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ECOLOGY OF SOME CUCURBIT VIRUSES*

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At the outset I express my sincere thanks to The Indian Botanical Society for the award of Panchanan Maheshwari medal to me this year.

One of the problems that a speaker on such occasions has to face is the selection of a suitable topic. I am conscious that I am addressing a gathering of Botanists- a few old, some not so old and many young from different parts of the country and who are engaged in tackling different disciplines of Botany. A majority of you may profess to be modern but I am not entering the controversy about 'traditional' and 'modern', except that as teacher I am reminded of the following words of Sir Geoffrey Vickers (1965): "Even the dogs may eat the crumbs which fall from the richman's table; and in these days when the rich in knowledge eat such specialised food at such separate tables, only the dogs have a chance of a balanced diet".

In the words of Professor Burton (1975) "I have been one of those fortunate dogs and I am sorry for the young botanists of today, most of whom sit at the separate tables, whether these are rich in knowledge or not".

I have chosen to speak today on a subject of Plant Virology with which I had a long association and which, unfortunately, even in modern times does not get as good a treatment as it deserves in the Botany curricula in our traditional universities.

Generally plant virus ecology is regarded as the study of the movement (spread) of viruses from one host to the other and is usually considered in terms of the number and spatial distribution of infected hosts (Burnet, 1955, Shope, 1954, Swenson, 1968b). On closer examination it becomes apparent that the main components involved in the study of virus ecology are the virus, vector, host and environment. It is a highly complex problem, which not only involves interaction between these components but involves interaction of factors within the components.

A successful transfer of a vector dependent virus will depend on the interaction of transmission participants viz. virus, vector and host in a manner that the virus is acquired from its source in a host plant (donor) by a vector and is carried over and inoculated to another susceptible host plant (recipient), the receiving host being so conditioned that infection site develops in it and allows the cell to cell and long distance movement of the virus in itself. A successful interaction of these three components will be possible when proper environment is available to them. In a recent publication Maramorosch and Harris (1981) has termed the effect of various biotic and abiotic components of the environment on pathogen-vector-host

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compatibility as measured by pathogen spread or vector efficiency as "transmission ecology".

THE HOST (DONOR OR RECIPIENT)

The role of plant in the ecology of plant viruses is three fold. It may act as a source of virus for its spread, it may be involved in the biology of the vector and it may be the host in which the damage occurs by the virus.

a. *Source of virus*

Plants act as source of virus if they are grown from infected vegetative parts, or they grow from seeds from infected plants or the seedlings are infected in a seed bed and are transplanted.

Crops propagated by cuttings, budding, tubers, corms, bulbs and suckers obtained from infected plants produce a diseased progeny. Cucumber mosaic virus (CMV) has been reported to be carried in Gladioli, Dahlia and Lilies often without symptoms (Matthews, 1970).

Until recently it was considered rare that true seeds carry plant viruses but the information acquired during the last decade has shown that seed transmission plays an important role in the ecology of different viruses by ensuring survival of the virus from one season to the next or for several years. There has been a phenomenal increase in the number of viruses transmitted through seeds from 53 (Bennett 1969) to 119 (Mandahar 1981) during the last 12 years.

Of the cucurbit viruses, Mandahar (1981) has listed 19 species in which CMV has been shown to be transmitted through seeds of both cucurbitaceous and non-cucurbitaceous hosts; cucumber green mottle mosaic virus through 2 cucurbits; Squash mosaic virus (SMV) through seeds of 8 cucurbits; water melon mosaic (WMV) virus through seeds of *Echinocystis lobata* (Lindberg *et al.*, 1956) and *Cucurbita pepe* (Bhargava and Joshi 1960) but Bhargava (1974) could not get any transmission of WMV though its seeds nor through the seeds of *Momordica dioica*. Grogan *et al.*, (1959) and Thomas (1971) also did not get any transmission of WMV through the seeds of some cucurbits. Other cucurbit viruses transmitted through seeds include melon mosaic virus (Kendrick 1934, Rader *et al.*, 1947, Komuro 1957) and cucumis virus 2B (Capoor and Verma 1948).

Tomlinson and Walker (1973) found that cucumber mosaic virus survived at least 21 months in buried *Stellaria media* seeds from one year to next, providing a means of perennation of the virus. More recently CMV has been shown to be present in bean seeds (Meiners *et al.*, 1977) and mung bean (Iwaki 1978). According to Leppik (1964) SMV was introduced into U. S. by seeds from Iran. From U. S. it was carried to New Zealand in the seeds of *Cucumis melo* (Thomas 1973).

Seedlings produced by germination of infected seeds serve as initial source of virus inoculum from which it is carried away by vectors. In some, seedling may be infected in the seed bed with aphid borne virus from older plantings and serve as primary infection in new planting when transplanted to the field (Broadbent 1957a).

Wild plants and weeds act as sources of virus specially when crops are short lived and absent from the field during winter or summer. Sometimes crops may be absent from the field for long periods of time in crop rotations. During these periods wild plants may be essential for virus survival on which the virus overwinters, over-summers or perennates. As early as 1925 Doolittle and Walker (1925) indicated that CMV overwinters in wild cucumber (*Echinocystis* (*Micrampelis*) *lobata*)

and in roots of other weeds like *Asclepias syriaca, Phytolacca decandra, Nepeta cataria* and *Physalis* sp. Faan and Johnson (1951) showed that CMV is able to overwinter in *Asclepias syriaca, Leoneurus cardiaca, Euphorbia cordata* and *Lychnis alba*. The virus has since been reported from various other weeds by Hein (1953) and Schwarz (1959). Orsenigo and Zitter (1971) showed that *Ptilimnium capillaceum* growing in out of way places was an important source of CMV to celery crop and both *Momordica charantia* and *Melothria pendula* as important source of WMV to cucurbit crops in South Florida. Recently Bruckart and Lorbeer (1976) detected CMV in 12 weeds and considered *Barbarea vulgaris, Cerastium arvense, Rorippa islandica* as likely overwintering hosts of CMV since they appear in the field very early. Rosettes of *B. vulgaris* and green plants of *C. arvense* have been observed throughout the year. Kazda and Hevert (1977) found that CMV passes autumn and winter in seeds of *Stellaria media* which germinate in the following season to produce infected seedlings from which it is carried by aphids to cucumber.

Quiot *et al.* (1979) has shown that seed transmission, infection of perennial weeds and winter growing annuals help CMV to overwinter.

WMV has been reported to be present in several wild plants and weeds viz. *Astragalus sinicus* (Inouye 1964), *Chenopodium album* (Nelson and Tuttle 1969), *Commelina diffusa* (Shanmugasundaram *et al.*, 1969), *Malva parviflora, Medicago sativa* and *Melilotus indica* (Grogan *et al.*, 1969), these having been shown to paly a vital role as natural sources of primary inoculum. More recently two wild perennial cucurbits *Momordica dioica* and *Coccinia grandis* have been shown to serve as a ready source of viral inoculum of WMV (Bhargava, Bhargava and Joshi, 1975). Fegla (1974) has presented data on weed hosts of cucurbit viruses in Egypt. Recently Bos (1981) has very ably discussed the role of wild plants in the ecology of virus diseases.

Similar crops or closely related ones and ornamentals provide yet another source of virus. *Trichosanthes dioica* reported as a natural host of WMV by Bhargava and Tiwari (1970) is largely propagated from creepers and root cuttings and thus is present throughout the year. *Lagenaria vulgaris* is commonly grown both as a summer and winter crop. Once the infection has entered these crops the virus inoculum is available throughout the year (Bhargava, Bhargava and Joshi, 1975). *Lathyrus odoratus* an ornamental has been reported infected by WMV by Nelson and Tuttle (1969).

CMV alone or in combination with other viruses is more widespread in ornamentals like Delphinium, Phlox, Petunia, Viola, Zinnia, Primula, Canna, Chrysanthemum, Gladioli, Lilies, Dahlias, Tulips and *Vinca* (=*Catharanthus*=*Lochnera*) *rosea*. Recently many perennial plants like almond (Topchiiska and Topchiiski, 1976), Rubus (Jones 1976), *Maranta leuconeura Kerchoveana* (Hearson 1979) hve been found to harbour CMV. In Gorakhpur CMV has been isolated from marigold (Joshi and Dubey 1972a), *Salvia splendens* (Joshi and Dubey, 1972b), *Bougainvillea* (Joshi, Dubey and Gupta, 1974), *Tabernaemontana coronaria* and *Clerodendron viscosum* (Joshi & Prakash, 1978).

Role of "volunteer", "self setts" or "groundkeepers" as source of virus inoculum within the crop is also significant. Doncaster and Gregory (1948) have shown that infected volunteer plant in a potato crop became serious sources of potato viruses.

Eulitz (1977) has shown that incidence of water-melon mosaic on vegetable marrow is more dependent on the avail-

ability of virus source than on the presence of aphid vectors.

b. *Availability of Virus in Plants*

Young and vigorously growing plants are often better sources of virus than old ones because the concentration of virus decreases as the plant ceases to grow rapidly. Bhargava (1948) working with CMV showed that the concentration of virus in the inoculated leaf increased upto 10 days but then decreased, probably because of the drying out of the tissue. Virus concentration was greater in systemically infected leaves than the inoculated leaves and reached a maximum in about 16 days after inoculation after which there was a sharp fall in the concentration of the virus with the apparent 'recovery' of the plant and that the concentration continued to decrease with increasing age.

Systemic viruses are supposed to invade all the parts of a plant, but due to differences in the physiological functions in them, viruses are subjected to different conditions in different parts and the virus content of different tissues may vary considerably. Bhargava (1974) in her studies on WMV has shown that the virus was present in all the parts of the plant viz. root, stem, petiole, leaf, sepal, petal, pollen and the carpel though concentration was highest in leaves followed by petiole and stem in *Cucurbita pepo* plants. In fruit trees distribution of viruses is uneven in the tissues and the virus does not become fully systemic, as in apple mosaic reported by Bhargava and Bist (1961).

Sometimes perennial host plants fail to act as a virus sources. *Scrofularia nodosa* and *Valeriana officinalis* perennial marsh plants and susceptible to cucumber mosaic have never been found infected in the field (Tjallingii 1952).

c. *Interaction of hosts with viruses*

Interaction of hosts with the viruses may be severe or mild. If the reaction is merely local, only a few cells of the host are killed and necrotic lesions are formed but if it is wider developing into a systemic disease, the entire plant is rapidly killed resulting in the loss of the virus. Contrary to this, in other cases, the reaction is mild and there is a minimal damage to host leading to a long established virus host association as long as the host is alive. This is helpful in the survival of the virus specially in biennials and perennials. In extreme cases the reaction is more mild and the hosts carry the viruses without any symptoms.

Viruses differ widely in the range of plants they are able to infect. Host range of some viruses is very specific and confined to one or two genera in a family while other viruses are able to infect a large number of plants distributed over numerous families, belonging to both monocots and dicots.

A diversity of hosts provides a virus greater opportunities to survive and spread while viruses with narrow host range are limited in their geographic distribution. Of the cucurbit viruses, CMV is now known to be infectious to 242 genera of 64 families of flowering plants including several wild species and perennial plants (Bos 1981); cucumber necrosis virus (CNV) is infectious to plants of 6 families. Natural hosts range of SMV is limited to cucurbitaceae and experimentally it infects plants in 5 other families. WMV has been found to occur naturally in cucurbitaceae and leguminosae but plants in 17 families could be infected experimentally. Consequently CMV and WMV have a world wide geographical distribution while squash mosaic virus has a limited distribution and cucumber necrosis virus is restricted to Canada only (Gibbs & Harri-

son 1970, Campbell 1971, Van Ragenmortal, 1971, Dias and Mckeen 1972).

Different cultivars of a crop may differ in susceptibility to infection. Bhargava and Bhargava (1976) studied the reaction of 80 cultivars of nineteen cucurbits to different strains of WMV and found that cultivars behaved differently with different strains. Some were susceptible while others were either resistant or symptomless carriers of one or more strains.

d. *Interaction with vectors*

Plants are involved in the biology of the vector.

Oman (1959) in discussing the role of plants in the bionomics of leaf hoppers refers to 2 categories of plants. "Host plants" on which eggs are deposited and on which nymphal development takes place, and "Food plants" which are utilised as sources of food by the adults but are not normally used for oviposition.

Kring (1959) has observed that true migratory (dispersing) aphids produced eggs on primary host on which oviparous females were found. On secondary hosts only viviparous females were found and male aphids were produced only on these.

It has also been observed that multiplication of aphids is faster on better fed plants. Janssen (1929) found that increasing nitrogen increased aphid numbers and a deficiency of potash favoured aphid reproduction on potato plants. During the dry season irrigated crops and pastures provided feeding and breeding areas for vectors for several months (Rose 1974).

Rate of intake of sap by aphids feeding on plants is determined by the concentration of aminoacids in their sap. Dixon (1975) has observed that intake was slow when the aminoacid concentration is high and was fast when it was low.

Different plant species may vary in their effectiveness as sources of the same virus for the aphids. Simons (1959) reported that although pepper is a better host plant than chard for aphids and more susceptible to the Southern cucumber mosaic virus, the aphids acquire the virus more readily form chard than from pepper.

e. *Interaction with environment*

Physiological state of a plant changes with the environment and susceptibility of a plant to infection depends greatly on its physiological state. Plants growing under shade conditions having pale delicate leaves are more susceptible than those grown in bright sunlight. Darkening the plants before inoculation usually increases their susceptibility (Bawden and Roberts, 1947, 1948). Bhargava (1948) working with bean plants and CMV found that plants which kept throughout in darkness, both before and after inoculation, gave the highest number of lesions.

There is, however, probably more than light intensity concerned in the seasonal variation in reaction of beans to CMV. Attempts to produce local lesions during summer by growing beans under various conditions of light intensity and exposed to light for various periods daily all failed (Bhargava, 1948, 1951).

Bhargava (1951) found that *Phaseolus vulgaris* culv. Prince and Bountiful were apparently immune from CMV infection during the summer but during the winter produced local lesions from which virus was recoverable. Badami (1959) working with the same CMV strains found that temperature at which inoculated plants are kept affects the development of symptoms. Spinach strain of CMV failed to multiply and cause symptoms in tobacco palnts kept above 30°

C, but the yellow strain infected at 36°C. Spinach strain was more active at 24°C while yellow strain caused more severe symptoms at 20°C.

THE VIRUS

a. Physical stability and concentration in tissues

A virus with a high degree of stability both inside and outside the plant has greater chances of survival and spread than one which is highly unstable. Also a virus in an infected plant may be more readily available to a vector when the concentration of the virus is high. Aphids have been found to acquire the virus more readily from younger leaves than from the older ones where the concentration of the virus is low (Kirkpatrick and Ross 1952).

b. Host range of a plant virus

Plant viruses vary greatly in the range of species which they are capable of infecting. Some are highly specific and have a narrow host range while others are able to infect a large number of species in many families including herbaceous and woody plants (cf. CMV and SMV). The latter viruses termed as 'polyphagous' have greater opportunity to maintain themselves and spread widely. Survival of viruses with narrow host range may probably be due to their having perennial hosts, or being vegetatively propagated or transmitted through seeds (Matthews 1970). The latter process is very evident in SMV.

c. Ability to change-mutability and strain selection

Living organisms which are generally able to adapt themselves to their changing surroundings have better chances of survival than others which are unable to adapt themselves and soon disappear. Survival of the virus and its dispersal may well be affected by the extent to which it can change or is able to mutate and produce strains that can cope with the changes in environment. These variants may differ in host range or in their reaction to a particular host or virulence or in vector specificity, thus affecting their ecology or spread.

Bhargava (1951) showed that different strains of CMV differ in their host range, symptoms caused, virulence towards different plants, transmissibility by aphids and concentration in hosts. One strain of CMV isolated form a spinach plant was readily transmitted by *Myzus persicae* until 1955 when it lost this property though it continued to be transmitted by *Aphis gossypii* and *Myzus ascalonicus* (Badami 1958).

Bhargava and Bhargava (1976) isolated seven strains of WMV which reacted differently with various cultivars of cucurbits.

d. Mixed infections and single infections

In several crops and fruit trees mixed infections of plant viruses are quite common in nature, but most plant viruses are probably transmitted independently and presence of two or more viruses shows no interaction between them. Some viruses, however, when transmitted by insect vectors are dependent on the presence of another virus or virus product. Dependent transmission of nonpersistent viruses has been reviewed by Pirone (1977). Aphids can acquire the helper component and the virus simultaneously, or they may acquire the helper component prior to acquiring the virus.

e. Distribution of virus and rate of movement

Many viruses which are systemic, spread through most tissues of the host plant. Those which can move into seeds or underground parts used for vegetative

propagation have better chances of survival and spread.

Viruses more from cell to cell within plants through plasmodesmata but move from one part of the plant to the other through the vascular system. Inability to enter the phloem may be the cause of restriction of some viruses to the inoculated leaves.

Viruses find it more difficult to enter mature leaves and many viruses are unable to establish in the meristematic regions of root or shoot.

Viruses which move rapidly through the plant from the point of infection have better chances of survival and spread, though in shrubs and trees slow movement of viruses has no adverse effect.

f. Interaction with the Vector

In the present treatment aphid vectors are being considered in details because CMV and WMV are carried by them. SMV is carried by beetles and cucumber necrosis virus is transmitted by zoospores of *Olpidium cucurbitacearum*. Viruses transmitted by aphids have been separated in different groups depending on the length of time the viruses persist in the aphids.

Watson and Roberts (1939, 1940) found that the viruses transmitted by aphids fall into two groups : "non persistent" which survived in their vectors for only short periods, less than the survival time of the virus in untreated leaf extracts. and "persistent" surviving in their vectors for long periods, sometimes for whole life of the insect. Kennedy *et al.* (1962) proposed the term "stylet borne" for viruses carried on the stylets and include non persistent viruses also. Persistent viruses were preferred to be called as 'circulative' and these were able to pass through the gut wall into the haemolymph of the vector, eventually reaching the mouth parts after passing the salivary glands but not multiplying in the vector. Those circulative viruses which multiplied in the vector were termed as "propagative". Another category which is intermediate is "semi persistent" which resembles non persistent but persists for long period in the aphid.

Various strains of CMV and WMV have been conclusively shown to be of stylet borne type. SMV is transmissible by beetles and a grass hopper. It is retained upto 20 days. Virus multiplication has not been reported and the virus has been recovered from regurgitating fluid, faeces and haemolymph. Haemolymph seems to be a reservoir of virus that may play an important role in virus transmission (Freitag 1956).

Some persistent viruses have been found to multiply in the aphid vector (potato leaf roll in *Myzus persicae*) by Stegwee and Ponsen (1958) but no non-persistent virus has been able to do so. With stylet borne viruses most aphid populations appear to benefit from the diseased plant either by an increase in the growth rate of the individual or by increased growth rate of the population. There appear to be few detrimental effects on their aphid vectors.

Recently evidence has been presented that chemical composition of the virus may affect transmission by aphids and play a critical role in vector specificity. Gera *et al.* (1979) have concluded that apparently coat protein determines transmissibility of CMV strains by aphids, replacement of coat protein increased or decreased its transmission. One strain of WMV has been described which is not transmissible by aphids (Molnar and Schmelzer 1964) and one isolate of WMV could not be transmitted by *Myzus persicae* or *Aphis gossypii* (Thomas 1971).

THE VECTOR

I. General

a. Type of Vectors

A majority of plant viruses are carried by members of insecta represented by aphids, white flies, leaf and plant hoppers, beetles, thrips, mealy bugs, psyllids and grass hoppers. In addition nematodes and fungi have been found to transmit soil borne viruses. CMV has been reported transmissible by more than 60 spp. of aphids (Gibbs and Harrison, 1970); WMV by about 11 spp. of aphids (Condriet, 1962, Karl and Schmelzer, 1971); Squash mosaic by 3 beetles and 1 grass hopper (Campbell 1971) and cucumber neocrsis by zoospores of *Olpidium cucurbitacearum* (Dias and Mekeen 1972).

b. Alatae (winged) or Apterae (wingless)

Swenson (1968b) has reported that in temperate areas aphids lay their eggs on the primary host in winter which hatch in spring and alatae (winged migrants) are produced by parthenogenetic reproduction sometime after the first generation. These migrants fly to the secondary hosts where colonies of apterae (wingless) are established.

Lal (1955) related production of alatae to the decrease in the water content of the aphids and of their hosts. Most authors agree that production of alatae is influenced by the factors of light, temperature, crowding, starvation, wilting of the host plant and even parentage (Carter, 1961). Lees (1966) has reported that production of alatae is associated with crowding or physical contact between aphids. Deteriorating physiological condition of a plant results in the movement of the aphids on the plant, increasing the amount of physical contact between aphids and providing the stimulus for formation of alatae. Kennedy et al. (1958) has reported increase in the production of winged forms due to increased concentration of solutes in the sap.

Both alate and apterous aphids transmit styletborne viruses. When apterous aphids are eliminated by insecticides, there is no control of stylet borne viruses and it is thought that these viruses are spread by alate aphids (Broadbent 1957 b). One must not conclude that apterae do not spread stylet borne viruses at all (Van der Wolf 1964), but that the long feeding periods of colonising apterae are not effective for acquisition of these viruses. Alate aphids would acquire and transmit stylet borne viruses too rapidly for insecticides to have any effect (Swenson, 1968b).

c. Feeding behaviour

All insect vectors of plant virus diseases, as far as known, are polyphytophagous.

Aphids have a uniform piercing and sucking habit. The mouthparts consist of two pairs of needle like stylets, with a labium and a labrum. Stylet bundle is composed of 2 mandibular stylets and 2 inner maxillary stylets. In most cases the virus is acquired primarily form the epidermis. In a longer feeding aphid's stylets usually followed an intercellular path through such tissue, and saliva is ejected ahead of the stylets and coagulates to form a sheath around them.

Beetles have biting mouthparts and no salivary glands. They regurgitate part of the contents of the foregut while feeding. Regurgitation brings previously eaten infective material into contact with the leaf. During process of mastication it is inoculated into the leaf (Matthews 1970).

d. Aphid biotypes

Generally identification of the aphid vector to the species level is usually considered sufficient. Yet genetic variants within a vector species may arise and may be perpetuated within a line of aphids by parthenogenetic reproduction. These variants or biotypes differ in the degree of feeding injury to host plants, their ability to develop on species or cultivars within the host range of their species, preference for yellow or orange colour, ability to form sexual forms, tendency to produce winged virginoparae, resistance to insecticides and virus transmission (Swenson 1968b). Biotypes of *Aphis gossypii* differing in ability to transmit CMV have been reported by Simons (1959).

e. Aphid specificity

Aphids vary widely in the number of different viruses they can transmit. *Myzus persicae* is known to transmit more than 70 stylet borne viruses while there are others which transmit only one. Quite closely related species may differ in their ability to transmit viruses, and one species may be very much more efficient than another. Similarly different forms of an aphid species (alate or apterous viviparous females) adapted to different seasons and different host plants may vary in their efficiency (Matthews 1970).

f. Pedators, parasites and helpers

Pedators and parasites help to determine the ultimate size of the population of vectors, and also alter their seasonal cycle. Hille Ris Lambers (1955) found that a year with numerous aphids was followed by one with few; parasites and predators multiply abundantly in seasons when aphids are numerous in summer, and many then overwinter and help to prevent the aphid infestation from developing the following spring.

Rizvi, Bhargava and Joshi (1972) have reported a case of helping role of ants like *Polyranchis binghami* and *Lophomyrmex bedoti* where these were found associated with the colonies of *Melanaphis sacchari, M. indosacchari* and *Crematogaster politula* with the colonies of *Rhopalosiphum maidis*. The ants feed on the honey dew secreted by the aphids, thus keeping the surface clean indirectly encouraging the aphids in their multiplication. They also built covering on them with fine grains of soil, dirt and borer frass to protect them from their natural enemies and rain.

II. Interaction with the host

Transmission by aphids decreases with the increasing age of the plants. Stimmann and Swenson (1967) have reported that the number of limabean plants infected with CMV by aphids feeding on the primary leaves was about halved with a two-day increase in age.

Alatae are produced on the primary hosts and increase in number by parthenogenesis. Colonies of wingless aphids (apterae) are established on the secondary hosts. The secondary hosts of an aphid species may consist of one of a few closely related species or of numerous unrelated plant species. Related species of aphids are likely to have related primary hosts (Hille Ris Lambers 1950).

In milder climates a species may exist entirely on secondary hosts as parthenogenetic viviparous generations, without need of the primary host.

Myzus persicae multiplied faster on yellows-infected sugarbeet plants as compared to healthy plants and this is attributed to the higher content of potassium in infected than in healthy leaves (Hijner and Martinez Cordon, 1955).

Alighting of aphids on hosts rather than nonhosts is nondiscriminatory, and is the result of different departure rates (Kennedy et al 1959a, b). Dispersal takes precedence over hosts finding in aphid migration (Kennedy, 1960). Aphids which alight on nonhosts probe briefly and depart, unless the environmental conditions are unfavourable. These qualities ensure spread of stylet borne viruses in crops that have no colonizing vector species as has been reported for CMV in gladioli (Swenson and Nelson, 1959).

III. Vectors and Virus Spread

a. Number of aphids

Generally there is a correlation between the virus spread and number of aphids. Broadbent (1950) has reported that annual spread of potato leaf roll and virus Y in England and Wales was correlated with the number of alate *Myzus persicae* caught on sticky traps.

b. Time of aphid flights in relation to crop

Time of aphid flights and subsequent spread early in season is more serious than late season spread because plants infected early in the season are more important virus sources for secondary spread than are plants infected later. Moreover early in the season plants are young and more susceptible to virus infection by insect transmission than are older plants (Swenson 1968a). Plants size also has been found to affect the incidence of disease because big plants are more likely to be visited by vectors than the small ones (Van der Plank, 1947, 1958).

c. Virus spread into the crop

If the crop is raised from diseased free seeds, the plants will be healthy until viruses are brought by vectors form sources of infection outside the crop. When the source is near, a gradient in the incidence of infection will be visible.

Non persistent viruses cannot be carried far, though persistent viruses may be, because aphids are known to travel long distances. Disease gradient will depend on the mobility of vector conditioned by weather conditions specially the wind and the temperature. If disease incidence is high in outer rows and low within the crop, it is diagnostic of spread from a nearby source outside the crop.

The incidence of disease in the crop which spread into the edges of fields from outside sources will be proportional to the total length of the edges of the crop. According to Van der Plank (1949a, b) large fields will contain a smaller proportion of diseased plants than small ones, because large crops have a smaller ratio of edge to the total area.

When plants are grown as a mixed crop, different plant species are not hosts for the same viruses and vectors. Viruses specially the stylet borne, spread less in such crops than in crops of a single species. In situations where the crop is surrounded or covered by other plants, virus may not spread at all. Tobacco plants in Japan are grown between rows of barely to protect them from aphids carrying CMV. (Gibbs & Harrison, p. 224, 1976).

d. Virus spread within the crop

It is a common observation that plants closest to primary infection were most likely to be infected, the probability of infection decreasing with increasing distances. It was believed that virus spread from one field to another was carried by alatae but the subsequent spread from these primary infections within the field was by wingless aphids (Watson, 1942).

Broadbent (1950), however, sugge-

sted that alatae were more important than apterae in within-field spread of viruses. This was supported by Jenkinson (1955) and Watson and Healy (1953) but challenged by Ribbands (1963, 1965). There is now general agreement that nonpersistent (stylet borne) viruses are spread primarily by alatae aphids but one must not conclude that apterae do not spread such viruses at all, but that the long feeding periods are not effective for acquisition of these viruses.

Disease incidence in crops brought about by winged aphids is influenced by planting density. When a given area contains few plants, a larger proportion will be infected than when it contains many because the number of virus carrying insects flying into a given area of crop is independent of the number of plants in that area (Gibbs and Harrison, 1976). Most insects bring virus into the crop land at random, so a greater proportion of plants will be visited when they are widely spaced than when they are crowded together.

THE ENVIRONMENT

As has been indicated in the beginning and also mentioned subsequently at certain places, environment is closely linked with other components of virus ecosystem and macroenvironment has been discussed in relation to some components.

The environment here is being discussed under physical and biotic environment.

I. Physical factors

a) Wind velocity

Wind determines the predominant direction of the spread of viruses by controlling the direction of aphid flight. Winged aphids do not fly when wind speed is too great, but their direction of flight is controlled by the prevailing wind. Sometimes strong winds transport aphids over long distances. Thomas and Vevai (1940) suggested a wind velocity below 5 m.p.h. suitable for aphid flights and Haine (1955) showed that aphids can control their flights when air is moving slower than 1½ mph.

b) Humidity

Moderate wilting near the time of inoculation increases susceptibility. Bean Plants in an atmosphere with high relative humidity before inoculation with tobacco necrosis virus increased their susceptibility as compared with plants kept at low humidities (Kimmins and Litz, 1967).

High humidity inhibits flight of vectors and relative humidity below 80 is favourable for flight. Long day periods are favourable for the production of alatae (Thomas and Vevai, 1940).

c) Temperature

Preincubation of plants at somewhat higher temperatures than normal before inoculation increases susceptibility while effect of treating plants at a higher temperature after inoculation may vary with the virus (Kassanis 1952) and the strain of virus tested (Helms 1965). More lima bean plants were infected with CMV by *M. persicae* when exposed to 30°C for 2 days immediately after inoculation than when exposed to 15°C for 2 days (Stimmann and Swenson 1967).

Temperatures from 70° to 90° F were the most favourable for flights of *Myzus persicae* (Thomas and Vevai 1940). High temperatures cause restlessness among aphids and reduce aphid population. Large fluctuations in temperature are favourable for the production

of alatae. The lowest temperature at which any aphid was observed to take off was 15.5°C (Johnson and Taylor 1957).

d) Light

As has been mentioned above darkening the plants before inoculation usually increases susceptibility. Stimmann and Swenson (1967), however, reported that susceptibility of lima bean plants to CMV was not affected by keeping the plants in darkness for two days before inoculation by *Myzus persicae*.

Light intensity has been found to affect flight activity. Light intensity between 100 and 1000 ft. C. made little difference to flight, but below 100 ft. C. flight activity declined rapidly in artificial light. Markkula (1953) recorded differences in aphid take off in varying degrees of sunlight. Aphids are more attracted to green, yellow and orange light (wave lengths of 500 to 560 m μ) than to short wave light (Kennedy et al. 1961).

II. Biotic Factors

Activity of man has been responsible for the spread of many viruses that were localized in restricted geographical areas of the world. Thus many virus diseases of potato have been spread from America to many countries of the world. Bos (1981) has recently pointed out the danger of spreading viruses over long distances even between continents, in wild plants through botanical gardens with diverse collection of wild species from all over the world and regular international exchange of material. Exploration of the world's wild gene centres and exchange and transport of wild germ plasm form country to country from gene banks also pose the same danger.

Nearer home man forces inter host activity of the vectors by disturbing them while removing the weeds harbouring them. This causes the alatae to fly off and apterae to move to the nearest crop in search of food. Similarly storage of bulbs and other crops like mangold and beets provide hibernating places to aphids as well as virus survival.

Another practice which helps in the survival and retention of virus in a locality is the successive cropping of the same plant or related plants in the same or nearby locality throughout the year WMV survives in many localities in cultivations of *Trichosanthes dioica* as reported by Bhargava and Tewari (1970) and *Lagenaria vulgaris* (Bhargava, Bhargava and Joshi, 1975). In many localities *L. vulgaris* is seen growing on thatched huts out of season and serves as a potential source.

Crop selection and location may influence disease incidence. Location of one particular crop infected with a virus may provide inoculum to another crop which is suceptible to that particular virus. Rishi, Bhargava and Joshi (1973) have reported crops of *Zea Mays, Sorghum vulgare, Pennisetum typhoides* and *Eleusine indica* acting as source of inoculum of sugarcane mosaic virus from nearby sugarcane fields.

CONTROL OF VIRUS DISEASES VIS-A-VIS THEIR ECOLOGY

Information gained from the ecological studies can be utilised for protecting crops from damage by viruses. This can be done by—

1. Avoidance or removal of sources of infection
2. Prevention and reduction of virus spread by preventing insect vectors and modifying cultural practices.

I have given you some idea about the various factors and their interactions in plant virus ecology. I have merely

touched the fringe of the problem as I have talked about only a particular group of viruses and vectors. There are numerous other virus diseases with a host of varying vectors. The interactions I have indicated are complicated and their study might indicate ways of managing crop ecosystems to control virus diseases. This will require continuous research by plant pathologists, if human society is to get the maximum yield of these crops. Snyder (1971) has rightly pointed out that "the obligation that the plant pathologist has accepted is to society—world society—not to a crop or a culture".

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