

Geminivirus Diseases

The geminiviruses are the most recently described group of plant-pathogenic viruses, and their distinctive morphology has been known only since 1974. In that year, two of the earliest agriculturally important diseases to be documented, beet curly top (subsequent to a major outbreak in California in 1899) and maize streak (reported from South Africa in 1901), were shown to be caused by viruses with a novel type of particle structure different from any previously described (3,7). Subsequent to these reports, geminiviruses were found to be the cause of, or associated with, several tropical and temperate plant diseases of hitherto unknown etiology.

This sudden and rapid emergence of a new group of plant pathogens is remarkable in that some of them are the causal agents of diseases that have long been the subject of a great deal of sustained and intensive research. Several are amenable to more or less conventional methods of purification and at least one attains sufficient concentration in experimental hosts to be seen in crude sap in the electron microscope.

As will be seen, one geminivirus, the cause of maize streak, was the subject of a series of classical studies on virus/vector relationships made by H. H. Storey in eastern Africa as long as 50 years ago (10). Another, tobacco yellow dwarf virus, was the cause of relocating a tobacco-growing industry in Australia. At least three diseases—maize streak (Fig. 1), cassava mosaic (Fig. 2), and bean golden mosaic—are the subject of ongoing intensive research programs in various parts of the tropics.

It is likely that yet another disease caused by a geminivirus has a further distinction. In the summer of the year 752 the Empress Kōken of Japan wrote a poem, which was to be included in the classical Japanese anthology Manyōshū, in which she described the beauty of the yellow leaves of the plant now called *Eupatorium chinense* (Fig. 3). She thus unwittingly contributed what is possibly the first report in the literature to symptoms of a plant virus, since the yellow vein mosaic of *Eupatorium* is caused by a geminivirus (6). The delay between recorded effect and knowledge of cause in this possible instance was over 1,200 years, a somewhat longer period

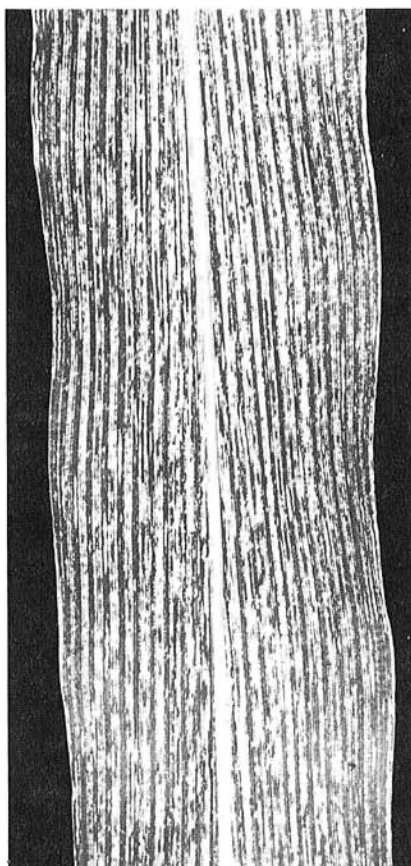


Fig. 1. Symptoms of maize streak virus in maize.

than that established for other geminiviruses.

Although the diseases caused by tropical geminiviruses are given special attention in this article, the pathogens are reviewed in summary as a group.

Morphology and Particles

The name geminivirus is derived from the characteristic double particle (from the Latin *gemi* = twins) in which isometric particles about 18–20 nm occur predominantly in pairs measuring about 20 × 30 nm (Fig. 4). Single particles appear in most preparations but are always greatly outnumbered. In addition to their morphology, the geminiviruses seem to be remarkably uniform in other properties. The sedimentation coefficient of particles is about 70 S. They contain one molecule of single-stranded DNA with a molecular weight of $0.7-0.9 \times 10^6$, and in this also they are for the moment unique. The majority of plant viruses contain RNA and only one other group possesses DNA, but the nucleic acid with

these is double-stranded. Particles also contain a single coat protein of molecular weight 28,000–34,000 and seem to be efficient immunogens. This is an important feature as it enables some confidence to be placed in serological relationships or nonrelationships.

There seems to be a tendency for the geminiviruses to be confined to the phloem, where particles occur in the nuclei of infected cells. While this is apparently so for at least one whitefly-transmitted virus (bean golden mosaic) and one leafhopper-transmitted virus (curly top), particles of chloris striate mosaic virus appear to accumulate in the nuclei of cells of all types of leaf tissue except the epidermis. Whatever their relationships with specific tissues might be, the fact that they are or are not confined to phloem does not seem in itself to be correlated with positive mechanical transmission.

Classification

While as viral entities the geminiviruses appear to be remarkably uniform, in several other respects they are a most disparate group. Table 1 indicates an obvious primary division into two subgroups based on vector specificity, one containing those transmitted by leafhoppers and the other, those transmitted by the whitefly *Bemisia tabaci*.

Each subgroup is amenable to further division. Thus, among the leafhopper-transmitted viruses are three that apparently infect members of the Gramineae only. All three vectors of these are contained in one Cicadellid tribe, the Deltoccephalini, but in two subtribes. The remaining two viruses (which are responsible for three distinct diseases) infect dicotyledonous plants: Beet curly top occurs in North America (and probably also in Central and South America) and bean summer death and tobacco yellow dwarf occur in Australia. For those with tidy minds, the leafhopper vectors of these serologically related American and Australian viruses are classified not only in the same tribe (Deltoccephalini) but also in the same subtribe (Platymetopiina), which seems to suggest to plant virologists, at least, that insect taxonomy is basically sound (5). None of the viruses in the leafhopper-transmitted subgroup has been transmitted mechanically by conventional sap inoculation, although curly top has been transmitted by making repeated punctures

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with insect pins through drops of infected sap into the crown of beet seedlings and, more recently, by using a high-pressure injector gun.

The subgroup transmitted by *B. tabaci*, which is of very great tropical interest indeed, can be divided simply into viruses that are more or less readily transmitted by conventional sap inoculation and those that seemingly are not. It is possible that at least some of the latter will ultimately prove to be so.

Transmission

None of the geminiviruses appears to be seed-transmitted, a factor of significance in international exchange of seed material of plants susceptible to them.

A great deal is known about the virus/vector relationships of maize streak (9,10), and we may assume that those of chloris striate mosaic (Australia) and wheat dwarf (Sweden) will prove, within limits, to be much the same. Maize streak virus is acquired by the vector in less than 1 hour. There follows a latent period in the vector of 6–12 hours (at 30 C) during which virus is not transmitted, but when this period has elapsed, virus may be inoculated in 5 minutes. Storey was able to account for this sequence of events. He found that the vector of maize streak, *Cicadulina mbila*, exists in two forms: those able to transmit (which he called *active* for transmission) and those unable to transmit (*inactive*). By puncturing the gut wall of inactive individuals or by injecting virus into their hemocele, Storey was able to render inactive leafhoppers active for transmission. From the results of these and other elegant experiments he deduced that the latent period represented the time needed for the virus to move through the gut wall into the body cavity and thence to the salivary glands, where it became available for inoculation. Storey also found that the ability to transmit is inherited as a simple dominant gene linked with sex, but that individual insects of an active race are not equally efficient in transmission because other genes, not sex-linked, apparently can modify the effect of the major gene for activity.

The virus/vector relationships of the tropical whitefly-transmitted geminiviruses are remarkably similar to those of maize streak, which may perhaps be regarded as the standard geminivirus model. Although there are expected individual variations in acquisition, latent, and inoculation periods, they are,

within limits, of the same order as those of maize streak, and the overall pattern is much the same. We may reasonably assume that the pathways of virus movement in the vector suggested by Storey for maize streak are also the same for the majority of the whitefly-transmitted geminiviruses.

Once virus has been acquired, leafhopper and whitefly vectors alike remain infective for periods varying individually from 7 to 21 days, or longer. With the vectors so far studied, the ability to transmit is retained during molting, but none is transmitted through the egg. With most of them, the transmitting efficiency of the female insect is higher or much higher than that of the male. One other



Fig. 2. Symptoms of cassava mosaic disease in cassava.



Fig. 3. Symptoms of *Eupatorium* geminivirus in *Eupatorium chinense*. The attractiveness of the yellow vein net of this disease possibly was the subject of a classical Japanese poem written in the year 752. (Courtesy T. Inouye)

feature common to most geminiviruses is the inconsistency of the transmission pattern. After successfully inoculating virus to successive plants, insects often fail to transmit, but then recover the ability to do so in subsequent serial transfers.

Host Range and Symptoms

The symptoms induced by the geminiviruses specializing in the Gramineae are similar, consisting mainly of leaf streaks. The natural host range of maize streak virus in Africa is extremely wide within the Gramineae but presents a complicated picture. This tropical geminivirus occurs in distinct strains, several of which are host-adapted to indigenous grass species. These strains differ greatly in individual host ranges and pathogenicities but are nevertheless serologically related. For example, the strain that induces maize streak disease either does not infect sugarcane or causes transient infection and symptoms. Only a few streaks appear, and the virus cannot be recovered from later-formed streakfree leaves. The sugarcane strain is transmissible to maize, where it induces persistent but mild symptoms. A third strain, from guinea grass (*Panicum maximum*), infects neither maize nor sugarcane. As a further complication, the maize strain itself exists as variants or substrains (incidentally a factor of importance in work involving selection for resistance).

It seems, thus, that maize streak is ecologically a most successful virus of great antiquity that, by specializing in a myriad of host-adapted strains, has insured itself against the possibility of short-term disaster to individual host species. This strategy of a multiplicity of strain-specific hosts has also provided a springboard that offers further endless opportunity for variation and thus evolutionary success. Thus, the disease one particular virus strain causes in maize (a species of plant exotic to Africa) is entirely incidental to survival strategies, and that crop represents no more than a fortuitously provided and seasonal host for the vector.

The host range of beet curly top virus is very wide indeed, and this virus infects more than 300 species in 44 plant families. It is to be expected that the host range of the related Australian virus (tobacco yellow dwarf) would be similarly wide (which it is—32 species in seven families, to date).

The host ranges of the whitefly-

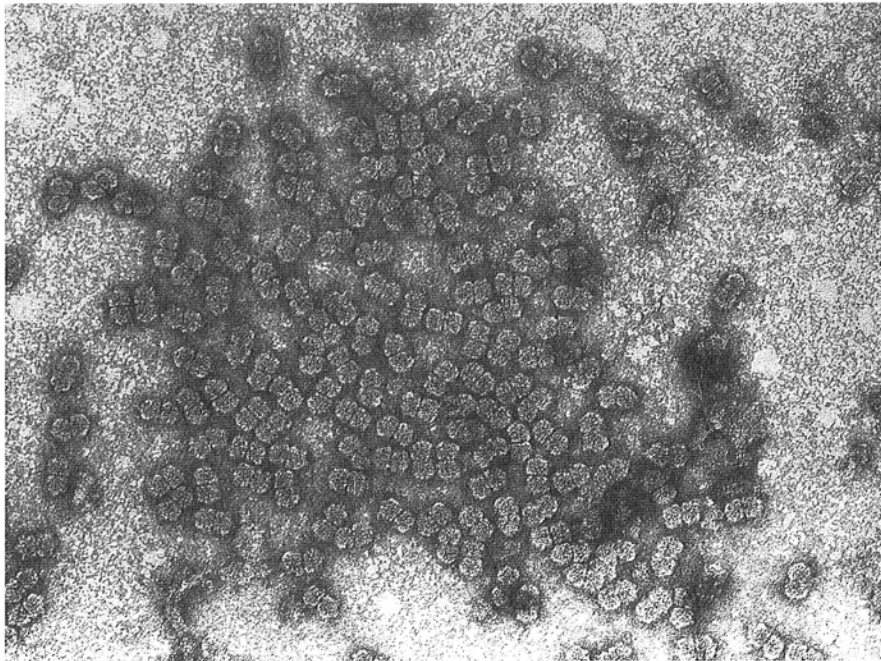


Fig. 4. Geminivirus particles of maize streak virus. A particle is about 30 nm long. (Courtesy R. D. Woods)

transmitted geminiviruses are somewhat more restricted. The most specialized of these is bean golden mosaic, which seems to be restricted to only certain species in a few genera of one tribe (Phaseoleae) of the Papilionaceae. All the rest, including the curly top viruses of the New World and Australia, infect at least one genus in the Solanaceae, regardless of the family in which their "natural" host occurs. Curiously, within the Solanaceae, they all infect species in the genera *Datura* (almost invariably *D. stramonium*) and *Nicotiana* (almost invariably *N. tabacum*). Significantly, most also infect *Lycopersicon* (*L. esculentum*, the tomato). Thus, for example, the African geminivirus associated with cassava (*Manihot esculenta*, Euphorbiaceae, a "field" host but not a "natural" host, as cassava is an exotic species in Africa) infects several species of *Nicotiana*, including *N. tabacum*, and at least two species of *Datura*, including *D. stramonium*. The penchant for these two genera shown by unrelated viruses of diverse geographic regions might well prove to be a consistent property of the less specialized geminiviruses and, when further data are accumulated as more geminiviruses are described, might provide a useful area for speculation on origins and evolutionary trends within the group.

The names assigned to the various diseases are indicative of the three most consistent symptoms induced by those geminiviruses that infect dicotyledons: leaf-curling, yellowing (often bright), and stunting. Thus we have curly top, leaf curl, yellow leaf curl, yellow dwarf, golden mosaic, and yellow vein mosaic. These symptoms are also to be found consistently in experimental hosts, particularly among the Solanaceae.

Agricultural Significance

Diseases caused by the tropical geminiviruses are notably damaging. Loss in yield caused by maize streak is directly related to time of infection. Although losses depend to some extent on virus strain and host variety, in some areas plants infected less than a week after germination produce no yield or may be killed. In other areas it has been found that seedlings infected at the second leaf stage suffer a grain weight loss of about 55%; plants infected at the fourth, sixth, eighth, and tenth leaf stage lose 45, 40, 33, and 25%, respectively.

Bean golden mosaic virus apparently reduces the number of pods, the number of seeds per pod, and seed weight. Reported yield losses vary from 40 to 100% and, as with maize streak, depend greatly on the age of the plant at time of infection, on varietal differences, and possibly on strains of the virus.

Although the precise role of cassava latent virus in cassava mosaic disease has not as yet been resolved, the geminivirus is invariably associated with it and is likely to prove to be the cause. In Kenya, cassava mosaic disease causes a loss of about 70% in tuber yield of plants derived from infected cuttings, and this loss seems to be more or less independent of the genetics of the variety.

Epidemiology

The epidemiology of two of the leafhopper-transmitted geminiviruses is known in some detail (1,8). The more intensive studies on maize streak disease have been restricted to Zimbabwe in subtropical southern Africa, where the climatic pattern is one of a cold dry season (May to August), a hot dry season (September to November), and a warm

wet season (November to March). In the natural rain-fed system, the population levels of the vector in the extensive natural grasslands it inhabits are lowest in September or October, at the end of the hot dry season. The November to March warm rainy season sustains the development of two generations, during which time populations increase but numbers are still low. April marks the beginning of the flight or dispersal season, when the natural grasses of the plateau mature and dry, and this dispersal peaks toward the end of April and during May. Subsequently, numbers decline until the advent, in November, of the rains. If the rainy season extends into May, growth flushes of grass occur on which the April dispersal peak settles, and another generation of hoppers occurs. This generation develops and matures slowly because of the lower winter temperature but subsequently leads to a pronounced peak in August.

Neither of these population sequences and dispersals results in streak disease epidemics in the rain-planted maize crops in November, and the disease is only rarely encountered. When, however, irrigated cereal crops are interpolated in the cold dry or hot dry season, streak assumes epidemic proportions. The dispersal flights in April encounter the winter wheat crop, and successive waves introduce streak and build up dense populations of infective insects. Then, as the wheat matures, dispersal flights ensue into irrigated maize crops, and it is in these that maize streak is most devastating.

The situation in equatorial Africa is not known but also seems to be primarily a function of rainfall distribution. In many areas, the disease attains epidemic proportions only in years when successive plantings of maize are possible—and invariably made. Whatever the situation, it is likely that natural grass reservoirs, while of supreme importance in maintaining the streak geminivirus, do not contribute to epidemics. The major source of virus is that derived from previously planted and infected maize.

When first reported in São Paulo, Brazil, in 1961, bean golden mosaic virus was considered a minor disease. It has since become an economically important disease in many countries of South and Central America and the Caribbean area (4). It is interesting to note that the disease's increasing seriousness in areas of Brazil has been attributed in part, at least, to an increase in populations of the whitefly vector. This, in turn, has been caused by the expanding cultivation of soybean; although not a host of the geminivirus, soybean supports dense populations of the vector that disperse to the bean crop when it matures. It has also been suggested that tobacco, tomato, and cotton plantings in El Salvador and Guatemala are largely responsible for the dense populations of whitefly in those

countries. It therefore seems likely that in the particular instance of bean golden mosaic, recent changes in agricultural emphasis and intensity have led to a situation greatly favoring the whitefly vector and thus the incidence of the disease.

The epidemiology of cassava mosaic disease in Kenya is of particular interest because, unlike the other diseases described, it has two distinct means of transmission and is a comparatively long-term crop, remaining in the ground for at least 12 months (compared with the 4-month annuals). The disease is transmitted by *Bemisia* but is also perpetuated by way of vegetative propagation of cassava. Farmers do not discriminate between mosaic-infected and mosaic-free cassava and use cuttings of either as planting material. This is hardly surprising, as about 80% of all plants in farmers' fields are usually infected.

By selecting, propagating, and multiplying only disease-free plants, it was possible to conduct a series of epidemiology studies in which the main interest lay in the rate of reinfection of mosaic-free plots (2). Surprisingly, the rate of reinfection is less than 2% per annum—and often less than 1%—irrespective of the size of the plot (0.02–3 ha or larger), irrespective of its location (on agricultural research stations or in the farmers' fields), and irrespective of climatic regions both in space and in time (coastal Kenya and western Kenya, 1974–1980). It seems likely, therefore, that in Kenya, at least, where the climate for extended periods of the year is inimical to the development of dense whitefly populations, the principal vector of mosaic is not the whitefly but man,

with his inadvertent use of infected cuttings. The situation with cassava mosaic disease epidemiology is probably different in other African regions where the climate is more favorable to the vector. However, the disease can be controlled in Kenya and probably in most other areas of eastern Africa by the simple expedient of introducing mosaic-free multiplication and distribution schemes.

Strategies for Control

In the tropical and subtropical countries where most of the geminiviruses seem to occur, insecticidal control of the vectors, assuming this to be possible, is more often than not precluded by economic considerations, and pathologists must resort to other strategies.

There are several obvious and well-known cultural practices that may be useful in reducing the amount of inoculum arriving into young crops. With bean golden mosaic virus the elimination of adjacent reservoirs of virus, such as volunteer hosts, has been recommended, together with avoidance of planting bean crops near earlier-sown crops, such as soybean or tobacco, that favor dramatic increases in whitefly populations (4). Time of planting has also been advocated as a means of escaping critical periods most favorable to the vector, as well as crop rotation and distribution within an area. The underlying principle of control in these instances is the knowledge that with many geminiviruses, inoculation efficiency is increased as population size of the vector is increased, and significant reduction in numbers may result in a significant decrease in disease incidence.

While these amelioratives are of

undoubted value, their stringent application in most tropical situations is unlikely. For example, there are many areas in Africa where, because of very unreliable rain distribution, it is essential to be an opportunist and to plant, when so dictated, successive plots of maize. As we have seen, this is an ideal situation for rapid development of epidemics of maize streak disease, but that chance clearly has to be taken. This is illustrative of the fact that, sooner or later, recourse must be made to field resistance or field tolerance, and this is being actively pursued in the case of several tropical geminiviruses.

Bean summer death disease has been controlled satisfactorily by the use of resistant cultivars, but no resistance to the related tobacco yellow dwarf virus has yet been found in commercial tobacco varieties. The solution to this problem was found only by avoiding high-risk disease situations, which necessitated relocating the tobacco-growing industry.

In spite of the evaluation of over 10,000 accessions of *Phaseolus vulgaris*, no source containing a high degree of genetic resistance or immunity to bean golden mosaic virus has yet been found (4). Acceptable degrees of tolerance, however, exist and these have been exploited with success. Apparently, when infected, tolerant varieties contain lower concentrations of virus than do susceptible cultivars.

An interesting situation exists in Central and South America with regard to the insistent local consumer preference for particular bean seed-coat colors, which must be taken into account in breeding and selection programs. Tolerance to bean golden mosaic has

Table 1. Geminiviruses and their vectors

| Virus | Vector | Conventional sap transmission | Susceptibility of: | | Geographic distribution |
|---|-----------------------------|----------------------------------|--------------------|------------------|--|
| | | | <i>Datura</i> | <i>Nicotiana</i> | |
| Transmitted by leafhoppers^a | | | | | |
| Maize streak | <i>Cicadulina</i> spp. | — | — | — | Africa, Mauritius, Réunion, Madagascar, India |
| Chloris striate mosaic | <i>Nesochutha obscura</i> | — | ... | ... | Australia |
| Wheat dwarf | <i>Psammotettix alienus</i> | — | ... | ... | Sweden |
| Beet curly top ^b | <i>Circulifer tenellus</i> | — | + | + | North America (Southwest) |
| Bean summer death ^b | <i>Orosius argentatus</i> | — | + | + | Australia |
| Tobacco yellow dwarf ^b | <i>O. argentatus</i> | — | + | + | Australia |
| Transmitted by whiteflies | | | | | |
| Bean golden mosaic | <i>Bemisia tabaci</i> | + | — | — | Central and South America, Caribbean area |
| Cassava latent | <i>B. tabaci</i> | + | + | + | Africa |
| Tobacco leaf curl ^c | <i>B. tabaci</i> | — | + | + | Japan |
| Honeysuckle yellow vein mosaic ^c | <i>B. tabaci</i> | — | + | + | Japan |
| Tomato yellow dwarf ^c | <i>B. tabaci</i> | — | + | + | Japan |
| <i>Eupatorium</i> yellow vein mosaic | <i>B. tabaci</i> | — | ... | ... | Japan |
| Tomato golden mosaic | <i>B. tabaci</i> | + | + | + | South America |
| <i>Euphorbia</i> mosaic | <i>B. tabaci</i> | + | + | + | South America |
| ?Tomato yellow leaf curl diseases | <i>B. tabaci</i> | — | (+) | (+) | Mediterranean basin, East Africa |

^a Cicadellidae, Deltocephalini tribe.

^b Serologically related.

^c Serologically related.



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been obtained in black-seeded beans preferred in certain areas of Central America. Other areas have a strong preference for mottled or red-seeded beans, and tolerance must be combined with these seed-coat colors to ensure a ready acceptability of the product. This situation has a parallel in some areas of Africa where white-seeded maize is exclusively grown and consumed in preference to yellow-seeded varieties. Unless disease resistance is transferred into white-seeded varieties, the product is unacceptable.

One known source of outstanding resistance to maize streak virus is contained in local maize from the western Indian Ocean island of Réunion, and it is possible to speculate on the origins of this resistance. Conditions on Réunion are most favorable for the vector and for the virus, both of which may be considered indigenous. The island was first settled in 1662, and the earliest and all subsequent importations of maize have been subjected to an intense and continuous natural selection pressure for resistance to maize streak disease. Highly susceptible plants that were killed or yielded little or

no grain were eliminated from the population, and resistance in the form of a very high degree of tolerance was selected for, in the absence of any conscious effort by the local farmers. This resistance seems to operate against all African mainland variants for maize streak virus and seems to be a classic example of horizontal resistance.

The situation with cassava mosaic disease in Kenya is broadly similar to that of maize in Réunion. Locally adapted Kenyan cassava varieties apparently contain a very useful level of field resistance. Measured as rate of spread of disease into mosaic-free plots, resistance is comparable to that of hybrid varieties deliberately bred and selected for resistance in eastern Africa. The local varieties also yield as well as the hybrids and have the added advantages of higher tolerance to drought and of being preferred by the farmers.

Cane streak, a disease of sugarcane caused by the sugarcane strain of maize streak virus, was of great economic significance in Natal between 1920 and 1945 because of the predominance of a highly susceptible sugarcane variety,

Uba. The replacement over the years of Uba with highly resistant sugarcane varieties provided a very effective solution to the problem, and the disease is now uncommon and unimportant. Nevertheless, all new clones must be screened for resistance to cane streak; about 2% of these are found to be susceptible each year and are consequently rejected. Cane streak disease is not widely known these days, but the geminivirus continues to demand attention.

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