The Origins and Epidemiology of Some Important Plant Virus Diseases

J. M. THRESH

East Malling Research Station, Maidstone, Kent

I. Introduction*

In the extensive literature on the epidemiology of plant virus diseases many reviews have dealt with general aspects of the topic or with the role of particular groups of animal or fungal vectors. This paper differs in that it seeks to explain why certain particularly important virus diseases have become prevalent within crops. There has been no previous approach along these lines, although several virus diseases were discussed in assessing diverse diseases categorized as "catastrophic" (Klinkowski, 1970) or "threatening" (Thurston, 1973). Plant virus diseases were not considered in an otherwise comprehensive discussion of the origins of many other pest, parasite, disease and weed problems (Cherrett and Sagar, 1977).

II. Some Limitations of the Available Information

In considering the literature on the epidemiology of plant virus diseases it is important to appreciate the serious limitations of the available information. These are mainly due to the recent origins of virology and to the difficulties encountered in detecting and identifying viruses and in assessing their distribution in crops, weeds and natural vegetation.

Viruses were not recognized as a separate group of plant pathogens until the work of Beijerinck (1898) and the main developments in virology have been very much more recent. The precise aetiology of the diseases discussed in many of the early papers is uncertain and there is a dearth of evidence on the history of almost all the diseases now known to be due to viruses. No relevant information has come from studying fossils or peat deposits and there has been little from old manuscripts or from the 18th and 19th century naturalists who contributed so much to ornithology, entomology, botany and some aspects of mycology. Nevertheless, there is little doubt that viruses have long been present within crops. Hop nettlehead disease was first described in England in 1574 and there are well-known 17th century paintings from the Netherlands of tulip flowers with symptoms now known to be due to an aphid-borne virus (Bos, 1978).

A recurring difficulty in epidemiology is that few viruses consistently cause obvious diagnostic symptoms that can be recognized without difficulty and recorded in routine surveys. In at least some

* No attempt has been made to explain the ultimate origins of viruses as this is beyond the scope of the present paper and there is little to add to previous discussions (Matthews, 1970; Gibbs and Harrison, 1976; Nahmias and Reanney, 1977).

hosts the symptoms are usually indistinct and may be very slight or even absent for all or part of the growing season. Symptoms may also be difficult to distinguish from those due to other viruses or to such pathogens as fungi, bacteria, protozoa, mycoplasmas, spiroplasmas or rickettsiae. Moreover, the effects of insects, mites, mineral deficiencies, water-logging, pesticides, herbicides and many other factors can closely resemble those due to viruses. At least some confirmatory tests are required for accurate diagnosis and various techniques are used including inoculations to sensitive indicator plants, serology or electron microscopy. Many of the available techniques are inadequate or unsuited for large scale surveys or they have only recently been developed. All require considerable expertise and the necessary facilities and suitably trained personnel are not yet available or have only just been provided in many important agricultural regions of the world.

The past emphasis has been on potato, sugar beet, brassicas, fruit and a few other particularly important crops in countries of the temperate regions with well-developed agricultural systems. Cacao, cotton, groundnut, citrus and sugar cane are some of the few tropical crops to have been studied in any detail, although increased attention is now being given to grain legumes, rice, maize and other food crops. There is only limited quantitative information in all regions on the prevalence of viruses within crops, and even less on their incidence in weeds and natural vegetation. Cropping practices must have had a major impact on the overall virus situation from the earliest phases of agricultural development. However, the magnitude of these effects can seldom be established because of the almost total lack of information on long-term trends in virus incidence.

Despite these limitations, there is sufficient information on some particularly well-known viruses to be able to describe their most recent history and to account for their present importance. It is clear that viruses resemble other pathogens in becoming prevalent for diverse reasons. Several different categories can be distinguished and these are considered in the following sections.

III. Viruses Becoming Prevalent in Periods of Unusually Favourable Weather

The influence of weather on the incidence of diseases has long been apparent to growers and detailed scientific studies of these effects have been in progress since the devastating epidemics caused by the potato late blight fungus (*Phytophthora infestans*) throughout northern Europe

TABLE I The principal viruses considered in this paper and their natural means of spread

CMI/AAB[1]	Virus	Group	Vector	Order
16	Arabis mosaic	Nepovirus	Nematode	Nematoda
32	Barley yellow dwarf	Luteovirus	Aphids	Hemiptera
	Beet curly top	Geminivirus?	Leafhopper	Hemiptera
	Beet mild yellowing	Luteovirus?	Aphids	Hemiptera
13	Beet yellows	Closterovirus	Aphids	Hemiptera
10	Cacao swollen shoot	—	Mealybugs	Hemiptera
33	Citrus tristeza	Closterovirus	Aphids	Hemiptera
	Cotton leaf curl	—	Whitefly	Hemiptera
28	Grapevine fanleaf	Nepovirus	Nematode	Nematoda
	Hop mosaic	Carlavirus	Aphids	Hemiptera
	Lettuce big-vein	—	Fungus	Chytridiales
	Maize dwarf mosaic	Potyvirus	Aphids	Hemiptera
72	Maize rough dwarf	Reovirus	Planthopper	Hemiptera
133	Maize streak	Geminivirus	Leafhoppers	Hemiptera
70	Plum pox	Potyvirus	Aphids	Hemiptera
36	Potato leafroll	Luteovirus	Aphids	Hemiptera
37	Potato Y	Potyvirus	Aphids	Hemiptera
67	Rice tungro	—	Leafhoppers	Hemiptera
149	Rice yellow mottle	—	Beetles	Coleoptera
	Subterranean clover stunt	—	Aphids	Hemiptera
119	Sugar cane Fiji	Reovirus	Leafhoppers	Hemiptera
17	Tobacco ringspot	Nepovirus	Nematode	Nematoda
156	Tomato mosaic	Tobamovirus	—	—
18	Tomato ringspot	Nepovirus	Nematode	Nematoda
8	Turnip mosaic	Potyvirus	Aphids	Hemiptera
77	Wheat soil-borne mosaic	Tobamovirus?	Fungus	Plasmodiophorales
167	Wheat spindle streak	—	Fungus	Plasmodiophorales
48	Wheat streak mosaic	—	Mite	Acarina

[1] The numbers used here and throughout this paper refer to the CMI/AAB Descriptions of Plant Viruses which provide further information on the properties of the viruses considered.

in the cool wet summer of 1845 (Bourke, 1964). Work on virus diseases has revealed equally striking instances of the impact of weather conditions. Indeed, several important viruses were entirely overlooked until they caused serious losses and attracted attention in seasons when conditions were particularly favourable for spread to occur or for symptoms to develop.

A. BARLEY YELLOW DWARF VIRUS

Barley yellow dwarf virus infects many annual and perennial grasses and various cereals including wheat, barley, oats, rye and maize. It is

now considered to be one of the most important and prevalent of all viruses and yet it was entirely unrecognized until 1951. In mid-April of that year barley crops near Davis, California, were affected by a disease causing conspicuous yellowing and stunting. Symptoms developed suddenly over a large area and at diverse sites where normal plants were found alongside or intermingled with affected ones. This distribution was unlikely to have been due to a soil-borne disease or to soil or other environmental conditions, and insect transfer experiments soon established the cause as a persistently-transmitted aphid-borne virus (Oswald and Houston, 1951).

Once the characteristic effects of barley yellow dwarf virus had been recognized and described in oats, barley and wheat it became apparent that infection had been present in the region since at least 1947 and could have been responsible for periodic outbreaks of unidentified disease recorded in cereals over the previous 60 years (Bruehl, 1961; Duffus, 1977). The major epidemic of 1951 was associated with exceptional weather conditions in the main cereal-growing areas of California (Oswald and Houston, 1953). The usual sequence of cultural operations was delayed by a prolonged period of almost continuous rain, which ended on 7 March 1951, and was followed by a 7-week drought. The main sowings were not completed until 20 March and the seedlings were still at an extremely vulnerable stage of development when there was a heavy influx of winged aphids. These moved into plantings in large numbers from annual grasses, which were wilting rapidly after making prolific growth and becoming heavily infested during the winter months. Many of the dispersing aphids must have been viruliferous because infected plants were first seen on 11 April and within two weeks were widespread in cereal crops throughout the Sacramento and San Joaquin Valleys. Growth and yields were very severely affected due to such extensive infection occurring at an early stage of development. Losses were much less in 1952 as much of the crop had been planted by 1 January and infection did not become prevalent until May, when many plants were at the late tillering stage.

Subsequent observations in other parts of North America and elsewhere have confirmed the importance of weather conditions influencing the incidence, spread and effects of barley yellow dwarf virus and its aphid vectors. For example, the virus was considered to be unimportant in England until 1957, when cereal aphids migrated early and in exceptionally large numbers after an unusually mild winter. Yellow dwarf was reported from spring-sown crops in various regions and became prevalent in autumn sowings, especially in the southern counties (Watson, 1959).

There is a particularly interesting situation in Wisconsin and neighbouring parts of the USA and Canada where there are great differences between seasons in the severity of the damage caused by cereal aphids and yellow dwarf virus. Losses are greatest in years when aphids appear early and build up rapidly following an influx of long-distance migrants from cereal crops maturing far to the south in Oklahoma and neighbouring states (Fig. 1). Such infestations can develop suddenly and over extensive areas of the north before there has been time for populations to have developed locally from eggs over-wintering on primary tree hosts.

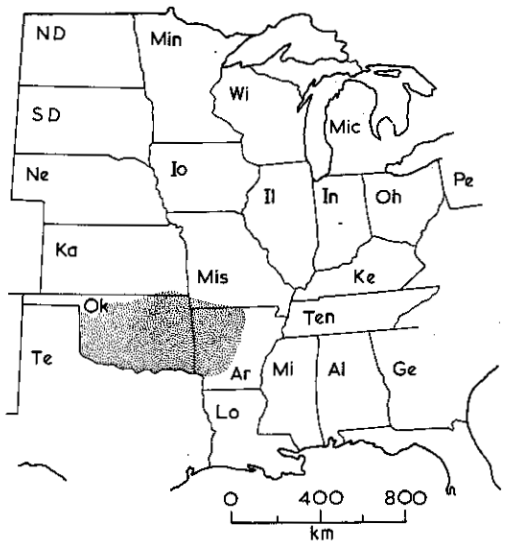

FIG. 1. Sketch map of the central areas of the United States. The stippling indicates parts of Oklahoma and neighbouring states that are south of the 2°C January isotherm and considered to be the major source of overwintering cereal aphids that migrate northwards each spring (Medler, 1960).

By detailed observations on cereal crops and an examination of meteorological data the influx of migrants has been associated with favourable winds from the south (Hodson and Cook, 1960; Medler and Smith, 1960; Orlob and Medler, 1961; Wadley, 1931) and with the subsequent appearance of yellow dwarf symptoms (Wallin *et al.*, 1967). In a 5-year study (1965–1969) suitable low-level jet winds from Oklahoma, Texas or Kansas to Iowa and more northerly states occurred annually on 9–11 days during the crucial March–April–May

period. Aphids appeared each year, but there were differences in the incidences of yellow dwarf and these were related to the age of the plants when they became infested and to weather conditions immediately after aphid fall-out occurred. Yellow dwarf was least in early sowings and in years with heavy rain and low temperatures (Wallin and Loonan, 1971). Temperature and rainfall in the south, earlier in the year, were also important as their influence on populations that develop during the winter months determines the magnitude and timing of the spring migration (Wallin, 1973).

These observations indicate the complex weather factors involved in the spread of yellow dwarf virus, that can be carried far by active vectors able to reach and exploit a sequence of short-lived plantings grown over very wide areas. For major epidemics to occur conditions influencing virus, host and vector must be favourable at successive stages of the season and in different localities. This necessitates a regional approach to epidemiology and control which has also become apparent from studies on other viruses transmitted persistently by aphids or leafhoppers. These include sugar beet curly top (Bennett, 1971), subterranean clover stunt (p. 7) and sugar beet yellowing viruses (p. 9). Further work is likely to establish that additional viruses including groundnut rosette (Adams, 1967), maize streak (Rose, 1978), rice hoja blanca (Everett and Lamey, 1969) and some of the other hopper-borne viruses of cereals need to be treated similarly for a full understanding of their epidemiology.

B. SUBTERRANEAN CLOVER STUNT VIRUS

The subterranean clover (*Trifolium subterraneum*) is a dominant or important component of the vegetation of over 20 million acres of southern Australia. The species is not native and was introduced accidentally on several occasions in the 19th century, probably with fodder for live-stock. It now thrives as an annual in natural or planted stands, germinating with the autumn rains, flowering the following spring and then dying-out during the summer drought (Morley, 1961).

Until May 1955, subterranean clover appeared to be little affected by virus or other diseases. Reports were then received of a stunted chlorotic condition affecting hundreds of acres of pasture land in New South Wales. A persistently-transmitted, aphid-borne virus was shown to be responsible and was designated subterranean clover stunt (Grylls and Butler, 1959). Subsequent experience suggests that stunt was first recognized in 1955 because the symptoms were particularly

conspicuous in that year after a period of unusually cool weather had delayed growth. In normal years slightly affected plants show partial recovery and severely affected ones are obscured or crowded-out by their healthy neighbours.

The main vector of clover stunt is the cowpea aphid (*Aphis craccivora*), which is an introduced species. It does not produce eggs under Australian conditions and survives as an anholocyclic form reproducing parthenogenetically. This means that each year the species is virtually eliminated from vast areas of pastureland along the Great Dividing Range as the host plants mature and die-out in the intense summer drought. Subsequent plantings in the autumn rains are infested by an influx of wind-borne migrants from the eastern coastal areas of New South Wales and Queensland, where moisture conditions remain favourable for plant growth and aphid reproduction throughout the summer (Fig. 2a). The virus is introduced to autumn plantings by incoming migrants that tend to alight and cause most infection around the margins of fields and alongside roadways or drainage channels. Secondary spread within crops is by the local movement of migrants and their progeny and continues until the onset of winter frosts. These eliminate populations over large areas until plantings are recolonized in spring by a further influx of migrants from the frost-free coastal districts (Fig. 2b). At least some of the virus-infected plants survive the winter, but it is not clear how

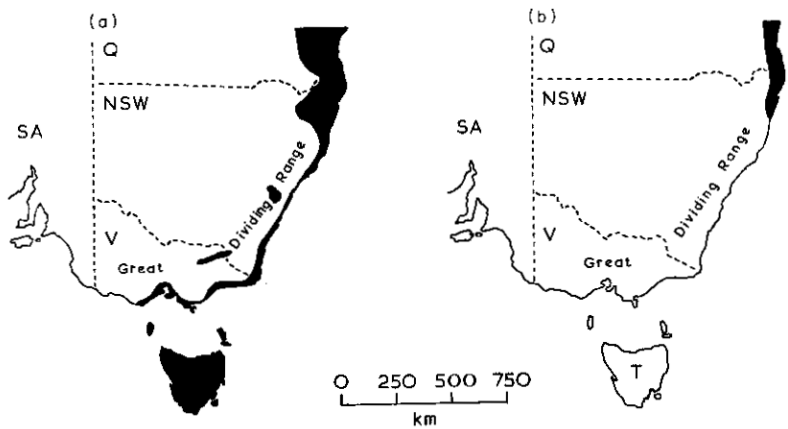

FIG. 2. Sketch map of south-east Australia showing in black the areas favourable for the development of the cowpea aphid during (a) the summer and (b) the winter. The limits of climatic favourability are based on 30-year mean moisture and temperature indices (Gutierrez *et al.*, 1974b).

much additional spread occurs in late spring and early summer, before the host plants cease to grow and die (Gutierrez *et al.*, 1971).

Some aspects of the complex epidemiology of clover stunt are incompletely described. Nevertheless, *A. craccivora* is clearly an extremely efficient vector which has been referred to as a "super migrant" because it is so well-adapted for reaching and exploiting the transient habitats provided by subterranean clover in southern Australia (Gutierrez *et al.*, 1971). Winged migrant forms are produced unusually early in population growth, before there is much crowding (Johnson, 1965). Moreover, the rate of development and reproductive potential are high by comparison with other species (Gutierrez *et al.*, 1974a). These features facilitate the production of enormous numbers of migrants that can be blown far to carry the virus from the localized areas with mild winters and/or summer rain (Johnson, 1957; Gutierrez *et al.*, 1974b). This strategy of dispersal is highly effective and distant crops are regularly colonized in the autumn and recolonized in the spring, despite the vast mortality occurring when aphids are swept out to sea or to inland areas far beyond the range of susceptible hosts.

C. SUGAR BEET YELLOWING VIRUSES

Sugar beet is widely grown in eastern England and parts of the west Midlands and north. The entire crop is processed by the British Sugar Corporation and field officers visit plantings regularly throughout the season to assess growth and record the prevalence of major pests and diseases (Hull, 1953). The published summaries of their observations are unique in the detailed data they provide on long-term trends and on regional and seasonal differences in the incidence of virus yellows.

This disease was first identified in England in 1938 (Watson, 1959), although symptoms had been described previously in England and mainland Europe (Petherbridge and Stirrup, 1935; Quanjer, 1934), where they were attributed to an aphid-borne virus originating from the wild *Beta maritima* (Roland, 1936). It was later established that yellows is caused by two distinct viruses, occurring singly or together (Russell, 1958). Sugar beet mild yellowing virus has a wide host range and is transmitted persistently by *Myzus persicae*, but not by *Aphis fabae*. By contrast, sugar beet yellows virus has a relatively restricted host range and is transmitted semi-persistently by both aphid species. The two viruses cause such similar symptoms in mature beet that they cannot be distinguished in routine visual surveys. However, transmission experiments have established that their relative importance differs between sites and seasons (Russell, 1958, 1963).

Yellows was initially of little importance in England because of its restricted distribution and avirulent effects. More damaging strains then appeared and the first serious outbreak occurred in 1943. This was in parts of Lincolnshire where plantings had been retained through the previous winter to produce seed. They acted as initial foci from which aphids spread virus rapidly to spring-sown crops grown in the area for processing. Outbreaks of increased size and severity were reported in 1944 and 1945, when only virulent strains of virus were detected (Watson, 1959).

The comprehensive surveys introduced in 1946 revealed a gradual build-up of yellows from very low levels to the unprecedented epidemics of 1949, when there was an overall incidence of 60% by the end of the growing season. Losses were so great that steps were taken to improve the health of seed crops by means of isolation, systemic insecticides, barrier crops and an official certification scheme (Hull, 1952). There was also increased use of insecticides on crops for processing and growers were alerted to the danger of aphids overwintering and spreading virus from harvested crops stored in clamps for use as animal fodder (Broadbent *et al.*, 1949). Despite these measures the incidence of yellows was high in 1957 and again in 1974, when losses were the worst ever recorded (Fig. 3).

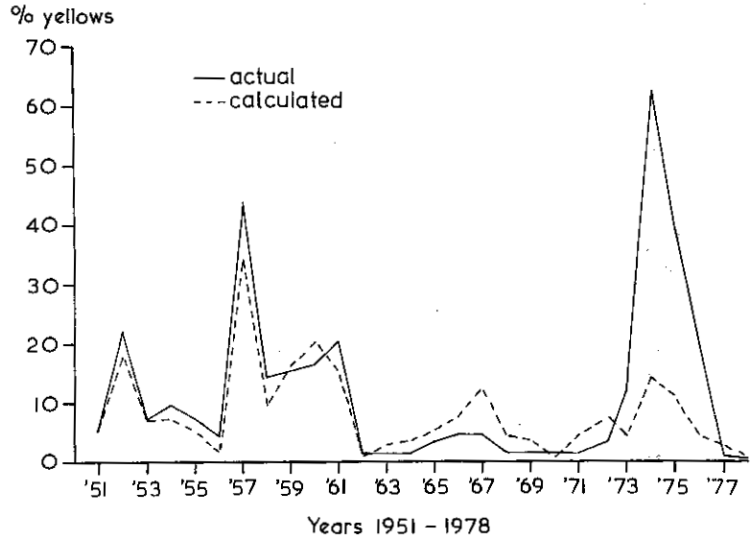

FIG. 3. The overall incidence of beet yellowing viruses in Britain, 1951–1978. Percentages observed (continuous line) and calculated (broken line) from January–April weather records (Watson *et al.*, 1975; G. D. Heathcote, unpublished data).

The great differences between seasons in the incidence of yellows present challenging problems of interpretation in relation to weather. A particularly rewarding approach has been to consider winter conditions influencing the number and infectivity of the aphid vectors migrating to beet and responsible for the initial foci of infection. The migration tends to be restricted and late after very severe winters, when many of the migrants originate from colonies that overwinter as eggs on trees or shrubs that are not hosts of beet viruses. In mild winters there is much greater survival of herbaceous hosts and the aphids they support. Aphid colonies continue to reproduce parthenogenetically throughout the winter and make an important early contribution of infective alates to the spring migration.

These features explain why surprisingly accurate predictions of the incidence of yellows can be made from weather records available at or soon after planting. Hurst (1965) first emphasized the association of severe outbreaks of yellows with above-average screen temperatures in the main growing areas of eastern England during February, March and April, considering these months either separately or together. In additional analyses there was a negative correlation between the incidence of yellows at the end of August and the total number of freezing days in January, February and March (Watson, 1966). The regression accounted for 60% of the total variance and for 89% when adjusted to take account of the positive correlation between yellows and April temperatures.

The weather records used in these calculations were obtained at Rothamsted Experimental Station, Hertfordshire, which is to the south west of the main areas of beet production. The precision of the forecasts was not improved by considering records of weather and virus yellows obtained locally in each of the factory areas. On average the percentages of variance accounted for by the regression were doubled and the errors halved when Rothamsted weather data were used rather than local records (Watson *et al.*, 1975). It was inferred that the main influx of aphids to sugar beet is from overwintering areas in southern and eastern England, or even in mainland Europe. This view has been supported by a subsequent analysis of aphid trapping data suggesting that the main areas where *M. persicae* survives are in parts of southern England with relatively high winter temperatures and low rainfall (Taylor, 1977).

Weather conditions that influence the overwintering, behaviour and effectiveness of *M. persicae* as a vector of sugar beet yellowing viruses also influence the spread of other viruses by aphids, which survive as eggs on woody hosts or as anholocyclic populations on

herbaceous plants. *Cavariella aegopodii* is one such species that spreads annually to carrot crops from willow trees, or from umbelliferous plants, including some that are hosts of the viruses causing carrot motley dwarf disease (Dunn and Kirkley, 1966). Hence, yields in annual plantings of carrots, 1959–1965, were usually high in years when yellowing viruses of beet were least prevalent (Watson, 1967). Similarly, viruses of potato transmitted by *M. persicae* and other species became more important than for many years in Britain and other parts of Europe following the sequence of unusually mild winters that were associated with the major epidemics of beet yellows in 1974 and 1975 (Hill, 1978a; Sparrow, 1976).

D. WHEAT STREAK MOSAIC VIRUS

Wheat streak mosaic virus is transmitted by an eriophyid mite (*Aceria tulipae*) and infects wheat, certain millets and maize. The virus was first described in North America and also occurs in Jordan, Rumania, Yugoslavia and Russia (Slykhuis, 1967). It has received considerable attention as the cause of extensive losses in the Great Plains and western areas of winter wheat production in USA and Canada.

There is no evidence that wild or cultivated grasses are important sources of infection and mosaic is only prevalent in areas where susceptible cereals are grown in close sequence throughout the year. Virus is carried into spring plantings by wind-borne mites that disperse from nearby autumn-sown crops as these mature in early summer. Similarly, there is spread later in the year from maturing spring-sown crops to those planted in early autumn. Self-sown "volunteer" plants developing in fallow areas, or from grain shed at or just before harvest, are also important in maintaining a continuous cycle of infection (Slykhuis, 1955).

Weather conditions are of crucial importance in epidemiology. They determine the precise sequence and vulnerability of the crops being grown and also the number of mites dispersing from old to new plantings. This is illustrated by detailed observations made during the major epidemic of mosaic in Kansas in 1959 (King and Sill, 1959). The 1958 crops were some of the highest ever recorded, but grain shed at harvest soon germinated and formed lush growth which became heavily infested with mites during late summer and early autumn. Favourable conditions permitted many crops to be sown early and these were soon invaded by mites that developed enormous populations during a subsequent period of unusually warm weather. Many plants were infected with virus at an early stage of growth and before

the onset of winter. Subsequent growth was poor and losses throughout the State were estimated at 20% of the entire 1959 crop. They would have been even greater if 40% of all plantings had not been of tolerant varieties. These were much less severely affected than sensitive ones with similarly high levels of infection.*

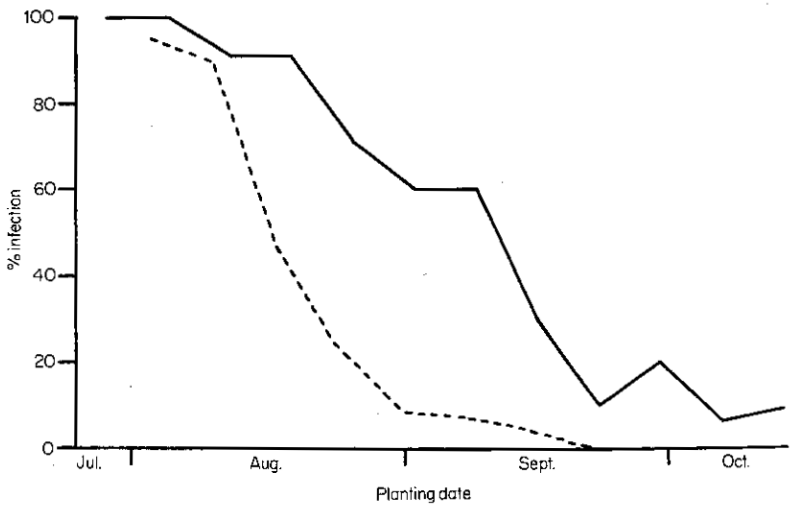

FIG. 4. The effect of sowing date on the final incidence of streak mosaic in plantings of winter wheat in Alberta, Canada. The 1953 plantings (continuous line) were alongside the site of an affected crop of winter wheat which matured in mid-August and the 1954 plantings (broken line) were adjacent to self-sown wheat which was removed by the end of August (Slykhuis *et al.*, 1957).

An unusual sequence of weather was also responsible for the unprecedented epidemics of wheat streak mosaic that developed in Alberta during 1963 (Atkinson and Slykhuis, 1963). Conditions were exceptionally dry from 1 April to 17 June and spring-sown crops failed to germinate or grew badly until a period of heavy rain began on 21 June. Rapid growth then occurred, further sowings were made and there was extensive development of "volunteer" wheat in areas being fallowed. Mite populations increased rapidly and spread virus

* Throughout this paper plants are referred to as sensitive or tolerant according to their reaction to infection. The term resistant is used for plants that are not easily infected. This usage has advantages and has been advocated by several workers. However, many use the terms less discriminatingly and refer to plants as resistant if they are little damaged by virus due to some form of tolerance or because they tend to escape infection. It is particularly difficult to interpret papers in which it is not clear whether virus is present in and can spread from symptomless plants simply described as "resistant".

amongst these late-developing plants, which became potent sources of infection for further spread to autumn-sown crops of winter wheat. Conditions then remained favourable for mite dispersal for an exceptionally long period. September temperatures were almost 5°C above the 30-year mean and there were no damaging frosts until 19 October, which was 24 days later than the 61-year mean. In these circumstances infection became prevalent in most crops sown early and many plants were dying by the end of October. Outbreaks were also encountered in plantings made as recommended during the first two weeks of September (Slykhuis *et al.*, 1957). In normal years this avoids any major carry-over of infection from older crops maturing at the usual time (Fig. 4). Serious losses are usually avoided by so breaking the cycle of infection and by eliminating volunteer growth before sowing commences. However, the 1963 experience emphasizes how control measures that are usually effective can become totally inadequate when weather conditions are exceptionally favourable for disease development.

IV. Viruses Becoming Important After Spread into New Areas

From the foregoing examples it is clear that the first reports of a disease in a particular area seldom provide a reliable indication of when it first occurred there. Symptoms can be overlooked or ignored for many years until they attract the attention of pathologists able and equipped to make a correct diagnosis. This greatly complicates the interpretation of distribution maps and records showing the incidence of diseases and their apparent spread into new areas. However, some diseases cause such conspicuous symptoms in widely-grown crops that they are unlikely to be missed for long, and it is therefore possible to make a realistic assessment of their recent history.

The progress of various fungal diseases has been followed in detail over large areas and even across whole continents. Blue mould of tobacco (*Perenospora tabacina*) in Europe and American corn rust (*Puccinia polysora*) in Africa, are two of the most thoroughly studied of recent examples. There are several comparable instances of viruses spreading into new areas, although the available information is much less precise and more difficult to interpret.

A. Plum pox virus

Plum pox virus causes very conspicuous and characteristic symptoms on the fruit of sensitive cultivars of plum, peach and apricot. Badly

affected fruits are virtually unsaleable and soon attract attention wherever they occur in orchards. This suggests that records of the first occurrence of symptoms in the various countries of Europe provide reliable evidence on the sequence of spread.

Infection was first seen in Bulgaria from 1915–1916 and has since been reported in Turkey and in many European countries to the north and west (Fig. 5). Losses have been particularly great in Yugoslavia, where infection was first reported in 1935. An estimated 16 million plum trees had become infected by 1952, when some areas were almost totally diseased. There have been much less serious problems in Switzerland, Netherlands and England, where the disease has been reported relatively recently (Adams, 1978). However, even in these countries there has been considerable expense and inconvenience to growers and official plant health organizations in mounting major eradication campaigns. These involve the location and destruction of infected trees in commercial nurseries and orchards.

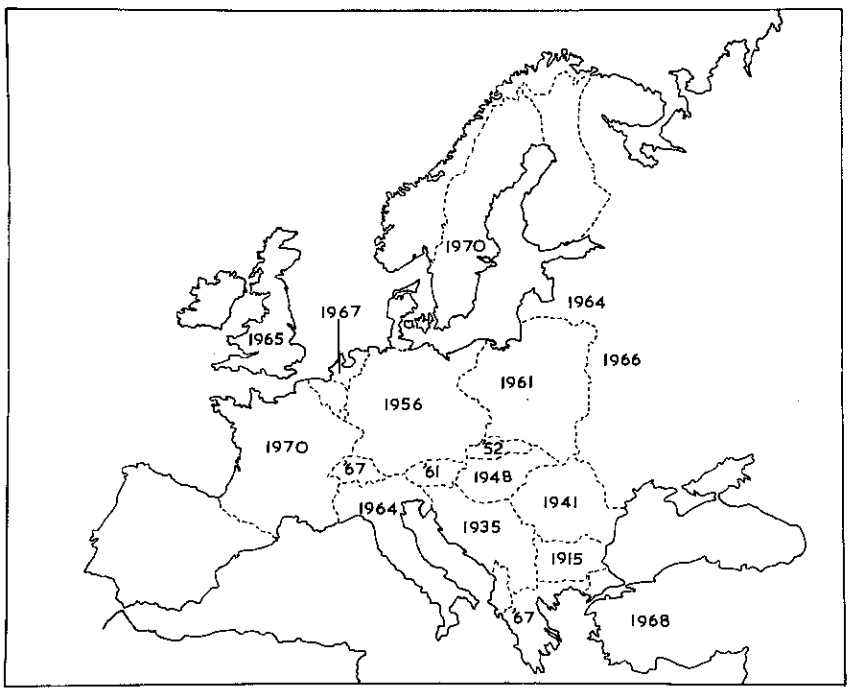

FIG. 5. Sketch map of Europe and western Asia showing the year in which plum pox was first observed in each of the countries where the disease has been recorded (A. N. Adams, unpublished compilation).

Several aphids including *Phorodon humuli* and *Myzus persicae* transmit plum pox virus non-persistently. The main information on their effectiveness in spreading virus into and within crops has come from observations in Yugoslavia (Jordovic, 1975).

Infection appeared first and spread most rapidly in new orchards established adjacent to heavily infected plantings. Elsewhere, spread decreased with increasing distance from the nearest source of infection. At an isolated forest site there was no spread at all until infected trees were planted in the locality. These results suggest that aphids make little contribution to the distant spread of plum pox into entirely new areas. This is due mainly to the dissemination of infected planting material from contaminated nurseries.

Plum, peach, apricot and several ornamentals including the red-leaved *Prunus pissardii*, are all vegetatively—propagated perennials. There has long been an extensive commercial trade in rootstocks, scion material and grafted trees. Much of this traffic is local, but there is also considerable movement between countries. Some of this involves bulk consignments to wholesalers for distribution to numerous outlets which have recently included shops and garden centres supplying amateurs. In addition, small amounts of material have been exchanged between plant breeders, horticulturalists and gardeners for collections and trials.

Until quite recently the stocks being distributed within and between countries were not subject to any official inspection, certification or quarantine procedures. Consequently, there have been abundant opportunities for plum pox to become widely disseminated. It has been suggested that the disease was introduced to Germany by soldiers returning from the Balkan countries during the Second World War (Anon., 1976). Recently Plant Health Inspectors in England and in the Netherlands have detected plum pox in recently imported material which was traced back to infected nurseries in the country of origin.

The evidence suggests that plum pox has been introduced to England from mainland Europe on various occasions over a period of several years. Infected orchards are now being found in increasing numbers and mainly in recent plantings. However, the disease has already spread into long-established orchards and into susceptible plants grown as wind-breaks or hedges. This will make it increasingly difficult to restrict spread and the main efforts of the official Plant Health Inspectors are now concentrated on nurseries in a determined effort to ensure that healthy stocks are available for new plantings (Adams, 1978).

The history of plum pox in Europe provides a striking example of the way in which viruses can be distributed by man at a speed and over distances impossible to achieve by other means. Natural barriers have been penetrated with ease due to inadequate controls on the movement of plant material. This justifies the stringent restrictions on imports of susceptible *Prunus* spp. to North America, where plum pox has not yet been reported.

B. CITRUS TRISTEZA VIRUS

The main areas of citrus production are in regions where the crop has been introduced. There has been much movement of bud-wood and rooted plants and this accounts for the presence of several important viruses in virtually all countries with major plantings (McClean, 1957). Particular problems have been encountered with the aphid-borne virus referred to as tristeza. When this virus spreads to sweet orange trees (*Citrus sinensis*) growing on sour orange rootstocks (*C. aurantium*) it causes a lethal decline (Bennett and Costa, 1949). Some other stock/scion combinations are less sensitive to infection, and they have, consequently, been widely used in South Africa, where tristeza appears to have been present for many years. The use of sour orange rootstocks had to be abandoned, despite their resistance to phytophthora root rot (Webber, 1943).

The reasons for the failure of sour orange as a rootstock and the prevalence of infection in South Africa were not apparent until investigations were initiated into a virus first identified as the cause of tristeza disease in Brazil (Meneghini, 1946) and the similar "quick decline" disease of citrus in California (Fawcett and Wallace, 1946). The extensive plantings of sweet orange on sour orange rootstocks had previously been grown successfully in these areas for many years, suggesting that tristeza had been introduced relatively recently or that it had only just begun to spread.

Tristeza has caused enormous losses in South America where it was first recorded in Argentina in 1930 and subsequently in Brazil (1937), Uruguay (1940), Venezuela (1950) and Paraguay. In the worst-affected areas almost all the vulnerable trees on sour orange rootstocks were killed within a few years or plantations became so worthless that they were abandoned. By 1949 an estimated 6 million trees had been destroyed in the São Paulo state of Brazil alone, and this amounted to 75% of all the orange trees pesent. The oriental citrus aphid (*Toxoptera* (≡*Aphis*) *citricidus*) proved to be a highly efficient vector and infection increased from 12 to 100% within 5

years, in a representative planting of 2606 trees. Spread between regions was facilitated by the distribution of large quantities of plant material, including tolerant varieties and stock/scion combinations (Bennett and Costa, 1949).

There is considerable information on the early history of citrus growing in Argentina, including detailed records of the introduction and wide distribution of two large shipments of nursery material from South Africa between 1927 and 1930. Tristeza could have been introduced at this time and it is known that the imported scionwood failed to develop when grafted onto sour orange rootstocks of local origin, although it grew normally on other varieties. Certainly the disease was already widespread when first discovered, causing the death of many trees in each of the main areas of production (DuCharme *et al.*, 1951).

Imports of citrus material have also been considered in relation to the occurrence of tristeza in various parts of the USA (Wallace *et al.*, 1956). The virus was detected in clones derived from South Africa, China, Japan and Hawaii, which were grown for many years at sites in California or Texas, where little or no spread to adjacent trees had occurred. This is convincing evidence that tristeza has been introduced from various localities, including Asiatic areas where citrus is considered to have originated. Many of the original shipments were small and had passed through the Official Import Station in Washington, DC. However, techniques have only become available relatively recently for detecting tristeza and other citrus viruses in tolerant varieties. This explains why infection was overlooked and became widely distributed by subsequent propagation, or in some localities by aphids. The vectors occurring in the United States (*Aphis gossypii* and *Aphis spiraecoli*) are inefficient and the consequences of introducing tristeza have been less devastating than in South America and other countries where *T. citricidus* occurs.

V. Viruses Causing Problems when Crops are First Introduced to New Areas

Introducing crops or livestock to entirely new areas can have far-reaching and unexpected consequences (Cherrett and Sagar, 1977). Sometimes the introductions flourish in the new environment, where they are free from pests or diseases prevalent in the country of origin (Purseglove, 1963). In other instances catastrophic losses occur when exotic crops are soon attacked by indigenous pathogens not previously encountered. There are also examples of pests or diseases being

introduced with material moved to fresh areas, where they thrive on their original hosts or spread to others already established in the region.

Many plant virus disease problems are likely to have arisen from crop introductions, but there is seldom adequate evidence to provide an unequivocal interpretation of the precise sequence of events (Bennett, 1952). In countries with well-developed systems of agriculture the main crop species were introduced long before virologists were available to recognize any immediate problems arising. Elsewhere, agriculturalists and horticulturalists are currently introducing extensive consignments of material before pathologists are appointed to assess the disease situation and record the viruses already present. An additional difficulty in all areas is that the effects of many viruses are much less conspicuous than those due to insects, mites or fungi which cause extensive necrosis, wilting or defoliation. This means that new viruses are seldom recognized until they have become so prevalent that their origin is obscure. The various problems of interpretation are illustrated by the following examples.

A. CACAO SWOLLEN SHOOT VIRUS

Cacao is an indigenous understorey tree of the Amazonian forests of South America, yet the main commercial areas of production are in Ghana, Nigeria and other West African countries. The first introductions to West Africa were made in the latter part of the 19th century and planting proceeded rapidly, mainly in the lowland rain forest areas. There was a particularly dramatic increase in production in Ghana, from 0·3 tonnes of dry cacao beans in 1891, to 40 640 tonnes in 1911. Many of the individual holdings were small, yet they were so numerous in the eastern region as to form vast almost continuous stands beneath the remaining trees of the selectively-thinned forests.

Shoots with conspicuous swellings were first reported in 1936, associated with a serious die-back of trees in the eastern region (Steven, 1937). The condition became known as swollen shoot and was first attributed to unfavourable soil factors or to environmental changes following deforestation. It was eventually shown to be due to a mealybug-transmitted virus that had become prevalent in many parts of Ghana and in other West African countries (Posnette, 1947). Infection was so widespread when discovered in the worst-affected areas of Ghana that it must have been present for many years and enquiries revealed a long history of dying cacao, almost certainly due to swollen shoot. This suggests that the disease is likely to have

appeared in the eastern region soon after the first plantings in the district around 1907. A generally accepted explanation of this sequence of events is that swollen shoot was introduced to cacao at an early stage by mealybugs originating from indigenous hosts. Several tree or shrub species of the Tiliaceae, Bombacaceae and Sterculiaceae are known to be susecptible (Posnette *et al.*, 1950; Tinsley and Wharton, 1958). Moreover, naturally infected trees of five species have been located and some are commonly infested by mealybug vectors, which also occur on cacao.

Major difficulties have been encountered in obtaining and evaluating evidence on the precise role of indigenous hosts. Many occur in remote, inaccessible areas and there are problems in obtaining satisfactory leaf samples, especially from giant forest trees. It can also be difficult to confirm the presence of virus in suspect plants and those located in areas where swollen shoot is prevalent in crops may have been infected before or after the nearby cacao. Despite these problems there have been important findings in several parts of Ghana.

At an early stage in the development of cacao-growing in the forests of the western province it was found that an understorey tree (*Cola chlamydantha*) was commonly infected with swollen shoot virus (Posnette *et al.*, 1950; Todd, 1951; Tinsley, 1971). Infected trees supporting colonies of mealybugs were detected in and around scattered outbreaks occurring in cacao. Infection also occurred in trees growing in secondary "bush" regenerating after food-crop farming, and in 32 of 80 trees sampled in secondary forest or in forest reserves up to 5 km from the nearest cacao (Fig. 6). The virus isolates from *C. chlamydantha* resembled those from swollen shoot outbreaks in the region. They caused distinctive symptoms in cacao different from those due to isolates from elsewhere in Ghana, where *C. chlamydantha* is absent.

The baobab (*Adansonia digitata*) is another naturally infected indigenous host of swollen shoot and its mealybug vectors (Attafuah and Tinsley, 1958; Dale, 1962). Infected trees are quite common in the derived savannah regions to the north and south of the main forest areas and up to 160 km from the nearest cacao (Fig. 6). Infection has also been found in or around localized outbreaks in the scattered cacao plantings of the Volta region. Infected trees have not been detected in the principal cacao-growing areas, where the few baobabs are mainly planted in or around villages. Moreover, all the isolates from baobab are of the unusual "mottle leaf" type that do not cause swellings and seldom occur in cacao.

These findings suggest that baobabs and *C. chlamydantha* were infected before cacao. They also emphasize the local importance of

these hosts in initiating outbreaks of swollen shoot of cacao in certain areas. Other species may have played an equivalent role in the worst-affected parts of Ghana and elsewhere. However, the situation has long been obscure because of the prevalence of infection in cacao. Infected specimens have been found of three other tree species (*Ceiba pentandra*, *Cola gigantea* and *Sterculia tragacantha*), but all were at sites where there could have been spread from extensive outbreaks nearby (Posnette *et al.*, 1950). This makes it impossible to provide a fully comprehensive account of the origin of swollen shoot virus in all areas (Tinsley, 1971).

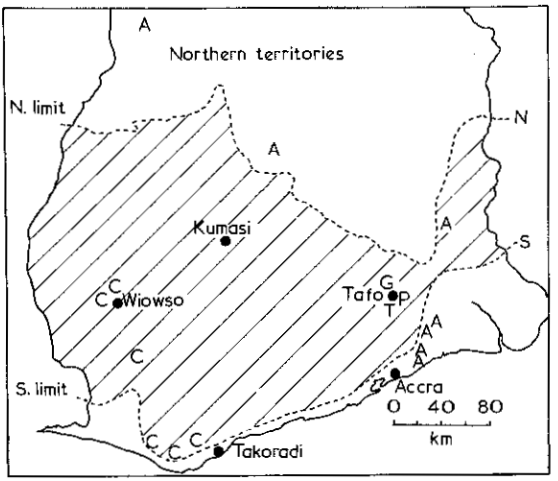

FIG. 6. Sketch map of southern Ghana showing the main cacao-growing area (shaded) and localities where cacao swollen shoot virus has been detected in wild hosts (adapted from Dale, 1962). A = *Adansonia digitata*. C = *Cola chlamydantha*. G = *Cola gigantea*. P = *Ceiba pentandra*. T = *Sterculia tragacantha*.

Whatever its origins, swollen shoot virus has caused incalculable damage to the economy and social structure of Ghana. Millions of infected trees have died in the worst-affected areas and, by 1977, 162 million trees had been cut out in attempts at control by eradication methods (Legg, 1979). At one stage in this costly campaign trees were being removed at the rate of 15 million a year, which is equivalent to 9400 ha at recommended spacings of 1600 trees per hectare. However, it is important to appreciate that it has never been practicable to treat more than a small proportion of the known outbreaks and there were an estimated 42 million infected trees still to be removed by the end of 1979. The fluctuations in cutting out figures (Fig. 7) largely indicate

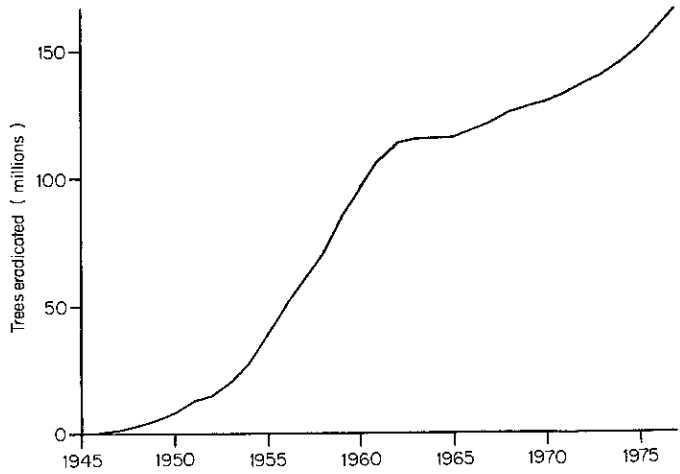

FIG. 7. Cumulative totals of trees eradicated in Ghana between 1946 and 1977 in attempts to control cacao swollen shoot virus (data compiled by J. T. Legg from the unpublished records of the Cocoa Division of the Ministry of Agriculture, Ghana).

changes in the Government resources and personnel allocated to the campaign.

B. RICE YELLOW MOTTLE VIRUS

Rice is widely grown in many parts of the world and the crop has received much attention from virologists, especially in recent years. However, the beetle-transmitted virus designated rice yellow mottle has only been reported from an ecologically isolated part of Kenya. This is in the Kisumu district, around the eastern shore of Lake Victoria (Bakker, 1970). Infected plants were first noticed by smallholders in 1966 and yellow mottle was soon recognized as a potential threat to the major irrigation projects being developed nearby. The limited areas of rice grown previously had been mainly in small, seasonal, rain-fed plantings which provide little opportunity for serious outbreaks to develop.

Irrigation allowed continuous cropping and an extensive growth of grasses, weeds and self-sown rice plants that persisted through the dry season. These conditions facilitated the build-up of virus and beetle vectors and yellow mottle soon became prevalent in some areas. The virus is not seed-borne and infected plants usually appeared first around the margins of large plantings. This indicated that beetles

were introducing virus from outside sources, likely to be indigenous grasses (Bakker, 1974). Several species were shown to be susceptible, including several perennials that survive the prolonged dry season. However, their exact role remains uncertain as the limited attempts to detect naturally infected grasses have been unsuccessful.

Maize streak in Africa (133) and rice hoja blanca in Central America are two other viruses of cereals that may also have originated in grasses. Both viruses are transmitted persistently by leafhoppers or planthoppers and occur in indigenous hosts. Streak does not occur in the New World and had been widespread in South Africa for some years when first described in 1901. It has since been found in grasses, maize or sugar cane elsewhere in Africa and more recently in India (Rose, 1978).

C. SUGAR BEET CURLY TOP VIRUS

The sugar beet industry is of comparatively recent origin and the crop was developed in Europe by breeding from other types of beet in selection work that did not begin until the 18th century. The first factory in America was started in 1837, but this and other initial attempts to establish an industry were unsuccessful. There were repeated failures until the first successful enterprise was founded in California in 1870 (Coons, 1949). In subsequent years severe problems were encountered due to recurrent outbreaks of "curly leaf" and other diseases causing low yields of poor quality beet and sometimes almost total crop failure.

Curly leaf later became known as curly top and first attracted attention in 1899, although it was undoubtedly present earlier (Bennett, 1971). The disease often developed suddenly over very large areas following a heavy influx of leafhoppers into beet-growing districts. This led to a search for primary hosts and to experiments which showed curly top to be caused by a virus persistently transmitted by the beet leafhopper (*Circullifer tenellus*).

Many overwintering hosts of virus and vector were soon discovered amongst the flora of vast areas of uncultivated or occasionally-cultivated parts of California and neighbouring states. Many of the species were introduced herbaceous weeds that thrive when land is overgrazed or abandoned after a period of cultivation. They support enormous populations of leafhoppers that migrate periodically and transport curly top over great distances into beet and other crops. Flights over low ranges of hills and for several kilometres along valleys are regarded as "local". Migrations over 50–100 km are

commonplace and spread has been traced over 640 km (Fulton and Romney, 1940).

Curly top initially caused catastrophic losses to growers and processors (Carter, 1930). In bad years crops were almost totally infected by the initial influx of infective hoppers and whole factories had to be abandoned or closed down temporarily until supplies of beet were again available. Improved systems of land management were devised to decrease the extent of the leafhopper breeding grounds (Piemeisel *et al.*, 1951). Moreover, tolerant varieties were introduced in 1934 and these grew and yielded satisfactorily, even when infected (Coons, 1953). This transformed the situation and made an important contribution to the greatly increased yields recorded between 1935 and 1960 (Fig. 8).

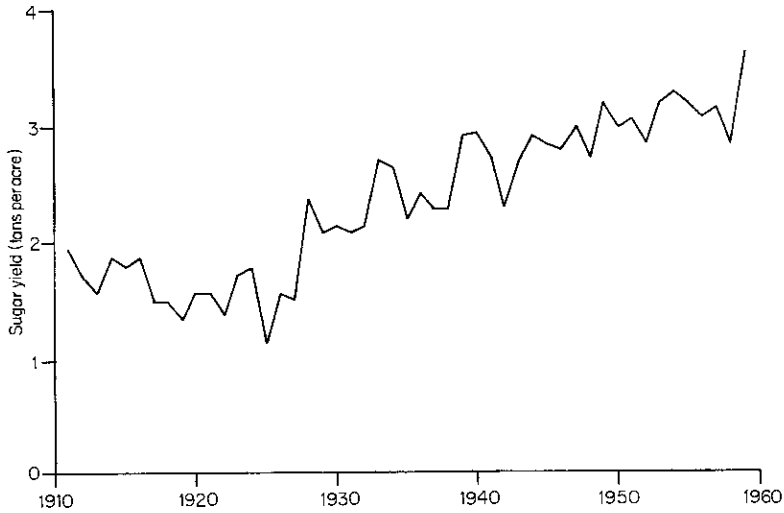

FIG. 8. Sugar beet yields in California, 1910–1960 (data collected by the California Beet Growers Association Ltd. as presented by Duffus (1977).

For some years curly top was known only in the western states of North America and it was assumed that virus and vector were indigenous and that they began spreading to beet soon after the crop was introduced. Diseases resembling curly top were then reported shortly after beet production started in Argentina, Brazil, Chile, Costa Rica and Puerto Rico (Bennett, 1967, 1971). Curly top was also discovered in Turkey, where beet growing began about 1925 (Bennett and Tanrisever, 1957) and in Iran where the disease spread with the expansion of the sugar beet industry (Gibson, 1971). Furthermore,

Oman (1948) observed that *C. tenellus* has no close relatives in North America, although they occur in Mediterranean countries.

These findings illustrate the serious virus problems encountered in many countries where beet has been introduced. They are considered by Bennett (1967, 1971), who discussed the evidence for curly top having originated in the Old World or in the New. There is insufficient evidence on which to base a definitive conclusion, although information is now being obtained on the properties of North American strains of beet curly top virus and it may soon be possible to compare their relationship to those occurring elsewhere. Meanwhile, it has been suggested that virus and vector were introduced to North America, possibly with beet carried as fodder for animals, accompanying some of the earliest settlers from Mediterranean countries (Bennett, 1971). An alternative proposal is that *C. tenellus* was introduced to North America, where curly top was already established in natural vegetation, and transmitted by indigenous leafhoppers as yet unidentified (Oman, 1969). There is certainly ample justification for adopting quarantine measures in attempts to restrict the spread of *C. tenellus* to South America and other areas where it is not yet established. Precautions are also necessary to prevent the introduction of curly top to Hawaii, South Africa and other regions where the vector already occurs.

D. POTATO VIRUS Y

The potato is indigenous to South America, although it is now grown widely in many parts of the world. There has long been extensive movement of tubers within and between countries, which accounts for the widespread distribution of several viruses, including potato virus Y. This virus is transmitted non-persistently by aphids and has a wide host range, causing important diseases of tobacco, tomato and pepper. Serious outbreaks in these crops often occur after initial spread from weed hosts. These can become important foci of infection once the virus has been introduced to localities in potato stocks, as described in Hawaii, Florida and Argentina (Sakimura, 1953; Simons *et al.*, 1956; Pontis and Feldman, 1963).

The history of potato virus Y has been followed in detail in Florida, where observations between 1953 and 1956 established that infection in pepper and tomato was restricted to areas with a history of commerical potato production (Fig. 9). Potatoes had not been grown in the unaffected areas, or they were not introduced until 1955 (Simons *et al.*, 1956).

It was anticipated that infection would appear in the new areas once there had been time for the virus to spread from potato and become established in weeds such as *Solanum nigrum* and *Solanum gracile*. This soon occurred and the first infected peppers and tomatoes were found in 1957, with far more in 1958. By 1959 some crops along the East Coast were totally ruined and overall losses were estimated at over 50% (Simons, 1959). This experience illustrates how serious problems can arise in existing crops of major economic importance, following the introduction of new crops of relatively low commercial value.

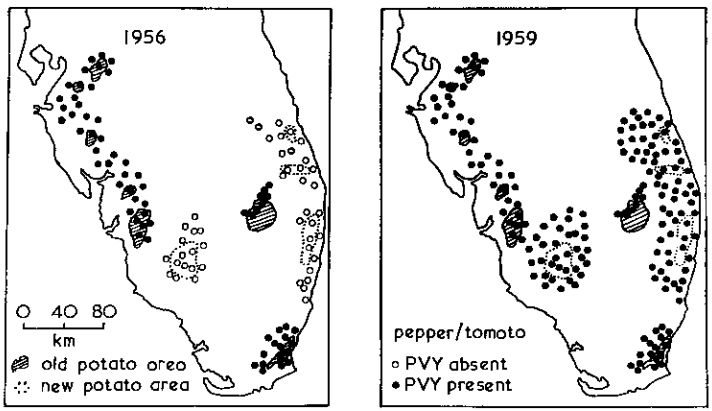

FIG. 9. The incidence of potato virus Y in pepper and tomato plantings in Florida in relation to the main potato-growing areas (Simons *et al.*, 1956; Simons, 1959).

E. VIRUSES WITH SOIL-INHABITING VECTORS

Considerable attention has been given recently to viruses transmitted by soil-inhabiting nematodes or fungi (Harrison, 1977). These vectors move slowly and over limited distances, but an important compensating feature is that they can persist for long periods at infested sites. Many of the species involved and the viruses they transmit also have a wide host range. Natural infection is common in weeds and wild plants, in which the virus can survive between crops or before crops are first introduced.

A particularly striking instance occurred in Arizona when lettuce was the first crop to be planted in irrigated land recently reclaimed from the desert (R. G. Grogan in Campbell, 1965). Big-vein disease affected 60% of the plants and the roots were heavily attacked by the fungus vector (*Olpidium brassicae*).

There are comparable instances of nematode-borne viruses causing problems in Britain and the Netherlands soon after land is brought into cultivation by clearing and ploughing long-established woodland, hedgerows or old permanent pastures. For example, strawberries and glasshouse-grown cucumbers became infected with arabis mosaic virus when planted in former pasture land (Harrison and Winslow, 1961; Dorst and van Hoof, 1965). The nematode vector (*Xiphinema diversicaudatum*) was widely distributed at one such locality and infective populations were detected in adjoining woodland and hedgerows (Pitcher and Jha, 1961). Other outbreaks have developed around the perimeter of plantings, in circumstances suggesting spread from adjoining hedges (Harrison and Winslow, 1961; Taylor and Thomas, 1968).

The situation is similar in parts of North America where tobacco ringspot and tomato ringspot viruses (17, 18) and their nematode vector (*X. americanum*) are widely distributed. Viruses and vectors have been encountered frequently in uncultivated areas, attacking indigenous hosts and introduced weeds from which spread can occur whenever susceptible crops are grown (Tuite, 1960; Frazier *et al.*, 1961; Rush, 1970).

VI. Viruses Favoured by Short-term Rotations and Mono-cropping

The sophisticated techniques of modern agriculture have been evolved gradually from primitive methods of simple food-gathering. An important long-term trend has been from shifting cultivation to more intensive systems of land-use. The change from long to short rotations and in some areas to mono-cropping has had profound effects on the prevalence of many pests and diseases. The impact has been particularly great on those that inhabit soil. Hence the increasing importance of "replant" diseases and "soil-sickness" problems encountered in glasshouses, nurseries, fields and plantations due to nematodes, fungi, viruses or to causes as yet unidentified. Intractable diseases of this type have been encountered in many parts of the world in crops ranging from tomatoes and cereals to fruit and forest trees. This restricts the adoption of intensive cropping systems by specialist growers who may otherwise be well-placed to exploit the advantages of particularly favourable sites or their proximity to major markets. There are serious economic consequences if suitable alternative crops or sites are not readily available.

The major problems that can arise when susceptible crops are

grown repeatedly at the same sites are well illustrated from experience with diseases caused by viruses which persist between seasons in soil-inhabiting vectors or in crop debris.

A. FUNGAL-BORNE VIRUSES

1. *Wheat soil-borne mosaic virus*

Soil-borne wheat mosaic virus causes serious diseases of wheat and barley, which have been recorded in Japan, Italy and the USA. Affected wheat plants were first reported in Illinois in 1919, where almost total crop failures have occurred over very large areas.

It was soon established that plants become infected when grown in soil from affected sites, even after this has been air-dried and stored for three years (McKinney, 1925, 1937). Subsequent work demonstrated the importance of crop sequence in determining the infectivity of soil. This remained high through four successive sowings of susceptible wheat varieties and declined under immune varieties of wheat, oats, maize or legumes. Moreover, steam-sterilized soil became increasingly infectious when mixed with small amounts of contaminated soil and sown with susceptible wheat varieties for four years (Koehler *et al.*, 1952).

These results became explicable when a root-infesting fungus (*Polymyxa graminis*) was implicated (Estes and Brakke, 1966) and eventually shown to be the vector of wheat mosaic. The fungus produces long-lived resting spores that can persist and retain the virus for prolonged periods in the soil. This accounts for the widespread losses encountered in intensive cereal-growing areas of North America, where productivity could not have been maintained but for the development of varieties that resist or tolerate infection.

2. *Wheat spindle streak virus*

Wheat spindle streak is another virus of cereals transmitted by *P. graminis*. It was first found in 1957, affecting autumn-sown wheat in southern Ontario, Canada. Subsequent surveys established great differences between sites and seasons in the severity and prevalence of infection and these are related to soil temperatures and to previous cropping history. Symptoms were particularly conspicuous in 1961, when over 80% of the plants were infected in 68% of the fields examined. Infection occurred almost throughout plantings of winter wheat in the main cereal-growing areas and was rare or entirely absent in fields where winter wheat had been grown infrequently (Slykhuis, 1970).

From these and other observations Slykhuis (1976) concluded that spindle streak is found only in fields in which winter wheat has been grown at least three times at intervals of five years of less. Only scattered plants are affected in the first year that disease appears. Numerous groups of diseased plants occur in scattered patches when the next crop of wheat is grown. Subsequent crops can be almost totally affected.

Wheat is the only host of spindle streak, but the virus can be retained in the long-lived resting spores of the fungus vector for at least five years. This accounts for the serious losses that occur in affected areas wherever cropping practices and soil temperatures are favourable for disease spread and symptom development.

B. NEMATODE-BORNE VIRUSES

1. *Grapevine fanleaf virus*

The grapevine originated in Asia and it is now one of the most important and widely grown of all fruit crops. In the main areas of production, suitable sites tend to be used and reused almost exclusively for vines over prolonged periods. This is done to exploit areas with particularly favourable soils and insolation and to utilize existing terracing, supporting posts and training wires. The practice has obvious advantages despite the serious limitation that it facilitates the spread of viruses transmitted by soil-inhabiting nematodes. Fanleaf is the most important and prevalent of these, but seven additional nematode-borne viruses have been isolated from vines in Europe or North America. Four other viruses of vine also appear to be soil-borne and may have nematode vectors (Martelli, 1978).

Fanleaf, yellow mosaic and related diseases of the vine have been known for many years and referred to by various local names in countries such as France, Germany, Italy and Austria. Symptoms were first mentioned as long ago as 1800 (Vuittenez, 1970) and it has been recognized since 1882 that healthy vines often become infected when planted at sites where diseased ones have been removed. Further work soon demonstrated that infection is soil-borne, although the virus responsible and its nematode vector (*X. index*) were not identified until much later work in North America (Hewitt *et al.*, 1958) and Europe (Cadman *et al.*, 1960).

Fanleaf probably occurs in virtually all the countries where vines are grown and it has already been reported from many parts of Europe, Asia, South America, North America, South Africa and Australia. Such a widespread distribution has been achieved despite

the limited mobility of the vector and the restricted host range of the virus, which has only been found in *Vitis* spp. This emphasizes the importance of the extensive traffic in plant material that has been particularly great since the ravages of phylloxera in the 19th century and the need to use varieties grafted onto resistant rootstocks. Scion varieties, rootstocks and grafted plants have been widely disseminated within and between countries. Many of the early consignments must have been shipped with considerable quantities of soil around the roots and this accounts for the occurrence of virus and vector in the many countries where indigenous host plants are absent.

X. index and fanleaf have been found in very old vineyards in eastern Mediterranean countries and western Asia. Some of these plantings were at remote sites where it is unlikely that plants had been introduced for many years. This led to the suggestion that virus and vector originated with the vine in Asia and were spread with the crop as it was introduced to Europe and later to North America and elsewhere (Hewitt, 1968; Vuittenez, 1970). The overall tolerance of European varieties of *Vitis vinifera* compared with the sensitivity of American species is consistent with this view. Moreover, fanleaf has not been detected in wild populations of *Vitis* spp. occurring in North America. It may also be significant that in South Australia, which is one of the regions least affected by fanleaf, all imports of vines have been prohibited since 1894 (Francki and Crowley, 1967).

Whatever its origin fanleaf now causes serious and intractable problems to many growers. The virus and its vector are introduced to sites with the plants used for new vineyards or to gap-up existing ones. Once established they spread between the roots of neighbouring plants to cause expanding patches of infection. These increase slowly over the years and spread of 1·0–1·5 m per year has been recorded in Californian and Italian vineyards (Hewitt *et al.*, 1962; Lamberti and Martelli, 1965). Such patches ultimately coalesce to form large areas of disease that reappear at the same sites in successive plantings, even if the interval between them is prolonged.

Persistence is facilitated by the ability of *X. index* to survive and retain virus for long periods in the absence of host plants. For example, some individuals survived for 14 months and retained the virus for at least eight months in pots of moist soil (Taylor and Raski, 1964). Even longer periods of survival are possible in vineyards because severed grapevine roots left in the ground can remain alive for years after the tops are removed (Hewitt *et al.*, 1962). This explains the persistence of *X. index* at a Californian site where infected vines were removed and the land was used for five successive crops of winter

barley with summer fallow. Roots were present to a depth of at least 2·4 m and some were still alive and contained the virus after five years (Raski *et al.*, 1965).

In these circumstances it is hardly surprising that growers with badly affected sites have great difficulty in breaking the sequence of infection. Few are likely to be able to adopt the recommended fallow period of 10 years (Vuittenez, 1970). An alternative approach is to use shorter fallows together with soil fumigants and measures to hasten the degeneration of severed roots. Some success has been achieved with nematicides, although treatment is expensive and requires special equipment. Moreover, there are problems in obtaining the necessary kill to the required depth, especially at sites where the soils are very deep or heavy (Raski and Schmitt, 1972). This accounts for the continuing importance of fanleaf in the traditional vine-growing districts and justifies the efforts being made to provide healthy stocks of planting material for use in establishing new areas (Over de Linden and Chamberlain, 1970; Uyemoto and Welsh, 1974). Several nematode-borne viruses have already been encountered in Ontario, New York State and Michigan (Dias, 1977; Ramsdell and Myers, 1974; Uyemoto *et al.*, 1977). They could become increasingly prevalent unless growers are more discerning than in the past in their choice of sites and planting material.

2. *Arabis mosaic virus* (*hop strain*)

The hop is a vegetatively propagated climbing perennial that is grown in many temperate countries to provide a basic raw material for the brewing industry. Expensive supporting posts and permanent wirework have to be provided and plantings are at carefully selected sites around buildings housing picking machines and drying equipment. There is only limited opportunity for crop rotation and particular sites are often retained for many years as they are used and reused repeatedly with little or no interval between successive plantings (Fig. 10). This greatly facilitates the spread of nettlehead and two other important diseases caused by arabis mosaic virus.

Nettlehead was first described in the 16th century (Scot, 1574) and the disease continues to cause serious losses in England. It also occurs in some other countries and is often associated with severe split leaf blotch and bare bine diseases (Thresh *et al.*, 1972). All three diseases tend to appear in distinct patches that spread slowly and reappear at the same sites in successive plantings. However, these observations were inexplicable until quite recently, when the unusual hop strain of arabis mosaic virus was isolated from affected plants and transmitted

by the dagger nematode *X. diversicaudatum* (Bock, 1966; Valdez *et al.*, 1974).

X. diversicaudatum is a long-lived species and populations are extremely stable, responding slowly to a change of crop or even to prolonged periods of bare fallow (McNamara and Pitcher, 1977). Moreover, nematodes that have become infective after feeding on the roots of infected plants can retain the virus for several months. This accounts for the reappearance of disease in successive crops when growers allow only a short interval between plantings. The virus can persist for even longer periods in the many hop plants that regenerate from pieces of stem left in the ground when the original crop is removed. Such plants can survive for several years in grass or weed fallow or even in subsequent crops such as potatoes or cereals, unless special measures are adopted. Their importance has now been appreciated and fallowing procedures have been used with or without soil fumigants as the basis of highly successful measures intended to eliminate populations of the nematode vector or render them non-infective (McNamara *et al.*, 1973; Thresh and Ormerod, 1974).

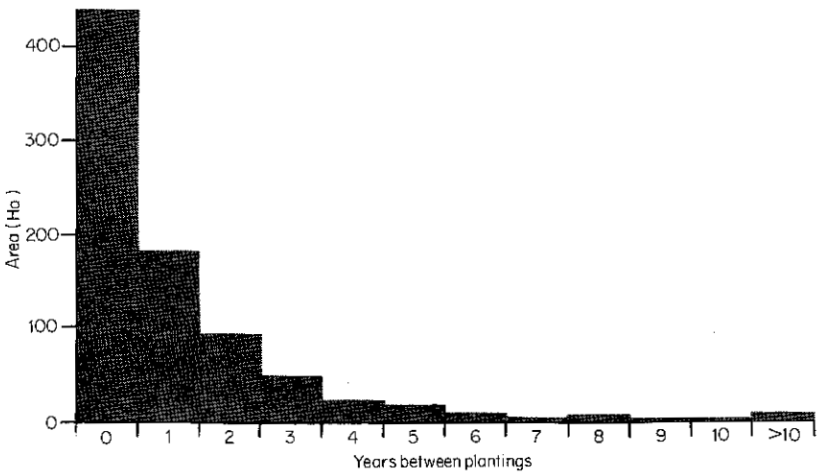

FIG. 10. The distribution of hop plantings according to the interval between grubbing and replanting sites in England. Data from the results of comprehensive surveys of cropping practices, 1968–1973 (Thresh and Ormerod, 1974).

The general adoption of these measures will permit growers to select the most suitable sites for all new plantings, irrespective of their previous history of nettlehead and related diseases. Previously there was little alternative but to abandon hop-growing at sites where serious problems were encountered and became progressively worse

with successive plantings. It was expensive and inconvenient to set up new posts and wirework at fresh sites and suitable ones were not always available.

C. ARTHROPOD-BORNE VIRUSES

Several important arthropod-borne viruses persist between crops or seasons in the debris of previous plantings. This greatly facilitates spread, which is closely related to the type and sequence of crops grown.

1. *Potato viruses*

The major problem posed by potato "groundkeepers" has received considerable attention in many countries. These plants regenerate profusely from small tubers left in the ground at harvest and persist for years in subsequent planting of cereals and other crops. They do not grow vigorously and are seldom infested with aphids, but they perpetuate any viruses that are present and act as important foci of infection from which spread occurs whenever potatoes are again planted at the site (Doncaster and Gregory, 1948).

Surveys in the main potato-growing areas of eastern England between 1944 and 1946 first revealed the full magnitude of the problem and the slow decline in the number of groundkeepers that occurs when sugar beet, cereals or other arable crops are grown after potato (Table II). At the time of these observations fields were being used for potato about once every three years and with such limited rotations groundkeepers were seldom entirely eliminated. Rapid spread of potato leafroll and potato virus Y occurred at one site where infected groundkeepers were particularly numerous. A planting of certified stock became almost totally infected within a single season, compared with 9% infection in "clean" ground about 150 m away.

TABLE II Numbers of potato "groundkeepers" recorded in arable crops surveyed in eastern England, 1944–1946 (data from Doncaster and Gregory, 1948)

Years from previous potato crop	Fields examined	"Groundkeepers" per ha
1	14	10468
2	14	4476
3	16	1936
4	6	469
5	4	182
6	1	0

Potato groundkeepers have continued to cause difficulties, despite the increased use of herbicides on cereals and other arable crops (Lumkes, 1974; Lutman, 1974, 1977). Current methods of mechanical harvesting leave more tubers in the ground than formerly and mortality was low during the mild winters of 1970–1975. This was an important factor contributing to the epidemics of aphid-borne virus diseases occurring subsequently in many parts of Europe (p. 12).

2. *Sugar beet viruses*

The sugar beet is another crop which regenerates from debris left in the ground at harvest or in the piles of refuse that accumulate at receiving stations. As with potato the numbers surviving depend on the severity of the ensuing winter and on various cultural practices. Their impact has been considered in Washington State, where "volunteer" beet provide initial foci of infection from which beet mosaic and yellowing viruses are spread by aphids (Wallis, 1967; Howell and Mink, 1971a).

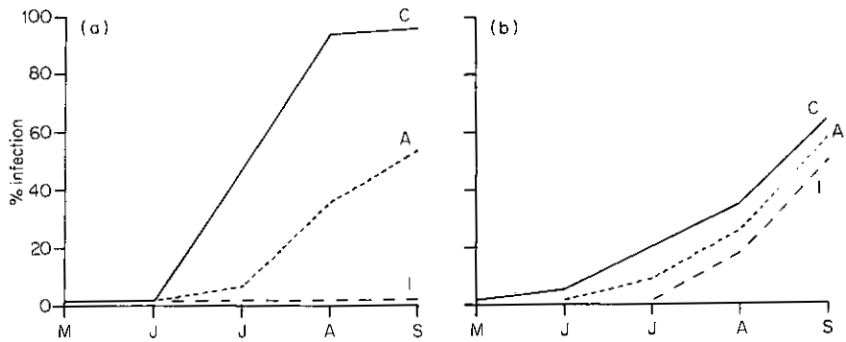

FIG. 11. The spread of sugar beet mosaic (a) and western yellows (b) during the 1968 season in fields containing (C), adjacent to (A) or isolated from (I) volunteer beets regenerating from previous crops (Howell and Mink, 1971a).

Infection appeared first and spread most rapidly at or alongside sites where beet had been grown the previous year and many volunteers had survived. The proximity effect was particularly marked with mosaic (Fig. 11), which is transmitted non-persistently and is seldom prevalent unless there are many local sources of infection. In Washington these are greatly decreased by delaying ploughing until the spring so that debris is left exposed throughout the winter to ensure maximum mortality. This practice has been advocated as one of several measures to decrease the carry-over of

inoculum and permit growers to continue intensive cropping systems in favourably situated areas around beet factories (Howell and Mink, 1971b).

3. *Cotton leaf curl virus*

It has been necessary to modify cropping practices in the large irrigated areas of the Sudan Gezira to decrease the losses caused by cotton leaf curl. This is transmitted by whiteflies and plants regenerating from the stumps of previous stands provide the main initial foci of infection. Their importance was recognized at an early stage of the project and special methods of stump removal were introduced, together with rotations involving periods of bare fallow. These were strictly enforced as basic measures of disease control that also decreased the losses due to bacterial "black arm" disease (Tarr, 1951).

In the arid conditions prevailing in the Gezira survival between crops depends on the severity of the dry season. It is least in dry years and when irrigation water is withheld prematurely, which explains why leaf curl tends to be most prevalent after seasons of above-average rainfall (Boughey, 1947).

4. *Other viruses*

Other disease problems arising from crop debris have been encountered by vegetable growers in Washington State, where much of the initial spread of motley dwarf and thin leaf viruses is from regenerating carrots left in the ground at harvest (Fig. 12). Many survive the cultivation and herbicide treatments given to subsequent crops and aphids readily spread virus to nearby carrot plantings (Howell and Mink, 1977). Similarly, in New Zealand, where wheat is often planted immediately after ploughing grass leys, and scattered patches of disease occur due to the spread of barley yellow dwarf virus from surviving plants of perennial ryegrass (Smith, 1963). Regeneration from roots left in the ground also hinders attempts to eradicate plum pox from orchards by removing infected trees (Adams, 1978).

Additional difficulties with short rotations and intensive cropping systems are due to volunteer plants developing from seed shed at harvest. These can support vector populations and become infected, so maintaining the cycle of infection between growing seasons, when crop plants are scarce or not available. Survival in this way can be of crucial importance in epidemiology as indicated by experience with wheat streak mosaic (p. 12), rice yellow mottle (p. 22) and rice tungro diseases (John and Prasada Rao, 1971).

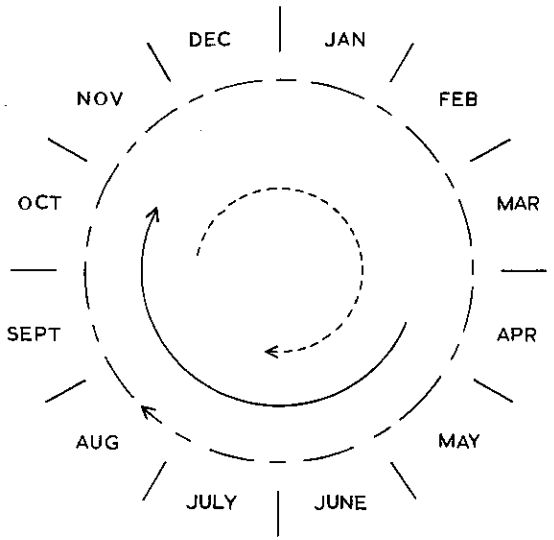

FIG. 12. Cyclical calendar indicating the growing seasons of carrot root (———) and seed crops (—·——) and of volunteer carrots (·····) in central Washington, USA (Howell and Mink, 1977).

The role of volunteer plants has been assessed in detailed studies on groundnut rosette in Africa. Alternative hosts of this aphid-borne virus have not been found and infection persists between seasons in plants that develop from unharvested seed. Self-set populations up to 234 000 per ha have been recorded in Tanzania, where many plants survive the dry season and can be found with aphids and virus symptoms. This accounts for the prevalence of rosette when groundnuts are planted at or alongside sites where they have been grown the previous year (Evans, 1954). Major problems have been encountered with short-season varieties, which germinate at or even before harvest, whilst the soil is still moist. Long-season types have a dormancy requirement which extends into the dry season, but they are inherently low-yielding. Other examples of the way in which the prevalence of disease is related to the varieties grown are considered in greater detail in the following section.

VII. Viruses Causing Serious Losses in Particular Cultivars

With many crops there are great differences between cultivars in their susceptibility or response to infection. This has a major and sometimes dominant impact on the prevalence of diseases and the losses

they cause. Hence the many reports in the literature of serious outbreaks being largely confined to certain cultivars or occurring soon after new ones are released. The importance of these aspects of epidemiology has long been apparent to pathologists and plant breeders working with fungal diseases. They have attracted increased attention after the devastating epidemics of southern corn leaf blight (*Helminthosporium maydis*) in the United States during 1970. These were associated with the release of hybrid maize varieties developed by using certain male-sterile lines later found to have introduced extreme blight susceptibility (Moore, 1970). Other problems have arisen in a similar way and this has led to considerable discussion on the implications of current trends in plant breeding. Particular emphasis has been placed on the consequences of developing a few cultivars of similar origin for use on a large scale in many different countries, where they tend to replace the numerous and diverse locally-adapted selections that have been grown satisfactorily for many years.

There are many examples of the importance of host response in relation to virus disease, and cacao swollen shoot would not have caused so much destruction in West Africa (p. 19) if trees of the Amelonado type had not been so widely grown. Almost all the early plantings were derived from a few initial introductions originating from areas where swollen shoot does not occur. They proved to be highly susceptible and sensitive to infection and were killed by the most prevalent strains of virus occurring in Ghana. These had less drastic effects on some of the types introduced later in attempts to obtain sources of resistance (Posnette and Todd, 1951).

Another striking instance of host vulnerability was observed in Illinois, where wheat soil-borne mosaic (p. 28) caused devastating losses when a susceptible cultivar was first grown at a site where the disease had not previously attracted any attention (Koehler *et al.*, 1952). More recently red clover necrotic mosaic virus (181) was first reported in England from trial plantings incorporating some of the newly-introduced tetraploid cultivars of early-flowering broad red clover (Bowen and Plumb, 1979). Hungaropoly and three other cultivars of similar genetic background were highly susceptible compared with many of the diploid types grown previously. The tetraploids were also vulnerable because they persisted for relatively long periods and so provided increased opportunities for infection to occur. This is likely to impair the performance of the new cultivars and restrict their use as reported with some of the other crops now considered.

A. TURNIP MOSAIC VIRUS

Turnip mosaic virus is transmitted non-persistently by aphids and causes prevalent diseases of cruciferous crops in many parts of the world. It was not reported in lettuce until 1966, when damaging outbreaks occurred in California (Zink and Duffus, 1969). They were restricted to a recently-introduced crisp-head cultivar "Calmar" which was being grown widely because of its resistance to downy mildew (*Bremia lactucae*). In comprehensive tests on a wide range of material all seven downy mildew-resistant cultivars of crisp-headed type were found to be susceptible to turnip mosaic virus, whereas all others were immune (Table III).

TABLE III The reaction of downy mildew-resistant and susceptible lettuce cultivars of different type to turnip mosaic virus (Zink and Duffus, 1969)

Lettuce type	Mildew resistant	Mildew susceptible
Crisp-head	7/7	0/40
Butterhead	0/4	0/11
Leaf	0/2	0/7
Cos	0/1	0/3
Latin	—	0/2
Stem	—	0/2

[1] The number of cultivars of each type that were susceptible to turnip mosaic virus as a proportion of the total number tested.

There was no segregation within populations of each cultivar and an examination of the available pedigrees suggested that mosaic-susceptibility was introduced to lettuce from two mildew-resistant lines of a closely related wild species (*L. serriola*). Different sources of resistance had been used in developing the mildew-resistant cultivars of butterhead and other types that were immune to mosaic.

The apparent linkage between the genes for mildew resistance and mosaic susceptibility in crisp-headed cultivars was confirmed in subsequent work (Zink and Duffus, 1970). This showed both reactions to be due to dominant genes designated *Tu* and *Dm*. There was some crossing-over and the linkage was broken with relative ease, indicating that it would not be difficult to select crisp-headed varieties resistant to both mildew and mosaic.

B. MAIZE ROUGH DWARF VIRUS

Maize was introduced from the New World to the Old over 400 years ago, and has been cultivated extensively in Italy and other Mediter-

ranean countries where rough dwarf disease occurs. However, the disease has a very recent history and its first appearance was closely associated with the introduction of high-yielding American hybrid varieties soon after the Second World War (Harpaz, 1972).

Trials of the new varieties started in Italy in 1946 and the first commercial plantings totalling 1500 ha were in 1948, when rough dwarf disease was first recorded. It attracted little attention until 1949, when outbreaks were so serious as to threaten the whole future of hybrid varieties. These were conspicuously more susceptible than those grown previously and 90% infection was reported in one area of northern Italy where local varieties were virtually immune (Trebbi, 1950; Grancini, 1962).

There was a similar sequence of events in Israel, where hybrid varieties were first released in 1952. Plantings totalled 7000 ha by 1957, when rough dwarf disease was first recorded. In 1958, major outbreaks occurred in all regions where hybrid varieties had recently been introduced. Up to 75% infection occurred in plantings along the Coastal Plains, with severe dwarfing and much premature death. Infection was only 5–7% in local open-pollinated dent varieties grown under similar conditions.

Maize rough dwarf is caused by a virus that is transmitted persistently by planthoppers. The species involved (*Laodelphax striatellus*) does not thrive or readily acquire virus when feeding on maize, which is not a breeding host (Harpaz, 1972). Consequently, all spread is into or within crops by hoppers that breed and acquire virus whilst feeding on grasses. These occur as weeds within crops or in adjacent cultivated or uncultivated areas. In Italy, perennating host plants of rough dwarf virus have not been found and infection persists between growing seasons in hoppers that overwinter as nymphs in diapause (Conti, 1972). These mature and disperse in spring, when virus is introduced to annual grasses and to early plantings of maize. Young seedlings are particularly susceptible to infection and rough dwarf is most prevalent in years when there is a heavy influx of infective migrants soon after sowing. Late sowings are less severely affected and transmission is impeded by a decline in the virus content of infective hoppers at high summer temperatures (Klein and Harpaz, 1970). This restricts spread during the hottest months in countries such as Israel, where losses are decreased by sowing early or late to avoid young seedlings becoming infected when they are most vulnerable (Harpaz, 1972).

Maize rough dwarf provides a striking example of the way in which new and unexpected problems can occur when long-established

varieties are replaced. Rough dwarf does not occur in North America and the hybrid varieties released to Mediterranean countries had not previously been exposed to infection. Consequently, it is hardly surprising that they were severely affected by comparison with established local varieties. These were so resistant or tolerant that infection was entirely overlooked until susceptible genotypes were grown.

C. RICE TUNGRO VIRUS

The first introduction of modern varieties to a region is frequently accompanied by major changes in traditional cropping practices. This makes it difficult to assess whether any immediate increase in the prevalence of pests or diseases is due to the exceptional vulnerability of the new genotypes or to other causes. The situation can be extremely complex as illustrated by the recent history of rice tungro disease.

What appears to be tungro has been known in some Asian countries for many years under various local names (Sōgawa, 1976). These include "mentak" which has been recognized in Indonesia since 1869 and "penyakit merah", first reported in Malaysia in 1934. The causal virus and its leafhopper vectors (*Nephotettix* spp.) received little detailed attention until 1963, when serious damage occurred in experimental plantings at the International Rice Research Institute (IRRI) in the Philippines (Rivera and Ou, 1965). Serious epidemics have occurred subsequently in the Philippines and in several other Asian countries (Fig. 13). These have been attributed to the widespread use of certain IRRI varieties (Buddenhagen, 1977), although this is but one of several inter-related factors involved.

The recently-introduced high-yielding varieties differ from many traditional ones in being heavy-tillering, photo-insensitive and of short growing season and stature (Khush, 1977). These features facilitate intensive methods of cultivation, using dense stands and artificial fertilizers, with increased use of irrigation to extend the natural growing season and permit successive crops to be grown in close or even overlapping sequences. Such conditions favour the build-up of pests and diseases, including some that seldom damaged old varieties grown by traditional methods (p. 22). Some of the new varieties soon proved to be vulnerable. For example, IR8, IR5 and especially IR22 were severely attacked in the Phillipines following their release between 1968 and 1970. In Malaysia, unprecedented losses occurred in a traditional long-season variety following an early

and heavy influx of leafhoppers from previous plantings of new short-season types (Lim, 1972). Infection was also reported for the first time in India and became prevalent in certain areas immediately after the use of T(N)-1 and its derivatives since 1964. These varieties proved to be highly susceptible to leafhoppers and planthoppers which previously had been only sporadic pests of rice in India (Kulshreshtha *et al.*, 1970).

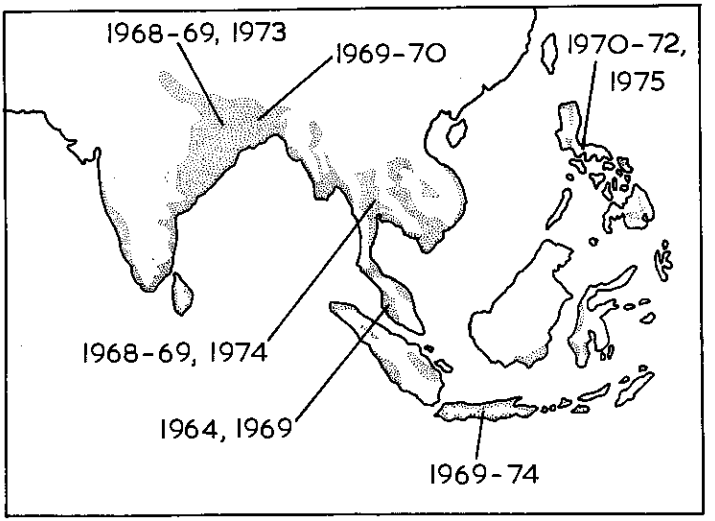

FIG. 13. Some recent epidemics of rice tungro disease in Asia. Stippling indicates main rice-growing areas (from Sōgawa, 1976).

The high-yielding varieties developed in recent years now account for over 25% of all plantings. Over large areas a few modern types have almost completely replaced the traditional ones grown previously. There have been equally dramatic changes in cultural practices and these have greatly increased the threat of pest and disease attack. This poses additional problems for the plant breeder and the recent losses caused by tungro and its leafhopper vectors emphasize the need for varieties that are more resistant than those grown previously (Khush, 1977).

D. HOP MOSAIC VIRUS

The many different hop varieties grown in England can be divided into two distinct groups according to their reaction to hop mosaic virus. When Golding varieties become infected they develop con-

spicuous symptoms and usually die or grow so badly that they have to be replaced. All other varieties are extremely tolerant and fail to produce symptoms, even though all established clones are infected throughout.

Hop mosaic virus is aphid-borne and serious outbreaks are likely to occur in Goldings whenever they are planted close to sources of infection such as other varieties, or when tolerant male plants are used as pollinators (Fig. 14). This became apparent at an early stage of the investigations which began soon after mosaic was first recorded in England between 1907 and 1910 (Salmon, 1923; Mackenzie *et al.*, 1929; Keyworth, 1947). It was soon established that losses could be avoided or kept to acceptable levels by growing Goldings with appropriately sensitive males at isolated sites or on entirely separate farms. Such measures are effective but not always convenient and it is expensive for nurserymen to grow Goldings away from other varieties in order to avoid any risk of mosaic and to qualify for the official certification scheme. Moreover, recent changes in market demand have induced some growers of Golding varieties to plant tolerants for the first time and heavy losses have occurred (Thresh, 1979a).

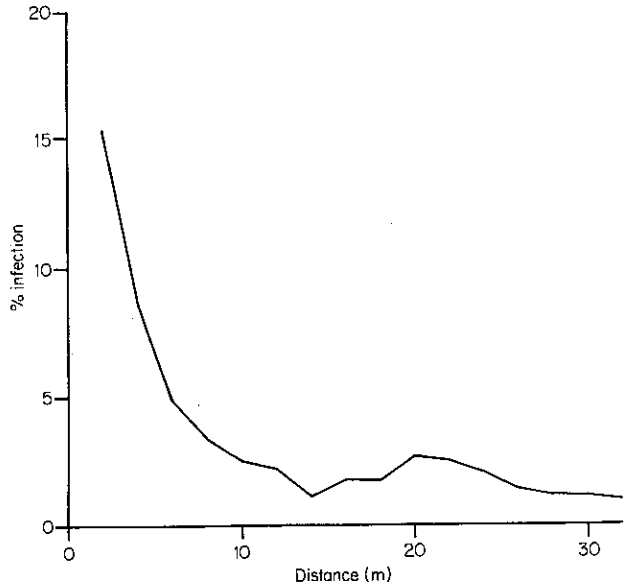

FIG. 14. The incidence of hop mosaic disease in a sensitive hop cultivar at increasing distances from an adjacent planting of symptomless tolerants (J. M. Thresh, unpublished data).

There is a similar problem in West Germany, where mosaic appeared recently at isolated sites in the Hersbruck area of Bavaria, soon after tolerant varieties were introduced from the main hop-growing areas of the Hallertau. Losses also occurred in the traditional Hersbruck variety when it was introduced to the Hallertau (Thresh, 1979b). There is no detailed information on the incidence of mosaic in Hallertau varieties, but they could have been responsible for introducing the virus to England. Some of the first outbreaks ever recorded were in variety collections and at a farm trial near recently introduced German material (Mackenzie *et al.*, 1929).

E. SUGAR CANE FIJI VIRUS

Additional ways in which disease problems are closely related to the vulnerability of the varieties being grown are illustrated by recent experience with sugar cane Fiji disease in Bandaberg and other parts of Queensland, Australia. This disease is caused by a leafhopper-borne virus that first attracted attention in the 1920s, at a time when current varieties were being replaced by POJ2878, which was less susceptible to a prevalent bacterial gumming disease. The new "wonder" cane proved to be highly susceptible to Fiji disease, which caused continuing difficulties despite well-organized control measures involving inspection, eradication and steps to safeguard the health of the vegetative material used for new plantings (Toohey and Nielsen, 1972).

The published figures for stools removed underestimate the magnitude of the losses as they exclude data for the worst-affected fields that were destroyed before counts were made. Nevertheless, they reveal a peak of infection during the 1944–1945 season, followed by a decline as POJ2878 was replaced by a less susceptible variety and eventually by resistant ones (Fig. 15). Losses became insignificant until there was an upsurge of disease in the late 1960s following an increase in leafhopper populations and the introduction of higher-yielding varieties that were less resistant to infection.

Particular problems have been encountered with the widely-grown N.Co.310, which is prone to attack by leafhoppers and moderately susceptible to infection with Fiji virus. Infected plants are slow to develop obvious systems and difficult to find by plant health inspectors responsible for routine surveys. This has decreased the effectiveness of eradication measures and by 1976 it was estimated that there were 10 million infected stocks in the Bundaberg area alone (Fig. 15). Virtually all farms were affected and thousands of hectares had to be

ploughed out prematurely. In some localities it has become difficult to produce adequate supplies of healthy planting material, and for any real solution to the problem it is considered essential to replace N.Co.310 by less susceptible varieties. Meanwhile, losses already total thousands of tons of sugar per year and are still increasing (Egan, 1976; Egan and Fraser, 1977). There is a striking parallel with the situation that arose in Europe when the tobacco veinal necrosis strain of potato virus Y became prevalent and made it extremely difficult to maintain healthy stocks of certain potato varieties (p. 46).

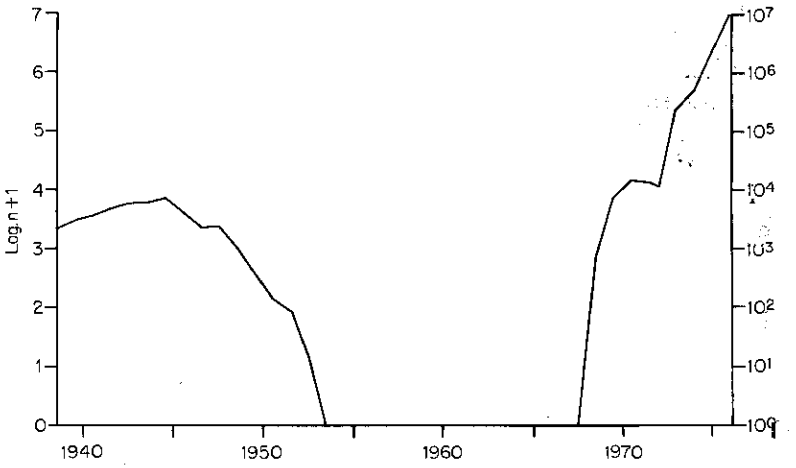

FIG. 15. Counts or estimates of the number of sugar cane stools affected by Fiji disease in the Bundaberg district of Queensland, Australia, 1939–1976 (Egan, 1976; Egan and Fraser, 1977). Note the use of a logarithmic scale to show the very wide range of values encountered.

F. VIRUSES OF FRUIT CROPS

Citrus tristeza would not have caused such devastating losses or attracted so much attention in South America but for the sensitivity of sweet orange trees when grown on sour orange rootstocks (p. 17). There is a somewhat similar situation with plum pox (p. 14), which causes serious diseases of Požegača and Victoria. It is far less damaging in rootstocks and in many other cultivars that have at least some degree of tolerance.

Many other examples of tolerance have been encountered in work on fruit crops (Posnette, 1977). This is inevitable because suitable cultivars are propagated vegetatively and retained for many years.

Any that are sensitive to commonly occurring viruses are likely to grow or crop badly. They are soon discarded by breeders or horticulturalists, even though the reasons for the unsatisfactory growth are not always apparent. For instance the use of sour orange rootstocks was abandoned in South Africa and some other countries long before the effects of tristeza were elucidated (p. 17). More recently several types of quince were found to be unsuitable for use as pear rootstocks in England because of "incompatibility" effects (Hatton, 1928), shown later to be due to the sensitivity of the quinces to viruses prevalent in pear varieties (Cropley, 1967).

The existence or prevalence of several other important viruses of fruit crops was not appreciated until serious problems were encountered after attempts to introduce new rootstock or scion varieties. This is illustrated by experience in North America where the use of Virginia Crab and Spy 227 as apple rootstocks or inter-stocks has been restricted by their sensitivity to viruses found to be widespread but latent in many commercial varieties (Gardner *et al.*, 1946; Tukey and Brase, 1943). Similarly, apple chat fruit and rubbery wood diseases were not known until a sensitive cultivar (Lord Lambourne) was introduced and revealed the prevalence of infection in rootstocks and other varieties (Luckwill and Crowdy, 1950).

VII. New Strains of Virus Causing Particular Problems

There are many examples in the plant pathological literature of major problems encountered after the appearance of new strains of fungi that are particularly aggressive or able to overcome the usual control measures. This aspect of epidemiology has received considerable attention following the emergence of strains tolerant of recently-introduced fungicides or able to break down host resistance factors.

Viruses also have the capacity to generate variants by mutation and in some instances by the production of pseudo-recombinants. Various biological or physico-chemical criteria are used to distinguish the strains occurring and differences have been reported in such features as host-range, virulence and transmissibility by vectors. For example, transmission tests with several aphid species are used to distinguish strains of barley yellow dwarf virus and differences of considerable epidemiological significance have been established between regions and between seasons in the predominant strains occurring (Rochow, 1969, 1979; Rochow and Jedlinski, 1970).

There is likely to be a comparable situation with many other

viruses, although it is seldom practicable to make very comprehensive surveys of strain distribution because of the lack of suitable techniques for dealing with numerous samples. Only limited use has been made of differential hosts to categorize virus strains in the detailed way that has become customary in comparable work with rusts, mildews and many other fungi. One of the main reasons for this is that there have been relatively few attempts to breed for resistance to viruses and there has been little opportunity to assess the long-term behaviour of resistant genotypes when grown commercially on a large scale.

Despite these limitations there are several instances of virus strains with distinctive characteristics appearing and causing important diseases of species or cultivars previously considered to be immune or little affected. Examples have been encountered in work on vegetables and field crops (Holmes, 1965), and with several other viruses there is evidence of the complex factors influencing the survival and prevalence of strains. For example, a strain of raspberry ringspot virus has been isolated in Scotland from the raspberry variety Lloyd George, which is immune to the usual strain of virus occurring there (Murant *et al.*, 1968). The aberrant strain has a restricted distribution and this has been attributed to a lack of competitive ability associated with limited invasiveness and low rates of seed transmission in weed hosts (Harrison, 1978).

The tobacco veinal necrosis strain of potato virus Y has been far more successful and its origin and spread have been discussed by Klinkowski (1964). The strain was first isolated in England in 1935, but was not known from mainland Europe until reported from Bulgaria in 1950. It has since become established and caused major epidemics in potato or tobacco plantings, in many parts of Europe, due to spread by aphids and in tubers. The veinal necrosis strain decreases potato yields without causing obvious symptoms and this makes it extremely difficult to control by long-established certification and roguing procedures (Todd, 1960). In the worst-affected regions of Europe there has been no alternative but to replace several popular and widely-grown varieties.

Another striking development has been in Israel where citrus tristeza virus has been present for many years and yet has only recently begun to spread and cause major problems. This has been attributed to the emergence of strains that are more readily transmitted by aphids than those previously occurring (Bar-Joseph, 1978). Other problems due to the appearance of new strains have been encountered in detailed studies on viruses of maize and glasshouse tomatoes.

A. MAIZE DWARF MOSAIC VIRUS

Sugar cane mosaic virus (88) is prevalent in cane fields and caused devastating losses in many counties, until tolerant varieties were introduced (Klinkowski, 1970). Numerous strains of the virus have been distinguished but those reported in early studies do not readily infect Johnson grass (*Sorghum halepense*) and they were seldom encountered in maize, except in plantings alongside sugar cane.

The position has changed dramatically in recent years and sugar cane mosaic virus now causes a severe disease of maize in many parts of the United States, including northern areas far from the nearest canefields. The disease and the causal virus are referred to as maize dwarf mosaic, which was first seen in 1962, and affected a few plants in Ohio. Dwarf mosaic reappeared in the same area and elsewhere in 1963, when infection was also reported in California and Arkansas. Twelve counties were affected in Ohio, mainly along the southern river valleys, where there was up to 50% infection in some fields and late plantings of sweet corn were severely damaged (Janson and Ellett, 1963).

By 1964 dwarf mosaic had been recorded in Illinois and in many parts of Ohio, where total losses were estimated at 5 million bushels of grain (Ellett *et al.*, 1965). The virus responsible was found to be related to sugar cane mosaic and in many areas the perennial Johnson grass was an important overwintering host (Shepherd, 1965; Williams and Alexander, 1965). However, additional strains of virus have been isolated from maize that do not infect Johnson grass, which has a more restricted distribution than dwarf mosaic (MacKenzie *et al.*, 1966). This suggests the occurrence of other overwintering hosts as yet unidentified.

There has been a greatly increased awareness of the virus disease problems of maize since dwarf mosaic was first reported. These studies have provided evidence of continuing changes in the prevalence and distribution of the various strains of sugar cane mosaic affecting the crop. For example, losses have increased in New York State since the first outbreaks in 1967 and infection was first seen in Minnesota during 1976. "A" strains that infect Johnson grass continue to predominate, although other ("B") strains are becoming increasingly prevalent. They were recorded from nine states in 1975 and from six more in 1976. Moreover, "B" strains occurred widely in Ohio during 1970, but they were not detected in a comprehensive survey of the state in 1968, when dwarf mosaic was still mainly restricted to the southern counties, where Johnson grass occurs

(Knoke, *et al.*, 1974). "B" strains were more widely distributed than ever before in 1976 when major epidemics developed throughout the state.

B. TOMATO MOSAIC VIRUS

The rapidity with which the predominant strain of virus can change in response to new selection pressures is apparent from studies on tomato mosaic virus in England. This virus causes serious losses to glasshouse crops and it is difficult to devise adequate methods of soil sterilization and cultural practices that prevent spread by contact or by handling (Broadbent, 1976). In such circumstances there are obvious advantages in introducing resistant genotypes, but progress has been slow and some forms of resistance were found to be associated with undesirable commercial characters. These problems have now been overcome and varieties are being introduced incorporating one or more genes derived originally from wild species. Particular attention has been given to *Tm-1,* which confers a form of tolerance to infection that is associated with low rates of virus multiplication. There are also *Tm-2* and its allelic form $Tm\text{-}2^2$ which lead to hypersensitivity (Pelham, 1966, 1972).

The new varieties were first grown in England during 1966, when there were extensive plantings of the Dutch "Eurocross" and "Virocross", incorporating *Tm-1.* These introductions were of great potential value because of their effectiveness against virus strain "0", which was the only one previously encountered in English glasshouses. However, the varieties were vulnerable to strain "1", which soon occurred at many sites. The proportion of strain "1" isolates in surveys during 1968 was greatest where tolerants had been grown for three successive years and declined immediately when growers reverted to other varieties (Table IV). It was suggested that strain "1" competes unsuccessfully with strain "0", except in *Tm-1* varieties which appear to select aggressive mutants that multiply more readily than strain "0" (Pelham *et al.*, 1970).

An additional factor influencing the relative importance of the different strains of tomato mosaic has been the deliberate introduction of an avirulent mutant. This was used by some growers in northern England in 1972 and more extensively in 1973 and 1974 when growers inoculated seedling plants in attempts to protect them from the effects of virulent strains (Fletcher and Rowe, 1975). The mutant was derived from strain "1" and surveys in 1974 revealed virulent forms of this strain in almost all the glasshouses where the mutant had been used. Strain "0" predominated in other houses and in a survey done

in 1971, immediately before mild strain protection was introduced (Fletcher and Butler, 1975).

The performance of "Eurocross" and "Virocross" and the problems experienced on using mild strain protection have been disappointing. However, there are obvious hazards in relying on these means of control, which are likely to fail whenever novel strains emerge. The risks could have been decreased by releasing an avirulent strain derived from strain "0". It has also been suggested that the use of *Tm-1* should have been delayed until the hypersensitive genes *Tm-2* and *Tm-2²* had also been incorporated into commercially acceptable varieties to confer resistance against additional virus strains (Pelham *et al.*, 1970). Such use of multiple genes gives the best opportunity of providing durable forms of resistance of the type now being sought in current work on many other crops (Day, 1977).

TABLE IV The relative frequency of tomato mosaic virus strain 1 in samples collected from English glasshouses in 1968 (from Pelham *et al.*, 1970)

Crop sequence at sites sampled[1]			Total sites sampled	Relative frequency of strain 1 (%)
1966	1967	1968		
S	S	S	26	9
T	S	S	5	0
S	T	S	28	26
S	S	T	7	43
T	T	S	17	47
S	T	T	14	57
T	T	T	18	93

[1] S = Varieties sensitive to strains 0, 1, 2 and 1·2
T = Varieties tolerating strains 0 and 2 but not 1 or 1·2.

Increasing attention is now being given to the possibility of decreasing the losses due to a wide range of viruses by breeding for some form of tolerance or resistance. There are also possibilities of using avirulent strains to avoid the worst effects of viruses such as citrus tristeza (Wallace and Drake, 1972). The experience gained with tomato mosaic emphasizes the problems likely to be encountered. It is important to develop carefully integrated strategies of control based on making the fullest use of complementary sources of resistance and any other measures available.

IX. Discussion

The approach adopted in this paper of categorizing viruses according to the immediate factors influencing their prevalence in crops is

somewhat simplistic and misleading. Diseases are unlikely to cause severe losses unless susceptible hosts encounter virulent strains in circumstances and seasons permitting many plants to be infected and severely damaged. Inevitably, weather, cultural practices and genetic features of pathogen and host are involved in all major epidemics and cannot be considered in isolation. Nevertheless, there are advantages in stressing the overriding importance of particular factors, whether these are climatic, biotic or associated with crop husbandry. This emphasizes the diverse behaviour of viruses, which can become prevalent for very different reasons.

It is clear even from the limited number of examples already considered that the effects of weather on viruses, vectors or their host plants are extremely complex. They contribute to the instability of the overall disease situation and account for some of the big differences in rates of spread observed between regions and between or within seasons. Apart from the differences due to long- or short-term fluctuations in weather conditions there are also those due to the activities of man and these warrant further discussion.

A. PLANT INTRODUCTIONS

The extent to which the main agricultural regions of the world are dependent on introduced crops is not always fully appreciated. Crops derived from indigenous species account for an estimated 24% of the total cultivated area in northern Europe. The proportion is even less in several other regions and the rural economies of Australasia and North America are based almost entirely on introduced species (Fig. 16).

This is an important outcome of the extensive movement of seeds, plants and plant material that has been going on for millennia along the trading routes of the world (Grigg, 1974; Purseglove, 1963). The scale of the traffic increased greatly during the 19th century due to improved methods of transport and to the activities of plant collectors, colonialists and settlers exploiting new areas for agricultural development. In recent years even greater changes have followed the introduction of air transport, with the rapid movement of large commercial consignments and continuing exchange between agriculturalists and plant breeders, including many involved in the various International Aid programmes.

The profound implications of these developments are emphasized by experience with many of the viruses and crops already considered. They illustrate the various ways in which major problems can occur

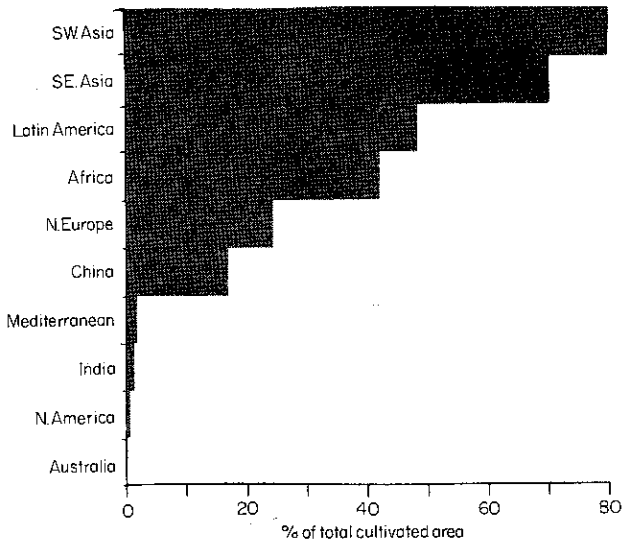

FIG. 16. Estimates of the percentage of the total cultivated area planted with indigenous crops in each of the main agricultural regions of the world (from Grigg, 1974).

when crops are first grown in new areas or when viruses are introduced or reintroduced to crops already established. Some of these problems could not have been foreseen, but others might have been avoided by adequate inspection or quarantine procedures. However, some of the most recent consignments are the only ones to have been subject to the close scrutiny of scientists and there are particular difficulties in preventing the dissemination of viruses in plant material. In these circumstances and considering the enormous traffic that has occurred over such long periods it might be expected that all viruses would have spread already to all the areas in which they can become established. This has probably occurred with many seed-borne viruses and lettuce mosaic (9), tomato mosaic, barley stripe mosaic (68) and several viruses of legumes now seem to be cosmopolitan. Viruses of important vegetatively-propagated crops have also been widely distributed, as illustrated by experience with citrus tristeza (p. 17), grapevine fanleaf (p. 29), potato virus Y (p. 25), sugar cane mosaic (88) and numerous viruses of ornamentals. Many other important viruses still have a relatively restricted distribution and there is abundant evidence of the effectiveness of oceans and other natural barriers in restricting spread. This justifies stringent controls on any further movement of plant material, despite

the delays, inconvenience and expense involved. For example, it is important to prevent plum pox spreading outside Europe (p. 14) and cacao swollen shoot is still largely restricted to West Africa (p. 19). Sugar cane Fiji (p. 43), maize rough dwarf (p. 38) and rice tungro (p. 40) are additional viruses that could become much more widespread than at present. There are many other instances and much can be achieved if a responsible attitude is adopted towards further introductions, taking full advantage of the knowledge and experience now available (Hewitt and Chiarappa, 1977). The various International Organizations and commercial firms have a particular role to play in setting and maintaining exacting health standards of the type now enforced by the Peruvian International Centre for Potato Research when distributing breeding stocks around the world.

B. PLANT BREEDING

Plant breeders have long been involved in the exchange of plant material and they also have an important and increasing impact on the prevalence of many viruses by influencing the ways in which crops are grown and their vulnerability to infection. The full magnitude and implications of these effects are not always recognized, despite the many occasions on which viruses have first been reported or become damaging following the introduction of new varieties (p. 36).

One of the main developments with many crops has been the production of versatile varieties that can be grown under a wide range of conditions because they are less sensitive than traditional ones to cold, drought or photoperiod. This has permitted expansion into new areas, extended growing seasons and facilitated multiple or relay cropping systems. The benefits have been enormous, with great increases in the total yield and productivity of many crops. However, new pests and diseases have been encountered and long-established ones have become increasingly prevalent as growing seasons have been prolonged. Additional difficulties have arisen following the introduction and widespread use of inadequately tested varieties that soon succumbed to disease.

These points are well-illustrated from past experience with viruses of maize (p. 38), rice (p. 40) and some of the other crops already considered. Further problems are likely to occur following increased plantings of cold-hardy varieties of crops such as onion that can be sown before winter to permit early establishment and extend the growing season. Moreover, the use of male-sterile lines to facilitate the production of hybrid wheat or barley varieties (Done, 1973) could

facilitate the spread of stripe mosaic virus. This is seed- and pollen-borne, although there is at present little opportunity for spread between plants by means of pollen because current varieties are almost entirely self-pollinated.

There are obvious advantages in close collaboration between breeders and virologists to exploit sources of resistance or tolerance and to avoid releasing varieties that are particularly vulnerable. Experience with sugar beet curly top (p. 23) and wheat soil-borne mosaic (p. 28) shows what can be achieved and this approach could be successful in decreasing losses due to other viruses. For example, work is now in progress to develop virus-resistant types of navy bean (*Phaseolus vulgaris*) for use in England from cold-tolerant accessions that are unsuitable because of their susceptibility to bean common mosaic virus (Walkey and Innes, 1979).

Additional studies are justified to gain a better understanding of the various mechanisms of resistance and tolerance. It is also important to ensure that healthy stocks of seed or vegetative material are available when new varieties are released. The advantages are apparent from surveys of barley stripe mosaic in North Dakota, where the virus was widespread and caused losses estimated at more than 2 million bushels of grain per year until improved varieties were introduced. These were multiplied rapidly from virus-free stocks and infection was virtually eliminated within a few years (Timian, 1971).

Difficulties have been encountered in obtaining virus-free clones of many vegetatively-propagated crops because there are ample opportunities for potential new varieties to become infected when grown alongside or grafted onto existing ones in preliminary screening tests or long-term trials. Nevertheless, effective methods of selection or therapy have been developed for use with potato, hop, strawberry and many other fruit crops and ornamentals (Hollings, 1965). This enables growers to decrease the virus problems likely to arise whenever vegetative propagation is used to exploit particular genotypes or to facilitate establishment, uniformity and early cropping.

C. CROPPING PRACTICES

There are numerous examples in this paper and elsewhere in the literature of the ways in which the incidence of viruses or the losses they cause are related to methods of husbandry and cropping practices. Apparently minor and unimportant changes in such features as sowing date or in the sequence, spacing, culture and siting of

crops can have disproportionately large effects on the prevalence of disease. The consequences of extending the natural growing season by introducing new varieties, irrigation or some form of protected cropping are even greater.

Many different factors are involved as almost any change in the way in which crops are grown influences the susceptibility or response of plants to infection or the ease and rapidity with which spread occurs into and within plantings. Horticulturalists and agriculturalists are frequently unaware of the existence or magnitude of these effects. This complicates the interpretation of field trials to evaluate new methods of crop production. There is an obvious need for pathologists to monitor plantings and assess the impact of diseases on the yields obtained. Innovations of great potential benefit could be entirely overlooked if any requirement for improved methods of control is not recognized at an early stage.

Changes in cropping practices continue and an important development in recent years has been the use made by major international horticultural enterprises of outdoor sites in Africa or other regions with mild, sunny winters (Ten Houten, 1974). Cuttings are raised of carnation, chrysanthemum and other ornamentals for use in glasshouses at more extreme latitudes. The movement of plants to and from these regions greatly complicates pest and disease problems. There are likely to be increased losses due to insect-borne viruses such as carnation vein mottle, that are currently much more prevalent in outdoor crops than in those grown exclusively in glasshouses (Hollings *et al.*, 1977; Hollings and Stone, 1979).

Striking differences in the prevalence of viruses in crops raised in different ways are already apparent from surveys of capsicum peppers in the Piedmont area of north west Italy (Conti and Masenga, 1977). Tobacco mosaic (151) was the only virus to become important in crops planted early in polythene tunnels, where spread by contact was facilitated by the frequent handling required and the tendency to use the same sites for successive plantings. By contrast, cucumber mosaic (1) and other viruses transmitted non-persistently by aphids predominated in outdoor stands. Much of the initial spread was into the margins of crops from adjacent foci of infection and occurred most readily when plantings were small, with a large proportion of the plants in the vulnerable peripheral areas. Consequently, large fields were less severely affected than small garden plantings where multiple infection was relatively common.

Several other viruses spread similarly and their incidence is also decreased by making plantings fewer and larger (Watson, 1967). This

is an important beneficial consequence of the current trend towards increased field size. Conversely, spread is facilitated by the increased use of sequential plantings of vegetables and other crops to extend the harvesting season and ensure a continuous supply of produce for retail outlets, freezing or processing.

Other recent developments in the horticultural industry have had further important and contrasting effects on the incidence and spread of several diseases. An important trend has been for cultural systems to become increasingly specialized and individual glasshouses are seldom used for more than one type of crop. This decreases the opportunity for aphids to spread aspermy virus between chrysanthemum and tomato. By contrast, two recent innovations have contributed to the increased prevalence of lettuce big-vein disease in England (Hill, 1978b). Many plants are now raised at large central nurseries by the system of close spacing in soil blocks, for eventual distribution to cropping sites throughout the country. Spread by the fungus vector is also facilitated by the increased use of the nutrient film technique of growing plants in dense stands fed by continuously circulating solutions (Tomlinson and Faithfull, 1980).

Such problems have not been encountered by glasshouse growers adopting peat or peat sand-substrates that are replaced after each crop. This has decreased the incidence of tomato mosaic virus, which previously persisted in soil that was difficult to sterilize satisfactorily between plantings (Broadbent, 1976). These examples emphasize the rapidity with which the incidence and prevalence of virus diseases can change in response to new innovations.

D. FUTURE PROSPECTS

Bawden (1955) considered some of the ways in which modern agricultural practices favour the spread of pests and diseases and concluded that the full benefits of improved technology would only be attained by developing more effective methods of control. The full significance of this comment has become increasingly apparent in recent years from experience with diverse crops in various parts of the world. Losses continue from many well-known diseases or they have become increasingly difficult and expensive to contain. New problems have been encountered and minor ones have increased in importance to such an extent as to impede progress and decrease the benefits of modern varieties and other advances in crop production.

Pathologists are presented with a formidable challenge and a powerful stimulus to epidemiological studies. Fungal diseases are

already receiving much attention from pathologists, breeders, population geneticists, physicists, statisticians and others working on many different aspects of spread and control. There has also been considerable discussion as to the underlying causes of recent major epidemics and the implications of current trends in plant breeding and crop husbandry (e.g. Day, 1977). Virologists have made only limited contributions to the debate as the main emphasis in present research is on aetiological studies and on the basic structure, properties and grouping of viruses. Virus disease epidemiology has been comparatively neglected and the multi-disciplinary teams required for comprehensive studies on the spread of vector-borne viruses have seldom been established and maintained for long.

There is an urgent need for additional studies and new approaches if losses are to be decreased by supplementing the existing largely inadequate methods of control. It is particularly important for pathologists to work in closer collaboration than hitherto with plant breeders, agriculturalists and horticulturalists to attain the maximum benefits of any further innovations without increasing the losses due to viruses or other pathogens. The need for broad-based studies along ecological lines is obvious from experience with many of the viruses discussed in this paper. These provide abundant evidence of the complex situation with many crops and of the continuing changes in the prevalence of disease due to the impact of weather factors, new varieties and cropping practices.

References

Adams, A. N. (1967). The vectors and alternative hosts of groundnut rosette virus in Central Province, Malawi. *Rhod. Zamb. Mal. J. agric. Res.* **5,** 145–152.

Adams, A. N. (1978). The incidence of plum pox virus in England and its control in orchards. *In* "Plant Disease Epidemiology" (Scott, P. R. and Bainbridge, A., eds), p. 213–219. Blackwell Scientific Publications, Oxford.

Anonymous (1976). Phyto-insanitary Britain. *Nature, Lond.* **259,** 449.

Atkinson, T. G. and Slykhuis, J. T. (1963). Relation of spring drought, summer rain and high fall temperatures to the wheat streak mosaic ephiphytotic in Southern Alberta, 1963. *Can. Pl. Dis. Surv.* **43,** 154–159.

Attafuah, A. and Tinsley, T. W. (1958). Virus disease of *Adansonia digitata* L. (Bombacaceae) and their relation to cacao in Ghana. *Ann. appl. Biol.* **46,** 20–22.

Bakker, W. (1970). Rice yellow mottle, a mechanically transmissible virus disease of rice in Kenya. *Neth. J. Pl. Path.* **76,** 53–63.

Bakker, W. (1974). Characterization and ecological aspects of rice yellow mottle virus in Kenya. *Agric. Res. Reptr.* 829.

Bar-Joseph, M. (1978). Cross protection incompleteness: a possible cause for natural spread of citrus tristeza virus after a prolonged lag period in Israel. *Phytopathology* **68,** 1110–1111.

Bawden, F. C. (1955). The spread and control of plant virus diseases. *Ann. appl. Biol.* **42,** 140–147.

Beijerinck, M. W. (1898). Over een contagium vivum fluidum als oorzaak van de vlekziekte der tabaksbladen. *Verhandel. Koninkl. Akad. Wetenschaf., Afdel. Wis – Natuurk* **7,** 229–235, *Phytopathological Classic*, **7.**

Bennett, C. W. (1952). Origin and distribution of new or little-known virus diseases. *Pl. Dis. Reptr. Suppl.* **211,** 43–46.

Bennett, C. W. (1967). Epidemiology of leafhopper-transmitted viruses. *A. Rev. Phytopath.* **5,** 87–108.

Bennett, C. W. (1971). The curly top disease of sugar beet and other plants. Monograph 7, American Phytopathological Society.

Bennett, C. W. and Costa, A. S. (1949). Tristeza disease of citrus. *J. agric. Res.* **78,** 207–237.

Bennett, C. W. and Tanrisever, A. (1957). Sugar beet curly top disease in Turkey. *Pl. Dis. Reptr.* **41,** 721–725.

Bock, K. R. (1966). Arabis mosaic and *Prunus* necrotic ringspot viruses in hop (*Humulus lupulus* L.). *Ann. appl. Biol.* **57,** 131–140.

Bos, L. (1978). "Symptoms of Virus Diseases in Plants". Centre for Agricultural Publishing and Documentation, Wageningen.

Boughey, A. S. (1947). "The Causes of Variations in the Incidence of Cotton Leaf Curl in the Sudan Gezira." Mycological Paper 22, Imperial Mycological Institute, Kew.

Bourke, P. M. A. (1964). Emergence of potato blight, 1843–1846. *Nature, Lond.* **203,** 805–808.

Bowen, R. and Plumb, R. T. (1979). The occurrence and effects of red clover necrotic mosaic virus in red clover (*Trifolium pratense*). *Ann. appl. Biol.* **91,** 227–236.

Broadbent, L. (1976). Epidemiology and control of tomato mosaic virus. *A. Rev. Phytopath.* **14,** 75–96.

Broadbent, L., Cornford, C. E., Hull, R. and Tinsley, T. W. (1949). Overwintering of aphids especially *Myzus persicae* Sulzer, in root clamps. *Ann. appl. Biol.* **36,** 513–524.

Bruehl, G. W. (1961). Barley yellow dwarf, a virus of cereals and grasses. Monograph 1, American Phytopathological Society.

Buddenhagen, I. W. (1977). Resistance and vulnerability of tropical crops in relation to their evolution and breeding. *Ann. N.Y. Acad. Sci.* **287,** 309–326.

Cadman, C. H., Dias, H. F. and Harrison, B. D. (1960). Sap-transmissible viruses associated with diseases of grapevines in Europe and North America. *Nature, Lond.* **187,** 577–579.

Campbell, R. N. (1965). Weeds as reservoir hosts of the lettuce big-vein virus. *Can J. Bot.* **43,** 1141–1149.

Carter, W. (1930). Ecological studies of the beet leafhopper. *Tech. Bull. U.S. Dep. Agric.* **206.**

Cherrett, J. M. and Sagar, G. R. (1977). "Origins of Pest, Parasite Disease and Weed Problems". Blackwell Scientific Publications, Oxford.

Conti, M. (1972). Investigations on the epidemiology of maize rough dwarf virus 1. Overwintering of virus in its plant-hopper vector. *Actas III Congr. Un. fitopat Medit. Oeiras, Portugal 22–28 Outubro* **1972,** 11–17.

Conti, M. and Masenga, V. (1977). Identification and prevalence of pepper viruses in Northwest Italy. *Phytopath. Z.* **90,** 212–222.

Coons, G. H. (1949). The sugar beet: product of science. *Scient. Mon., N.Y.* **68,** 149–164.

Coons, G. H. (1953). Disease resistance breeding of sugar beets, 1918–1952. *Phytopathology* **43,** 297–303.

Cropley, R. (1967). Decline and death of pear on quince rootstocks caused by virus infection. *J. hort. Sci.* **42,** 113–115.

Dale, W. T. (1962). Diseases and pests of cocoa. A. Virus diseases. *In* "Agriculture and Land Use in Ghana" (J. B. Wills, ed.), pp. 286–316. Oxford University Press, London.

Day, P. R. (1977). The genetic basis of epidemics in agriculture. *Ann. N.Y. Acad. Sci.* **287,** 1–400.

Dias, H. F. (1977). Incidence and geographic distribution of tomato ringspot virus in De Chaunac vineyards in the Niagara Peninsula. *Pl. Dis Reptr.* **61,** 24–28.

Doncaster, J. P. and Gregory, P. H. (1948). The spread of virus diseases in the potato crop. A.R.C. Rep. Ser. No. 7. Stationery Office, London.

Done, A. C. (1973). Implications of the introduction of hybrid cereals on disease patterns. *Ann. appl. Biol.* **75,** 144–149.

Dorst, H. J. M. van, Hoof, H. A. van. (1965). *Arabis*-mozaiekvirus bij komkommer in Nederland. *Neth. J. Pl. Path.* **71,** 176–179.

DuCharme, E. P., Knorr, L. C. and Speroni, H. A. (1951). Observations on the spread of tristeza in Argentina. *Citrus Mag.* **13,** 10–14.

Duffus, J. E. (1977). Aphids, viruses and the yellows plague. *In* "Aphids as Virus Vectors" (Harris, K. F. and Maramorosch, K., eds), pp. 361–383. Academic Press, New York and London.

Dunn, J. A. and Kirkley, J. (1966). Studies on the aphid *Cavariella aegopodii* Scop. II. On secondary hosts other than carrot. *Ann. appl. Biol.* **58,** 213–217.

Egan, B. T. (1976). The fall and rise of Fiji disease in Southern Queensland. *Proc. 43rd Conf. Qd. Soc. Sug. Cane Technol.* 73–77.

Egan, B. T. and Fraser, T. T. (1977). The development of the Fiji disease epidemic at Bundaberg. *Proc. 44th Conf. Qd. Soc. Sug. Cane Technol.* 43–48.

Ellett, C. W., Janson, B. F. and Williams, L. E. (1965). Maize dwarf mosaic in Ohio in 1964. *Pl. Dis. Reptr.* **49,** 615–616.

Estes, A. P. and Brakke, M. K. (1966). Correlation of *Polymyxa graminis* with transmission of soil-borne wheat mosaic virus. *Virology,* **28,** 772–774.

Evans, A. C. (1954). Groundnut rosette disease in Tanganyika I. Field Studies. *Ann. appl. Biol.* **41,** 189–206.

Everett, T. R. and Lamey, H. A. (1969). Hoja blanca. *In* "Viruses, Vectors and Vegetation" (Maramorosch, K., ed.), pp. 361–377. John Wiley and Sons, New York and London.

Fawcett, H. S. and Wallace, J. M. (1946). Evidence of the virus nature of quick decline. *Calif. Citorg.* **32,** 88–89.

Fletcher, J. T. and Butler, D. (1975). Strain changes in populations of tobacco mosaic virus from tomato crops. *Ann. appl. Biol.* **81,** 409–412.

Fletcher, J. T. and Rowe, J. M. (1975). Observations and experiments on the use of an avirulent mutant strain of tobacco mosaic as a means of controlling tomato mosaic. *Ann. appl. Biol.* **81,** 171–179.

Francki, R. I. B. and Crowley, N. C. (1967). Investigations of suspected grapevine viruses in South Australia. *Aust. J. agric. Res.* **18,** 461–466.

Frazier, N. W., Yarwood, C. E. and Gold, A. H. (1961). Yellow bud virus endemic along California coast. *Pl. Dis. Reptr.* **45,** 649–651.

Fulton, R. A. and Romney, V. E. (1940). The chloroform-soluble components of beet leafhoppers as an indication of the distance they move in the spring. *J. agric. Res.*

61, 737–743.

Gardner, F. E., Marth, P. C. and Magness, J. R. (1946). Lethal effects of certain apple scions on Spy 227 stock. *Proc. Am. hort. Soc.* **48,** 195–199.

Gibbs, A. and Harrison, B. D. (1976). "Plant Virology: The Principles". Edward Arnold, London.

Gibson, K. E. (1971). The incidence of curly-top virus on sugarbeets in Iran—1966 to 1969. *Pl. Dis. Reptr.* **55,** 85–86.

Grancini, P. (1962). Ulteriori notizie sul nanismo ruvido del mais. *Maydica* **14,** 17–25.

Grigg, D. B. (1974). "The Agricultural Systems of the World: An Evolutionary Approach". Cambridge University Press, Cambridge.

Grylls, N. E. and Butler, F. C. (1959). Subterranean clover stunt, a virus disease of pasture legumes. *Aust. J. agric. Res.* **10,** 145–158.

Gutierrez, A. P., Morgan, D. J. and Havenstein, D. E. 1971). The ecology of *Aphis craccivora* Koch and subterranean clover stunt virus. I. The phenology of aphid populations and the epidemiology of virus in pastures in south east Australia. *J. Appl. Ecol.* **8,** 699–721.

Gutierrez, A. P., Havenstein, D. E., Nix, H. A. and Moore, P. A. (1974a). II. A model of cowpea aphid populations in temperate pastures. *J. Appl. Ecol.* **11,** 1–20.

Gutierrez, A. P., Nix, H. A., Havenstein, D. E. and Moore, P. A. (1974b). III. A regional perspective of the phenology and migration of the cowpea aphid. *J. Appl. Ecol.* **11,** 21–35.

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

Harrison, B. D. (1977). Ecology and control of viruses with soil-inhabiting vectors. *A. Rev. Phytopath.* **15,** 331–360.

Harrison, B. D. (1978). The groups of nematode-transmitted plant viruses, and molecular aspects of their variation and ecology. *In* "Plant Disease Epidemiology" (Scott, P. R. and Bainbridge, A., eds), p. 255–264. Blackwell Scientific Publications, Oxford.

Harrison, B. D. and Winslow, R. D. (1961). Laboratory and field studies on the relation of arabis mosaic virus to its nematode vector *Xiphinema diversicaudatum* (Micoletzky). *Ann. appl. Biol.* **49,** 621–633.

Hatton, R. G. (1928). The behaviour of certain pears on various quince rootstocks. *J. Pomol.* **7,** 216–233.

Hewitt, W. B. (1968). Viruses and virus diseases of the grapevine. *Rev. appl. Mycol.* **47,** 433–455.

Hewitt, W. B. and Chiarappa, L. (1977). "Plant Health and Quarantine in International Transfer of Genetic Resources". CRC Press Inc., Cleveland, Ohio.

Hewitt, W. B., Raski, D. J. and Goheen, A. C. (1958). Nematode vector of soil-borne fanleaf virus of grapevine. *Phytopathology,* **48,** 586–595.

Hewitt, W. B., Goheen, A. C., Raski, D. J. and Gooding, G. V. (1962). Studies on virus diseases of the grapevine in California. *Vitis* **3,** 57–83.

Hill, S. A. (1978a). Current virus infection of seed potatoes in Britain. *In* "Plant Disease Epidemiology" (Scott, P. R. and Bainbridge, A., eds), pp. 229–234. Blackwell Scientific Publications, Oxford.

Hill, S. A. (1978b). Lettuce big vein virus. *Newsletter of the Federation of British Plant Pathologists* **1,** 25–27.

Hodson, A. C. and Cook, E. F. (1960). Long-range aerial transport of the Harlequin bug and the greenbug into Minnesota. *J. econ. Ent.* **53,** 604–608.

Hollings, M. (1965). Disease control through virus-free stock. *A. Rev. Phytopath.* **3,**

367–396.
Hollings, M. and Stone, O. W. (1979). Production and use of virus-free stocks of ornamental and bulb crops; some phytosanitary and epidemiological aspects. *In* "Plant Health: the Scientific Basis for Administrative Control of Plant Parasites" (Ebbels, D. L. and King, J. E., eds). Blackwell Scientific Publications, Oxford.
Hollings, M., Stone, O. M., Atkey, P. T. and Barton, R. J. (1977). Investigations of carnation viruses IV. Carnation vein mottle virus. *Ann. appl. Biol.* **85,** 59–70.
Holmes, F. O. (1965). Genetics of pathogenicity in viruses and of resistance in host plants. *Adv. Virus Res.* **11,** 139–161.
Howell, W. E. and Mink, G. I. (1971a). The relationship between volunteer sugar beets and occurrence of beet mosaic and beet western yellows viruses in Washington beet fields. *Pl. Dis. Reptr.* **55,** 676–678.
Howell, W. E. and Mink, G. I. (1971b). The relationship between agricultural practices and the occurrence of volunteer sugarbeets in Washington. *J. Am. Soc. Sug. Beet Technol.* **16,** 441–447.
Howell, W. E. and Mink, G. I. (1977). The role of weed hosts, volunteer carrots and overlapping growing seasons in the epidemiology of carrot thin leaf and carrot motley dwarf viruses in Central Washington. *Pl. Dis. Reptr.* **61,** 217–222.
Hull, R. (1952). Control of virus yellows in sugar beet seed crops. *Jl. R. agric. Soc.* **113,** 86–102.
Hull, R. (1953). Assessments of losses in sugar beet due to virus yellows in Great Britain, 1942–1952. *Pl. Path.* **2,** 39–43.
Hurst, G. W. (1965). Forecasting the severity of sugar beet yellows. *Pl. Path.* **14,** 47–53.
Janson, B. F. and Ellett, C. W. (1963). A new corn disease in Ohio. *Pl. Dis. Reptr.* **47,** 1107–1108.
John, V. T. and Prasada Rao, R. D. V. J. (1971). Factors favouring the incidence of tungro and methods of control. *Oryza* **8,** 365–368.
Johnson, B. (1957). Studies on the dispersal by upper winds of *Aphis craccivora* Koch in New South Wales. *Proc. Linn. Soc. N.S.W.* **82,** 191–198.
Johnson, B. (1965). Wing polymorphism in aphids. II. Interaction between aphids. *Entomologia exp. appl.* **8,** 49–64.
Jordović, M. M. (1975). Study of sharka spread pattern in some plum orchards. *Acta Hort.* **44,** 147–154.
Keyworth, W. G. (1947). Mosaic disease of the hop: a study of tolerant and sensitive varieties. *Rep. E. Malling Res. Stn.* **1946,** 142–148.
Khush, G. S. (1977). Breeding for resistance in rice. *Ann. N.Y. Acad. Sci.* **287,** 296–308.
King, C. L. and Sill, W. H. (1959). 1959 wheat streak mosaic epiphytotic in Kansas. *Pl. Dis. Reptr.* **43,** 1256.
Klein, M. and Harpaz, I. (1970). Heat suppression of plant-virus propagation in the insect vector's body. *Virology,* **41,** 72–76.
Klinkowski, M. (1964). Die ausbreitung von viruskrankheiten unserer nutzpflanzen im europäischen masstab. *Sber. dr. Akad. LandWiss.* **13,** 5–25.
Klinkowski, M. (1970). Catastrophic plant diseases. *Ann. Rev. Phytopath.* **8,** 37–60.
Knoke, J. K., Louie, R., Anderson, R. J. and Gordon, D. T. (1974). Distribution of maize dwarf mosaic and aphid vectors in Ohio. *Phytopathology* **64,** 639–645.
Koehler, B., Bever, W. M. and Bonnett, O. T. (1952). Soil-borne wheat mosaic. *Bull. 556 Ill. agric. Exp. Stn.* pp. 566–599.
Kulshreshtha, J. P., Kalode, M. B., Prakasa Rao, P. S., Misra, B. C. and Varma, A.

(1970). High yielding varieties and the resulting changes in the pattern of rice pests in India. *Oryza* **7,** 61–64.

Lamberti, F. and Martelli, G. P. (1965). On the distribution of *Xiphinema index* Thorne et Allen in some Apulian vineyards. Proceedings of the International Conference on Virus and Vector on Perennial Hosts with Special Reference to *Vitis,* Davis, 1965, 353–363.

Legg, J. T. (1979). The campaign to control the spread of cocoa swollen shoot virus in Ghana. *In* "Plant Health: the Scientific Basis for Administrative Control of Plant Parasites" (Ebbels, D. L. and King, J. E., eds). Blackwell Scientific Publications, Oxford.

Lim, G. S. (1972). Studies on penyakit merah disease of rice. III. Factors contributing to an epidemic in North Krian, Malaysia. *Malay. agric. J.* **48,** 278–294.

Luckwill, L. C. and Crowdy, S. H. (1950). Virus diseases of fruit trees. II. Observations on rubbery wood, chat fruit and mosaic in apples. Progress report. *Rep. agric. hort. Res. Stn Univ. Bristol* **1949,** 68–79.

Lumkes, L. M. (1974). Research on the control of volunteer potatoes in the Netherlands. *Proc. 12th Br. Weed Control Conf.* **1974,** 1031–1040.

Lutman, P. J. W. (1974). Factors affecting the overwintering of volunteer potato tubers and the emergence of sprouts in the spring. *Proc. 12th Br. Weed Control Conf.* **1974,** 285–292.

Lutman, P. J. W. (1977). Investigations into some aspects of the biology of potatoes as weeds. *Weed Research* **17,** 123–132.

MacKenzie, D. R., Wernham, C. C. and Ford, R. E. (1966). Differences in maize dwarf mosaic virus isolates of the northeastern United States. *Pl. Dis. Reptr.* **50,** 814–818.

Mackenzie, D., Salmon, E. S., Ware, W. M. and Williams, R. (1929). The mosaic disease of the hop; grafting experiments, II. *Ann. appl. Biol.* **16,** 359–381.

Martelli, G. P. (1978). Nematode-borne viruses of grapevine. *In* "Plant Disease Epidemiology" (Scott, P. R. and Bainbridge, A., eds), pp. 275–282. Blackwell Scientific Publications, Oxford.

Matthews, R. E. F. (1970). "Plant Virology". Academic Press, New York and London.

McClean, A. P. D. (1957). Virus infections in citrus trees. *Pl. Prot. Bull. F.A.O.* **5,** 139–141.

McKinney, H. H. (1925). A mosaic disease of winter wheat and winter rye. Bull 1361, U.S. Dep. Agric.

McKinney, H. H. (1937). Mosaic disease of wheat and related cereals. Circ. 442 U.S. Dep. Agric.

McNamara, D. G. and Pitcher, R. S. (1977). The long-term effects of four monocultural regimes on two field populations of the nematodes *Xiphinema diversicaudatum* and *Longidorus* spp. *Ann. appl. Biol.* **86,** 405–413.

McNamara, D. G., Ormerod, P. J., Pitcher, R. S. and Thresh, J. M. (1973). Fallowing and fumigation experiments on the control of nettlehead and related virus diseases of hop. *Proc. 7th Br. Insect. Fung. Conf.* **1973,** 597–602.

Medler, J. T. (1960). Long-range displacement of homoptera in the central United States. *Proceedings XIth Internationale Kongress für Entomologie* **3,** 30–35.

Medler, J. T. and Smith, P. W. (1960). Greenbug dispersal and distribution of barley yellow dwarf virus in Wisconsin. *J. econ. Ent.* **53,** 473–474.

Meneghini, M. (1946). Sôbre a natureza e transmissibilidade da doença 'Tristeza' dos citrus. *O Biológico* **12,** 285–287.

Moore, W. F. (1970). Origin and spread of southern corn leaf blight in 1970. *Pl. Dis. Reptr.* **54,** 1104–1108.

Morley, F. H. W. (1961). Subterranean clover. *Adv. Agron.* **13,** 57–123.

Murant, A. F., Taylor, C. E. and Chambers, J. (1968). Properties, relationships and transmission of a strain of raspberry ringspot virus infecting raspberry cultivars immune to the common Scottish strain. *Ann. appl. Biol.* **61,** 175–186.

Nahmias, A. J. and Reanney, D. C. (1977). The evolution of viruses. *Ann. Rev. Ecol. Syst.* **8,** 29–49.

Oman, P. W. (1948). Notes on the beet leafhopper *Circulifer tenellus* (Baker) and its relatives (Homoptera: Cicadellidae). *J. Kans. ent. Soc.* **21,** 10–14.

Oman, P. W. (1969). Criteria of specificity in virus-vector relationships. *In* "Viruses, Vectors and Vegetation" (Maramorosch, K., ed.), pp. 1–22. John Wiley and Sons, New York and London.

Orlob, G. B. and Medler, J. T. (1961). Biology of cereal and grass aphids in Wisconsin (Homoptera). *Can. Ent.* **93,** 703–714.

Oswald, J. W. and Houston, B. R. (1951). A new virus disease of cereals, transmissible by aphids. *Pl. Dis. Reptr.* **35,** 471–475.

Oswald, J. W. and Houston, B. R. (1953). Host range and epiphytology of the cereal yellow dwarf disease. *Phytopathology* **43,** 309–313.

Over de Linden, A. J. and Chamberlain, E. E. (1970). Production of virus-free grapevines in New Zealand. *N.Z. Jl. agric. Res.* **13,** 991–1000.

Pelham, J. (1966). Resistance in tomato to tobacco mosaic virus. *Euphytica* **15,** 258–267.

Pelham, J. (1972). Strain-genotype interaction of tobacco mosaic virus in tomato. *Ann. appl. Biol.* **71,** 219–228.

Pelham, J., Fletcher, J. T. and Hawkins, J. H. (1970). The establishment of a new strain of tobacco mosaic virus resulting from the use of resistant varieties of tomato. *Ann. appl. Biol.* **65,** 293–297.

Petherbridge, F. R. and Stirrup, H. H. (1935). Pests and diseases of sugar beet. *Bull. Minist. Agric. Fish. Fd., London* **93.**

Piemeisel, R. L., Lawson, F. R. and Carsner, E. (1951). Weeds, insects, plant diseases and dust storms. *Scient. Mon. N.Y.* **73,** 124–128.

Pitcher, R. S. and Jha, A. (1961). On the distribution and infectivity with arabis mosaic virus of a dagger nematode. *Pl. Path.* **10,** 67–71.

Pontis, R. E. and Feldman, J. M. (1963). A common weed, *Physalis viscosa,* new host for potato virus Y. *Pl. Dis. Reptr.* **47,** 22.

Posnette, A. F. (1947). Virus diseases of cacao in West Africa. I. Cacao viruses 1A, 1B, 1C and 1D. *Ann. appl. Biol.* **34,** 388–402.

Posnette, A. F. (1977). Virus diseases of woody plants. *Scient. Hort.* **29,** 7–13.

Posnette, A. F. and Todd, J. McA. (1951). Virus diseases of cacao in West Africa. VIII. The search for virus-resistant cacao. *Ann. appl. Biol.* **38,** 785–800.

Posnette, A. F., Robertson, N. F. and Todd, J. McA. (1950). Virus diseases of cacao in West Africa. V. Alternative host plants. *Ann. appl. Biol.* **37,** 229–240.

Purseglove, J. W. (1963). Some problems of the origin and distribution of tropical crops. *Genet. agr.* **17,** 105–122.

Purseglove, J. W. (1965). The spread of tropical crops. *In* "Genetics of Colonizing Species" (Baker, H. G. and Stebbins, G. L., eds), pp. 375–386. Academic Press, New York and London.

Quanjer, H. M. (1934). Enkele kenmerken der 'vergelings'-ziekte van suiker-en voederbieten ter onderscheiding van de zwarte heutvatenziekte. *Tijdschr. PlZiekt.* **40,**

201–214.
Ramsdell, D. C. and Myers, R. L. (1974). Peach rosette mosaic virus, symptomatology and nematodes associated with grapevine degeneration in Michigan. *Phytopathology* **64,** 1174–1178.
Raski, D. J. and Schmitt, R. V. (1972). Progress in control of nematodes by soil fumigation in nematode-fanleaf infected vineyards. *Pl. Dis. Reptr.* **56,** 1031–1035.
Raski, D. J., Hewitt, W. B., Roheen, A. C., Taylor, C. E. and Taylor, R. H. (1965). Survival of *Xiphinema index* and reservoirs of fanleaf virus in fallowed vineyard soil. *Nematologica* **11,** 349–352.
Rivera, C. T. and Ou, S. H. (1965). Leafhopper transmission of 'tungro' disease of rice. *Pl. Dis. Reptr.* **49,** 127–131.
Rochow, W. F. (1969). Biological properties of four isolates of barley yellow dwarf virus. *Phytopathology* **59,** 1580–1589.
Rochow, W. F. (1979). Field variants of barley yellow dwarf virus: detection and fluctuation during twenty years. *Phytopathology* **69,** 655–660.
Rochow, W. F. and Jedlinski, H. (1970). Variants of barley yellow dwarf virus collected in New York and Illinois. *Phytopathology* **60,** 1030–1035.
Roland, G. (1936). Researches sut la juanisse de la betterave et quelques observations sur la mosaique de cette plant. *Sucr. Belge.* **55,** 213–217.
Rose, D. J. W. (1978). Epidemiology of maize streak disease. *A. Rev. Ent.* **23,** 259–282.
Rush, M. C. (1970). Transmission of tobacco ringspot virus from native hosts to *Cucumus sativus* by *Xiphinema americanum. Phytopathology* **60,** 917–918.
Russell, G. E. (1958). Sugar beet yellows: a preliminary study of the distribution and inter-relationships of viruses and virus strains found in East Anglia, 1955–1957. *Ann. appl. Biol.* **46,** 393–398.
Russell, G. E. (1963). Some factors affecting the relative incidence, distribution and importance of beet yellows virus and sugar beet mild yellowing virus in eastern England, 1955–1962. *Ann. appl. Biol.* **52,** 405–413.
Sakimura, K. (1953). Potato virus Y in Hawaii. *Phytopathology* **43,** 217–218.
Salmon, E. S. (1923). The 'mosaic' disease of the hop. *J. Minist. Agric. Fish.* **29,** 1–7.
Scot, R. (1574). "A Perfite Platforme of a Hoppe Garden". H. Denham, London.
Shepherd, R. J. (1965). Properties of a mosaic virus of corn and Johnson grass and its relation to the sugarcane mosaic virus. *Phytopathology* **55,** 1250–1256.
Simons, J. N. (1959). Potato virus Y appears in additional areas of pepper and tomato production in south Florida. *Pl. Dis. Reptr.* **43,** 710–711.
Simons, J. N., Conover, R. A. and Walter, J. M. (1956). Correlation of occurrence of potato virus Y with areas of potato production in Florida. *Pl. Dis. Reptr.* **40,** 531–533.
Slykhuis, J. T. (1955). *Aceria tulipae* Keifer (Acarina Eriophyidae) in relation to the spread of wheat streak mosaic. *Phytopathology* **45,** 116–128.
Slykhuis, J. T. (1967). Virus diseases of cereals. *Rev. appl. Mycol.* **46,** 401–429.
Slykhuis, J. T. (1970). Factors determining the development of wheat spindle streak mosaic caused by a soil-borne virus in Ontario. *Phytopathology* **60,** 319–331.
Slykhuis, J. T. (1976). Wheat spindle streak mosaic virus. *C.M.I./A.A.B. Descriptions of plant viruses* **167.**
Slykhuis, J. T., Andrews, J. E. and Pittmann, U. J. (1957). Relation of date of seeding winter wheat in southern Alberta to losses from wheat streak mosaic, root rot and rust. *Can. J. Pl. Sci.* **37,** 113–127.
Smith, H. C. (1963). Control of barley yellow dwarf virus in cereals. *N.Z. Jl. agric. Res.*

6, 229–244.
Sōgawa, K. (1976). Rice tungro virus and its vector in tropical Asia. *Rev. Pl. Prot. Res.* **9,** 21–46.
Sparrow, L. A. D. (1976). Recent trends in the activ of aphids infesting potatoes in south-east Scotland in relation to virus incidence i he crop. Bull. etin No. 11 of the Scottish Horticultural Research Institute Associ on, pp. 8–14.
Steven, W. F. (1937). A new disease of cocoa in th Coast. *T rop. Agric. Trin.* **14,** 84.
Tarr, S. A. J. (1951). "Leaf Curl Disease of Cot omm onwealth Mycological Institute, Kew.
Taylor, C. E. and Raski, D. J. (1964). On t ission of grape fanleaf by *Xiphinema index*. *Nematologica* **10,** 489–495.
Taylor, C. E. and Thomas, P. R. (1968). The ase e n of *Xiphinema diversicaudatum* (Micoletsky) with strawberry latent ringspot ar arabis mosaic viruses in a raspberry plantation. *Ann. appl. Biol.* **62,** 147–157.
Taylor, L. R. (1977). Migration and the spatial dynamics of an aphid, *Myzus persicae*. *J. Anim. Ecol.* **46,** 411–423.
Ten Houten, J. G. (1974). Plant pathology: char ing agricultural methods and human society. *A. Rev. Phytopath.* **12,** 1–11.
Thresh, J. M. (1979a). Recent changes in the health statu of En ish hop plantings. *F Br. Crop Prot. Conf.* **1979,** 169–176.
Thresh, J. M. (1979b). Hop-growing in Germany. *Newsletter of the Federation of Britis Pathologists* **2,** 47–48.
Thresh, J. M. and Ormerod, P. J. (1974). Problems caused by wild and re ng hop plants. *Proc. 12th Br. Weed Control Conf.* **1974,** 323–331.
Thresh, J. M., Pitcher, R. S., McNamara, D. G. and Ormerod, P. J. The spread and control of nettlehead and related diseases of hop. *Rep. E g Res Stn.* **1951,** 155–162.
Thurston, H. D. (1973). Threatening plant diseases. *A. Rev. Phytopath.* 27–52.
Timian, R. G. (1971). Barley stripe mosaic virus in North Dakota. *N. Dak. Fm. R* **28,** 3–6.
Tinsley, T. W. (1971). The ecology of cacao viruses. I. The role of wild hosts i incidence of swollen shoot virus in West Africa. *J. Appl. Ecol.* **8,** 491–495.
Tinsley, T. W. and Wharton, A. (1958). Studies on the host ranges of viruses fro *Theobroma cacao* L. *Ann. appl. Biol.* **46,** 1–6.
Todd, J. McA. (1951). An indigenous source of swollen shoot disease of cacao. *Nature Lond.* **167,** 952.
Todd, J. McA. (1960). Tobacco veinal necrosis on potato in Scotland: control of the outbreak and some characters of the virus. *Proc. 4th Conf. Potato Virus Diseases, Braunschweig 1960*, 82–92.
Tomlinson, J. A. and Faithfull, E. M. (1980). Studies on the control of lettuce big-vein disease in re-circulating nutrient solutions. *Acta Hort.* (In press.)
Toohey, C. L. and Nielsen, P. J. (1972). Fiji disease in Bundaberg. *Proc. 39th Conf. Qd. Soc. Sug. Cane Technol.* 191–196.
Trebbi, T. (1950). Il nanismo del mais in Provincia di Brescia nel 1949. *Notiz. Mal. Piante* **8,** 13–16.
Tuite, J. (1960). The natural occurrence of tobacco ringspot virus. *Phytopathology* **50,** 296–298.
Tukey, H. B. and Brase, K. D. (1943). An uncongeniality of the McIntosh apple when top-worked onto Virginia Crab. *Proc. Am. Soc. Hort. Sci.* **43,** 139–142.

Uyemoto, J. K. and Welsh, M. F. (1974). Viruses threaten vineyards, nursery certification reduces the threat. *Proc. N.Y. St. hort. Sci.* **119,** 180–184.

Uyemoto, J. K., Cummins, J. R. and Abawi, G. S. (1977). Virus and virus-like diseases affecting grapevines in New York vineyards. *Am. J. Enol. Viticult.* **28,** 131–136.

Valdez, R. B., McNamara, D. G., Ormerod, P. J., Pitcher, R. S. and Thresh, J. M. (1974). Transmission of the hop strain of arabis mosaic virus by *Xiphinema diversicaudatum*. *Ann. appl. Biol.* **76,** 113–122.

Vuittenez, A. (1970). Fanleaf of grapevine. *In* "Virus Diseases of Small Fruits and Grapevines" (Frazier, N. W., ed.), pp. 217–228. University of California.

Wadley, F. M. (1931). Ecology of *Toxoptera graminum* especially as to factors affecting importance in the northern United States. *Ann. ent. Soc. Am.* **24,** 325–395.

Walkey, D. G. A. and Innes, N. L. (1979). Resistance to bean common mosaic virus in dwarf beans (*Phaseolus vulgaris* L.) *J. agric. Sci., Camb.* **92,** 101–108.

Wallace, J. M. and Drake, R. J. (1972). Use of seedling yellows, recovery and protection phenomena in producing tristeza-tolerant, susceptible, scion-rootstock combinations. *In* "Proceedings of the 3rd Conference of the International Organization of Citrus Virologists" (Price, W. C., ed.), pp. 36–39. University of Florida Press, Gainsville.

Wallace, J. M., Oberholzer, P. C. J. and Hofmeyer, J. D. J. (1956). Distribution of strains of tristeza and other diseases of citrus in propagative material. *Pl. Dis. Reptr.* **40,** 7–10.

Wallin, J. R. (1973). Low-level jet winds, virus vectors, local weather and virus epidemics. Abstract 0314 2nd International Congress of Plant Pathology, Minneapolis.

Wallin, J. R. and Loonan, D. V. (1971). Low-level jet winds, aphid vectors, local weather and barley yellow dwarf virus outbreaks. *Phytopathology* **61,** 1068–1070.

Wallin, J. R., Peters, D. and Johnson, L. C. (1967). Low-level jet winds, early cereal aphid flights and barley yellow dwarf detection in Iowa. *Pl. Dis. Reptr.* **51,** 527–530.

Wallis, R. L. (1967). Some host plants of the green peach aphid and beet western yellows virus in the Pacific Northwest. *J. econ. Ent.* **60,** 904–907.

Watson, M. A. (1959). Cereal virus diseases in Britain. *N.A.A.S. q. Rev.* **10,** 93–102.

Watson, M. A. (1966). The relation of annual incidence of beet yellowing viruses in sugar beet to variations in weather. *Pl. Path.* **15,** 145–149.

Watson, M. A. (1967). Epidemiology of aphid-transmitted plant-virus diseases. *Outl. Agric.* **5,** 155–166.

Watson, M. A., Heathcote, G. D., Lauckner, F. B. and Sowray, P. A. (1975). The use of weather data and counts of aphids in the field to predict the incidence of yellowing viruses of sugarbeet crops in England in relation to the use of insecticides. *Ann. appl. Biol.* **81,** 181–198.

Webber, H. J. (1943). The 'tristeza' disease of sour-orange rootstock. *Proc. Am. Soc. hort. Sci.* **43,** 160–168.

Williams, L. E. and Alexander, L. J. (1965). Maize dwarf mosaic, a new corn disease. *Phytopathology* **55,** 802–804.

Zink, F. W. and Duffus, J. E. (1969). Relationship of turnip mosaic virus susceptibility and downy mildew (*Bremia lactucae*) resistance in lettuce. *J. Am. Soc. Hort. Sci.* **94,** 403–407.

Zink, F. W. and Duffus, J. E. (1970). Linkage of turnip mosaic virus susceptibility and downy mildew, *Bremia lactucae*, resistance in lettuce. *J. Am. Soc. Hort. Sci.* **95,** 420–422.