few, small samples were adequate because the disease only became serious when there was much early infection (35).

The incidence of cotton leaf curl in the Sudan has been associated with the severity of the preceding dry season. Severity determines the amount of regeneration from the infected stumps of previous crops that act as primary foci. Regeneration was greater after additional dry-season irrigations than when water was withheld prematurely (11). Similarly, rainfall influences the survival of overwintering hosts of curly top virus in Washington state. This explains the correlation between winter rain and the later incidence of infection in sugar beet. Infection is also related to the number of summer days with above average temperatures that increase the activity of vectors (21).

These examples show that specific features early in the season may be of crucial importance in the epidiomology of a disease, despite the apparent complexity of the overall situation. Ultimately it may be possible to use remote scanning techniques to provide data on a comprehensive or even global scale for detailed computer assessment, analysis, and prediction of disease incidence. There is much interest in the possibilities of such methods, and high-flying aircraft and satellites are being used with sophisticated photographic and electronic equipment. These developments have yet to make a major impact in virus epidemiology, and much remains to be done in reconciling aerial photographs with ground observations.

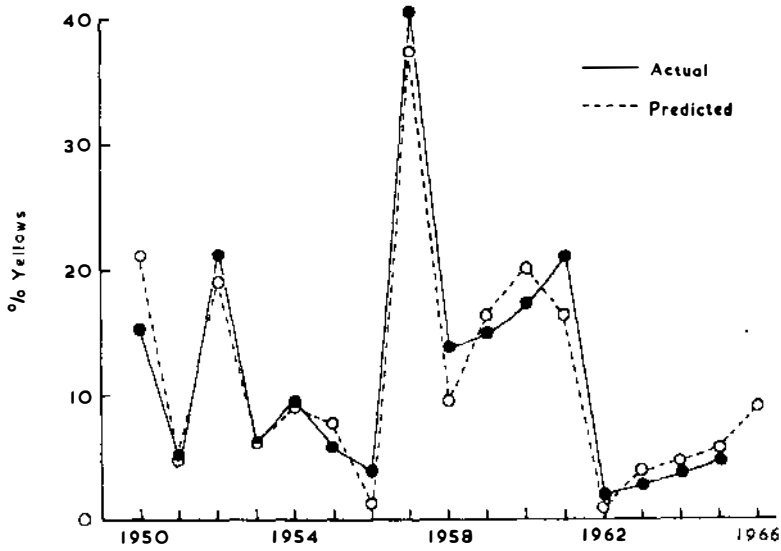

Figure 10 Overall incidence of yellowing viruses in English sugar beet crops for the years 1950–1966. Observed levels at the end of August compared with those calculated from the multiple regression involving numbers of freezing days between January and March and deviations from average April temperatures (88).

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