Chapter 15

APHID-TRANSMITTED VIRUSES

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General Introduction

Various aphid-borne viruses infect beans and include bean common mosaic virus (BCMV), bean yellow mosaic virus (BYMV), cucumber mosaic virus (CMV), soybean mosaic virus (SMV), and alfalfa mosaic virus (AMV). This chapter will review the geographical distribution, economic importance, host range, physiochemical properties, purification, transmission, epidemiology, symptomatology, and control of these viruses.

Bean Common Mosaic Virus

Introduction

Bean common mosaic was one of the first virus diseases reported in the world when Iwanoski (1894) observed it in the Soviet Union. Since then the seed-borne virus has been reported in nearly every country of the world. It is economically important throughout Africa, Europe, North America, and Latin America (Cafati-K. and Alvarez-A., 1975; Costa et al., 1971; Crispín-Medina and Campos-Avila, 1976; Dean and Wilson, 1959; El-Shamy et al., 1972; Gámez, 1973; Hampton et al., 1983; Inouye, 1969; Joshi et al., 1981; Kaiser et al., 1968; Klesser, 1961; Kulkarni, 1973; Lockhart and Fischer, 1974; Moreno et al., 1968; Provvidenti et al., 1982; Schieber, 1970; Yerkes and Crispín-Medina, 1956; Zaumeyer and Thomas, 1957).

Plant infection may reach 100% in fields and yield losses range from 35% to 98% (Gálvez and Cárdenas-A., 1974; Hampton, 1975;

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Zaumeyer and Thomas, 1957). Hampton (1975) reported that pod number per plant was reduced 50%-64% and seed yield per plant was reduced 53%-68%, depending upon the virus strain. Gálvez and Cárdenas-A. (1974) reported that yield losses varied from 6% to 98%, depending upon the cultivar and time of infection.

The host range for BCMV is more limited than that reported for BYMV, but still includes common bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.), tepary bean (*P. acutifolius* var. *acutifolius*), *Vigna angularis* (Willd.) Ohwi *et* Ohasi, *V. aconitifolia* (Jacq.) Maréchal, *V. umbellata* (Thunb.) Ohwi *et* Ohashi, urd bean (*V. mungo* (L.) Hepper), scarlet runner bean (*P. coccineus* L.), siratro (*Macroptilium atropurpureum* (DC.) Urb.), *V. radiata* (L.) Wilczek var. *radiata*, *P. polyanthus* Greenman, *Vigna unguiculata* spp. *unguiculata* var. *sesquipedalis* (L.) Verdc., cowpea (*V. unguiculata* (L.) Walp. ssp. *unguiculata*), broad bean (*Vicia faba* L.), *Crotalaria spectabilis* Roth., *Canavalia ensiformis* (L.) DC., *Lupinus albus* L., *Nicotiana clevelandii*, *Macroptilium lathyroides* (L.) Urb., pea (*Pisum sativum* L.), alfalfa (*Medicago sativa* L.), *Lablab purpureus* (L.) Sweet, common clover (*Trifolium pratense* L.), and *Rhynchosia minima* (L.) DC. (Bos, 1971; Kaiser and Mossahebi, 1974; Kaiser et al., 1971; Meiners et al., 1978; Ordosgoitty, 1972; Zaumeyer and Thomas, 1957). *Sesbania exaltata* (Raf.) V.L. Cory and siratro (*Macroptilium atropurpureum* (DC.) Urb.) are reported as symptomless hosts (Meiners et al., 1978). R. O. Hampton (personal communication) has pointed out that additional research is needed to confirm that *Vicia faba* and *Vigna* species are true hosts, particularly with regard to seed transmission.

Chenopodium quinoa (Willd.), *Gomphrena globosa* L., *Tetragonia expansa* J. Murr., and cultivars of *Phaseolus vulgaris* serve as local-lesion indicators to various strains of BCMV (Alvarez-A. and Sepúlveda-R., 1982; Bos, 1971; Castaño-J. et al., 1982; Polak and Chod, 1972; Saettler and Trujillo, 1972; Schneider and Worley, 1962; Trujillo and Saettler, 1972a and 1973; Zaumeyer and Goth, 1963). In nature, however, BCMV is primarily restricted to *Phaseolus* spp., particularly *P. vulgaris*. It is possible that some susceptible hosts reported above were infected by serologically related viruses and not by BCMV strains.

Bean common mosaic virus was called bean virus 1 and *Marmor phaseoli* Holmes (Zaumeyer and Thomas, 1957). The name given to

bean common mosaic virus in Latin America is "mosaico común" in Spanish and "mosaico comum" in Portuguese.

Symptomatology

Bean common mosaic virus may incite three types of symptoms: mosaic, systemic necrosis (black root), or local lesions or malformations, depending upon the cultivar, time of infection, strain, and environmental conditions. Mosaic symptoms appear in systemically infected cultivars and may cause mottling, curling, stunting, and malformation of primary leaves (Figure 106), especially if primary infection occurred through infected seed. The trifoliolate leaves may exhibit leaf malformation and mosaic (Figure 107). Infected leaves may appear narrower and longer than uninfected leaves (Figure 108).

Systemically infected plants may have smaller and fewer pods than infected plants. Infected pods occasionally may be covered with small dark green spots and mature later than uninfected pods (Zaumeyer and Goth, 1964; Zaumeyer and Thomas, 1957).

Systemic necrosis (black root) symptoms may appear in cultivars having hypersensitive resistance (I gene) to systemic mosaic upon infection by necrosis-inducing strains, especially at high temperatures (26-32 °C). However, some necrosis-inducing strains are temperature independent (Drijfhout, 1978). The incidence of black root in Latin America is usually negligible but may reach 100% in Africa.

Black-root symptoms initially appear as a progressive vein necrosis (Figure 109) of the young trifoliolates which then die. The older leaves start to wilt and, eventually, the entire plant dies. Characteristic reddish brown to black streaks appear on the stems, roots, and pods (Figure 110). The entire vascular system soon becomes necrotic (Figure 111) (Drijfhout, 1978; Hubbeling, 1972; Zaumeyer and Thomas, 1957).

Local lesions may appear on the leaves of some cultivars. These lesions may be induced by mechanical inoculation or aphid transmission. They manifest as reddish to dark brown necrotic ring-shaped lesions or spots (Figure 112), depending upon the

cultivar, strain, and environmental conditions. Cultivars which are known local-lesion hosts include Great Northern U.I. 31 and 123, Pinto U.I. 111, Potomac, Stringless Green Refugee, Plentiful, and Monroe (Polak and Chod, 1972; Saettler and Trujillo, 1972; Schneider and Worley, 1962; Trujillo and Saettler, 1972a, 1972b, and 1973; Zaumeyer and Goth, 1963).

Physical properties

Bean common mosaic virus particles can be observed easily with the electron microscope in crude sap or partially purified preparations. The filamentous flexuous virus particles are 730-750 nm in length and 12-15 nm in width (de Camargo et al., 1968; Morales, 1979). Cytoplasmic inclusions are also induced by the virus and readily appear in the light or electron microscope as cylindrical pinwheels (Figure 113) (de Camargo et al., 1968; Hoch and Provvidenti, 1978; Valdés et al., 1982). Virus particles are transported throughout the phloem. They can be detected in upper plant parts within 24-48 hours and in the root system within 60 hours after inoculation (Ekpo and Saettler, 1974 and 1975).

Bean common mosaic virus particles are inactivated in sap at 56-65 ^{0}C, have a dilution end point of 10^{-3} to 10^{-4}, and are infectious for one to four days (Bos, 1971; Gámez, 1973).

Morales (1979) developed a purification method which isolates BCMV with a high degree of purity and in adequate amounts to produce a specific antiserum.

Epidemiology

Bean common mosaic virus can be transmitted mechanically, in pollen and seed, and by insect vectors. BCMV-infected leaves, used as inoculum, can be homogenized in water or buffers such as potassium phosphate, and then manually applied to leaves of healthy susceptible plants (Morales, 1979). Many workers have also added abrasives such as Carborundum powder to inoculum to help introduce virus particles into plant cells (Cafati-K., 1968; Zaumeyer and Thomas, 1957).

An inoculation efficiency of nearly 100% can be achieved in the greenhouse, while in the field efficiency is lower because adverse environmental factors affect both viruses and plants.

Virus particles can be transmitted in pollen grains, ovules, and flowers of infected plants (Ekpo and Saettler, 1974; Wilson and Dean, 1964; Zaumeyer and Thomas, 1957). Seed transmission likewise can occur in susceptible cultivars of *Phaseolus vulgaris*, *P. acutifolius*, *P. coccineus*, *P. polyanthus*, *Macroptilium lathyroides*, *Rhynchosia minima*, and in *Vigna* species (Kaiser and Mossahebi, 1974; Meiners et al., 1978; Noble and Richardson, 1968; Phatak, 1974; Provvidenti and Braverman, 1976; Provvidenti and Cobb, 1975; Robertson, 1962; Skotland and Burke, 1961). The percentage of seed transmission varies from 3% to 95%, according to cultivar and time of infection, especially before flowering (Alconero and Meiners, 1974; Alvarez-A., 1977; Crispín-Medina and Grogan, 1961; Gálvez and Cárdenas-A. 1974; Gálvez et al., 1977; Kulkarni, 1973; Montenegro-B. and Galindo-A., 1974; Ordosgoitty, 1972; Schippers, 1963; Zaumeyer and Thomas, 1957). BCMV particles are reported to survive in bean seed for at least 30 years (Zaumeyer and Thomas, 1957).

Insect vectors such as aphids (Figure 114) can transmit BCMV effectively from infected plants to healthy plants. Reported aphid vectors include *Macrosiphum solanifolii* (Ashmead), *M. pisi* (Kalt.), *M. ambrosiae* (Thomas), *Myzus persicae* (Sulzer), *Aphis rumicis* L., *A. gossypii* Glover, *A. medicaginis* Koch, *Hyalopterus atriplicis*, and *Rhopalosiphum pseudobrassicae* Davis (Zaumeyer and Thomas, 1957; Zettler and Wilkinson, 1966). Studies have determined that aphid populations are often lower than those of other insect species in bean fields, but that the aphids are responsible for transmission of BCMV. The efficiency of transmission depends upon the source of inoculum, but usually virus acquisition and transmission (Zettler, 1969) occurs within one minute.

In the tropics and other regions, infected seeds and plants of susceptible bean cultivars serve as sources of primary inoculum for BCMV (Hampton, 1967; Robertson and Klostermeyer, 1961 and 1962). Aphids are responsible for the secondary transmission of the virus. In Colombia, CIAT studies determined that relatively high aphid populations were able to incite 100% plant infection from a seed source that was only 2%-6% infected.

Control by cultural practices

Various cultural practices such as planting date and clean-seed production, minimize BCMV incidence in susceptible cultivars. Burke (1964) found a correlation between planting date and virus incidence which was associated with aphid population levels. Bean plantings, therefore, must be adjusted to minimize the period during which susceptible cultivars are exposed to infection by aphids migrating from other crops to beans during the growing season.

Planting BCMV-free seed can effectively reduce the initial inoculum. However, to reduce transmission of BCMV from other infected bean plants or weed hosts, it may also be necessary to control aphids with insecticides (Sánchez and Pinchinat, 1974). No chemicals or other treatments are available to remove or destroy BCMV particles present within infected seed (Zaumeyer and Thomas, 1957).

Control by plant resistance

Plant resistance to bean common mosaic virus has been available for nearly 60 years after the cultivar Robust was discovered to be resistant. The resistance of Robust is conferred by a single recessive gene (Baggett et al., 1966; Cafati-K. and Alvarez-A., 1975; Guerra et al., 1971; Hernández-Bravo and Gálvez, 1976; Zaumeyer and Thomas, 1957). Cultivars that were subsequently developed, having Robust resistance, include Great Northern U.I. 1, 59, 81, and 123; Red Mexican U.I. 3 and 34; Royal Red; and Pinto U.I. 72, 78, and 111 (Burke et al., 1969; Smith, 1962a and 1962b; Zaumeyer and Thomas, 1957). These cultivars have been resistant to the type strain of BCMV for more than 50 years (Zaumeyer and Meiners, 1975).

Nearly 50 years ago another source of resistance was identified in Corbett Refugee. This resistance is conferred by a dominant hypersensitive gene which conditions the black-root reaction. The majority of snap bean cultivars and some of the common bean cultivars developed in United States have derived their resistance from Corbett Refugee. They include Wisconsin Refugee, Idaho Refugee, and Refugee U.S. 5 (Zaumeyer and Thomas, 1957). This resistance has been effective for nearly 50 years. Burke and

Silbernagel (1974) and van Rheenen and Muigai (1984) have suggested that the Corbett Refugee type of resistance be widely incorporated into commercial cultivars.

These sources of resistance also have been used to develop resistant cultivars in Latin America such as ICA Tui and ICA Pijao in Colombia, Titán in Chile, Peru 257 in Peru, Tacarigua in Venezuela, and Jamapa and Sataya 425 in Mexico (Cafati-K. and Alvarez-A., 1975; Drijfhout, 1978; Montenegro-B. and Galindo-A., 1974; Ortega-Y. and Barrios-G., 1972; Trujillo and Saettler, 1972b; Ziver-M. and Cafati-K., 1968).

Hagel et al. (1972) have reported that certain BCMV-resistant cultivars such as Black Turtle Soup, also express tolerance to insect vectors such as aphids. Additional studies are necessary to determine the effectiveness of this type of aphid resistance and its application to commercial production.

Plant resistance to BCMV is affected by the nature of the gene(s) conferring resistance, variability between virus strains, and environmental conditions. Various workers have investigated the relationships between different virus strains and sources of resistance (Alvarez-A., 1977; Alvarez-A. and Ziver-M., 1965; Bercks, 1960; Drijfhout, 1978; Drijfhout and Bos, 1977; Drijfhout et al., 1978; Innes and Walkey, 1980; Silbernagel, 1969). Drijfhout (1978) assigned 22 cultivars to 11 resistance groups and divided the 15 known viral strains in seven pathogenicity groups (Table 1).

Cultivars in resistance groups one to six do not express systemic necrosis to any viral strains. However, they do express systemic mosaic symptoms to one or more of the BCMV strains. These cultivars have recessive genes only. The experimental line IVT 7214 (resistance group 7) does not exhibit systemic mosaic nor necrosis upon inoculation with any known viral strain. It possesses a recessive gene bc 3 which is effective against all known strains at this time. Cultivars in resistance groups 8 to 10 may exhibit only systemic necrosis to one or more of the necrosis-inducing strains of BCMV. These cultivars, therefore, have the dominant I gene. The IVT 7233 line has the dominant I gene, together with a recessive gene of cultivar group 6 which protects against systemic necrosis. This line exhibits only local necrotic lesions when inoculated with a

Table 1. Differentiation and grouping of BCMV strains and host resistance groups.

Host resistance group	Differential cultivar name	Pathogenicity group of the virus														
		I			II	III	IVa	IVb			Va	Vb	VIa	VIb	VII	
		Westlandia NL 1	Type US 1	Puerto Rico PR 1	NL 7	NL 8	Florida US 5	Western US 4	Idaho or B US 3	Colana NL 6	NY 15 US 2	Imuna NL 2	Michelite NL 3	Jolanda NL 5	Mexico US 6	Great North. NL 4
Cultivars with recessive alleles (I*I*) of the necrosis gene																
1	Dubbele Witte	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
	Str. Gr. Ref	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
2	Redl. Gr. C	—	—	—	+	—	+	+	+	+	+t	+	+t	+	+	+
	Puregold Wax	—	—	—	+	—	+	+	+	+	+t	+	+t	+	+	+
	Imuna	—	—	—	+t	—	+	+	+	+	+t	+	+t	+	+	+
3	Redl. Gr. B	—	—	—	—	—	+	+	+	+	—	—	+	+	+	+
	Gr. North. 123	—	—	—	—	—	+	+	+	+	—	—	+t	+t	+	+
4	Sanilac	—	—	—	—	+	—	—	—	—	+	+	+	+	—	—
	Michelite 62	—	—	—	—	+	—	—	—	—	+	+	+	+	—	—
	Red Mex. 34	—	—	—	—	+	—	—	—	+	+	+	+	+	—	—
5	Pinto 114	—	—	—	—	—	—	—	—	—	+	+	+	+	—	—
6	Monroe	—	—	—	—	—	—	—	—	—	—	—	—	—	+	+
	Gr. North. 31	—	—	—	—	—	—	—	—	—	—	—	—	—	+	+
	Red. Mex. 35	—	—	—	—	—	—	—	—	—	—	—	—	—	+	+
7	IVT 7214	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

(Continued)

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		I			II	III	IVa	IVb			Va	Vb	VIa	VIb	VII	
		Westlandia NL 1	Type US 1	Puerto Rico PR 1	NL 7	NL 8	Florida US 5	Western US 4	Idaho or B US 3	Colana NL 6	NY 15 US 2	Imuna NL 2	Michelite NL 3	Jolanda NL 5	Mexico US 6	Great North. NL 4
Cultivars with dominant alleles (II) of the necrosis gene																
8	Widusa	—	—	—	—	+n	—	±n	±n	±n	—	—	+n	+n	—	—
	Bl. Turtle S.I	—	—	—	—	+n	—	±n	±n	±n	—	—	+n	+n	—	—
9a	Jubila	—	—	—	—	—	—	+n	+n	+n	—	±n	+n	+n	—	—
9b	Top Crop	—	—	—	—	—	—	±n	±n	±n	—	±n	+n	+n	—	—
	Imp. Tendergr.	—	—	—	—	—	—	±n	±n	±n	—	±n	+n	+n	—	—
10	Amanda	—	—	—	—	—	—	—	—	—	—	—	—	+n	—	—
11	IVT 7233	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

\+ Susceptible, sensitive, systemic mosaic.
+t Susceptible, tolerant, systemic symptoms questionable or very weak, virus recovered from uninoculated leaves by back-inoculation onto Dubbele Witte.
— Resistant, no systemic symptoms, virus not recovered from uninoculated leaves by back-inoculation.
+n Susceptible, sensitive, usually all plants with systemic necrosis, not clearly dependent on temperature.
±n Susceptible or resistant, dependent on temperature, from none to all but mostly only a few plants with systemic necrosis, the number varying in repeated tests and increasing with temperature. Greenhouse mean temperature 22-26 °C, day and night fluctuation at most 20-24 °C in winter and 20-30 °C in summer.

SOURCES: Drijfhout, 1978; Drijfhout et al., 1978.

necrotic BCMV strain. These genes have been successfully incorporated to produce mosaic and black-root resistant, commercial cultivars (Drijfhout, 1978).

Bean Yellow Mosaic Virus

Introduction

Bean yellow mosaic virus (BYMV) is widely distributed throughout the world. However, it usually occurs in legumes other than beans. The virus occurs in North America, Europe, East Africa, Japan (Bos, 1970; Inouye, 1969, Vanderveken, 1963; Zaumeyer and Thomas, 1957), Chile (Cafati-K. et al., 1976), Argentina (von der Phalen, 1962), Brazil (Costa et al., 1971; Kitajima and Costa, 1974), Uruguay, and possibly northern Mexico.

BYMV infected up to 100% of the plants grown in a field in United States (Zaumeyer and Thomas, 1957). Hampton (1975) reported that BYMV could cause serious yield losses with a 33% and 41% reduction in pod number and seed yield, respectively.

Bean yellow mosaic virus has been called Phaseolus virus 2, Gladiolus mosaic virus, pea mosaic virus, and bean virus 2 by earlier workers (Zaumeyer and Thomas, 1957). Common names for BYMV in Latin America include "mosaico amarillo" and "moteado amarillo" in Spanish, and "mosaico amarelo" in Portuguese.

Bean yellow mosaic virus strains have a wide host range which includes common bean (*Phaseolus vulgaris*), mung bean (*Vigna radiata* var. *radiata*), lima bean (*P. lunatus*), pigeonpea (*Cajanus cajan* (L.) Millsp.), chickpea (*Cicer arietinum* L.), sweet pea (*Lathyrus odoratus* L.), lentil (*Lens culinaris* Med.), *Melilotus albus* Med., *Cucurbita sativum*, pea (*Pisum sativum*), broad bean (*Vicia faba*), *V. americana*, *V. monanthos* Desf., hairy vetch (*V. villosa* Roth.), *V. sativa* L., *V. atropurpurea* Desf., *Vigna unguiculata* ssp. *unguiculata* var. *sesquipedalis*, cowpea (*Vigna unguiculata* ssp. *unguiculata*), common clover (*Trifolium pratense*) *T. incarnatum* L., *T. hybridum* L., alfalfa (*Medicago sativa*), *M. lupulina* L., soybean (*Glycine max* (L.) Merrill), *Gladiolus* spp., *Trigonella foenum-graecum* L., *Crotalaria spectabilis*, *Lupinus*

densiflorus Benth., *Proboscidea jussieui* J.C. Keller, *Cladrastis lutea* (Michx. f.) C. Koch, *Robinia pseudoacacia* L., *Freesia* Eckl. ex Klatt sp., *Babiana* Ker-Gawl sp., *Ixia* L. sp., *Sparaxis* Ker-Gawl sp., *Tritonia* Ker-Gawl sp., *Viola* L. sp., tobacco (*Nicotiana tabacum* L.), *N. sylvestris* Speg. *et* Comes, and *N. rustica* L. (Bos, 1970; Jones and Diachun, 1977; Provvidenti and Hunter, 1975; Provvidenti and Schroeder, 1972; Zaumeyer and Thomas, 1957; Zettler and Abo-El-Nil, 1977). Not all BYMV strains infect or induce symptoms in these hosts.

Symptomatology

BYMV-induced infection and symptoms vary considerably, depending on the strain, host, environmental conditions, and time of infection. Initial symptoms of BYMV systemic infection appear as small chlorotic spots which gradually enlarge and coalesce to produce a general chlorosis on affected leaves (Figure 115). Young leaves may become malformed (Figure 116). Yellow and green mottling becomes more intense on leaves as they age. Infection causes shortened internodes, proliferation of branches, epinasty, and plant stunting. It also may delay maturity (Zaumeyer and Thomas, 1957).

Systemic necrosis symptoms can be induced by specific strains of BYMV. Other BYMV strains are able to incite local necrotic lesions on leaves. The typical chlorotic leaf symptoms also may be present (Cafati-K. et al., 1976; Zaumeyer and Thomas, 1957). Epinasty and early plant death may also occur (Tatchell et al., 1985). Reddish brown spots may form on infected pods which can be malformed, depending upon the specific virus strain (Zaumeyer and Thomas, 1957).

Physical properties and purification

Particles of BYMV are indistinguishable from those of BCMV because they belong to the same virus group. BYMV particles are flexuous rods (Figure 117), 750 nm in length and 15 nm in width (Varma et al., 1968). BYMV induces crystalline inclusions in both cytoplasm and nuclei; the cytoplasmic cylindrical inclusions, or

pinwheels, are typical of the potyvirus group (Bos, 1969 and 1970; de Camargo et al., 1968; Inouye, 1973; Kitajima and Costa, 1974; Tapio, 1972) (Figure 113).

Bean yellow mosaic virus has a thermal inactivation point between 50 and 60 °C and a dilution end point between 10^{-3} and 10^{-4}. Particles retain their infectiousness for one to two days and occasionally up to seven days in sap at room temperature. These properties depend upon the virus source, host plant, and experimental conditions (Bos, 1970; Musil et al., 1975; Zaumeyer and Thomas, 1957).

Purification of BYMV was difficult in early work because particles aggregate easily and also agglutinate to plant chloroplasts. Various workers have developed methods to partially purify BYMV (Bancroft and Kaesberg, 1959; Huttinga, 1973; Huttinga and Mosch, 1974). Morales (1979) developed a procedure which yields highly purified and yet natural BYMV preparations. Jones and Diachun (1977) also developed a reliable purification procedure.

Bean yellow mosaic virus and its various strains are serologically distinguishable (Beczner et al., 1976; Bercks, 1960 and 1961; Bos, 1970; Bos et al., 1974; Granett and Provvidenti, 1975; Jones and Diachun, 1977; Musil et al., 1975; Uyemoto et al., 1972; Zaumeyer and Thomas, 1957). Jones and Diachun (1977) identified three BYMV subgroups within a collection of BYMV isolates obtained from infected red-and-white clover. These subgroups differ for serological and biological factors such as host range and symptoms. Additional work is required to establish an acceptable set of host differentials and strain classification.

Epidemiology

Bean yellow mosaic virus is easily transmitted mechanically and by aphids, but it is not transmitted in the seed of *P. vulgaris*. However, it can have a low transmission in the seed of *Vicia faba* and other legumes (Bos, 1970).

Aphid vectors include *Acyrthosiphon pisum* (Harris), *Macrosiphum euphorbiae* (Thomas), *Myzus persicae*, and *Aphis fabae* Scopoli (Bos, 1970; Grylls, 1972; Hagel and Hampton, 1970; Sohi,

1964; Swenson and Welton, 1966; Thottappilly et al., 1972). Aphid transmission from infected beans or other hosts is primarily responsible for natural epidemics of BYMV. Some strains of BYMV are not easily transmitted by aphids (Evans and Zettler, 1970; Sohi, 1964; Thottappilly et al., 1972). Some BYMV strains may lose aphid transmissibility during storage or maintenance by mechanical inoculation.

Control

Plant resistance is the most reliable control measure available (Zaumeyer and Meiners, 1975). Resistance to specific strains is conditioned by plant genes such as By-2 (Dickson and Natti, 1968; Schroeder and Provvidenti, 1968). Sources of resistance to the BYMV strain inducing pod malformation have been identified in various Great Northern lines such as G.N. U.I. 31, 59, 123, and 1140. This resistance is conferred by three recessive genes with modifiers (Baggett, 1957; Baggett and Frazier, 1957; Cafati-K. et al., 1976; Guglielmetti, 1974; Provvidenti and Schroeder, 1973; Zaumeyer and Meiners, 1975). G.N. U.I. 31 also contains two recessive genes for resistance to the severe strain. Breeding for combined resistance to type and severe strains is best done by testing large F_2 populations with one strain, followed by testing progeny with the alternate strain (Tatchell et al., 1985). Resistance to BYMV strains has been found in interspecific crosses between *Phaseolus vulgaris* and *P. coccineus* (Baggett, 1956; Baggett et al., 1966; Zaumeyer and Thomas, 1957).

Cucumber Mosaic Virus

Introduction

Cucumber mosaic virus (CMV) is widely distributed throughout the world (Bird et al., 1974; Bos and Maat, 1974; Jayasinghe, 1982; Marchoux et al., 1977; Meiners et al., 1977; Milbrath et al., 1975; Zaumeyer and Thomas, 1957), affecting over 750 susceptible species in more than 80 plant families (Doine et al., 1979; Price, 1940). *Phaseolus vulgaris* is naturally infected by CMV and some commercial plantings have been noticeably affected by this virus (Bird et

al., 1975; Bos and Maat, 1974; Marchoux et al., 1977; Provvidenti, 1976; Whipple and Walker, 1941). No cultivar or germplasm accession is immune, although good levels of tolerance exist.

Cucumber mosaic virus has been called cucumber virus 1, *Cucumis* virus 1, *Marmor cucumeris*, spinach blight virus, and tomato fern leaf virus. The common name frequently used for CMV in Latin America is "virus del mosaico del pepino."

Cucumber mosaic virus can be propagated in *Nicotiana* species such as *N. clevelandii*, and assayed in local-lesion hosts such as cowpea (*Vigna unguiculata* ssp. *unguiculata*), *Chenopodium amaranticolor* Coste *et* Reynier, and *C. quinoa* (Francki et al., 1979).

Symptomatology

Symptoms of CMV infection may consist of a mild mosaic, vein clearing, vein banding, leaf rolling or distortion, epinasty, and/or apical necrosis. Both local and systemic symptoms are usually observed in *P. vulgaris* (Jayasinghe, 1982). The intensity of symptom expression may vary, depending upon the cultivar, strain, and time of infection. Symptoms may become less noticeable in older tissue if infection occurred in very young plants. Pod distortion may also occur (Bird et al., 1974 and 1975; Milbrath et al., 1975; Provvidenti, 1976).

Physical properties

Cucumber mosaic virus is the type strain of the cucumovirus group whose isometric particles (about 28 nm in diameter) encapsidate three functional molecules of single-stranded RNA (Francki et al., 1979). CMV has a thermal inactivation point of 70 ^{0}C, a dilution end point between 10^{-4} and 10^{-5}, and is infectious in vitro for three to six days at 23 ^{0}C (Milbrath et al., 1975).

Various purification procedures have been developed (Bock et al., 1975; Bos and Maat, 1974; Francki et al., 1979; Gibbs and Harrison, 1970; Meiners et al., 1977; Murant, 1965; Scott, 1963). These procedures have enabled researchers to develop antisera to study CMV and its strains.

Transmission

Cucumber mosaic virus is transmitted mechanically, in seed, and by insect vectors such as aphids. It can be transmitted mechanically from infected beans, tobacco, cucumbers (Figure 118), and other hosts (Bird et al., 1974; Marchoux et al., 1977; Meiners et al., 1977). Seed transmission varies from less than 1% to 40%, depending upon the bean cultivar (Bird et al., 1974; Bos and Maat, 1974; Jayasinghe, 1982; Marchoux et al., 1977; Meiners et al., 1977; Provvidenti, 1976). Bos and Maat (1974) reported that CMV retained its infectiousness in stored bean seeds for 27 months.

More than 60 species of aphids may transmit CMV. They include *Aphis gossypii* and *Myzus persicae* (Meiners et al., 1977; Provvidenti, 1976). Meiners et al. (1977) report that aphids retained CMV for as long as 40 minutes after a 10-minute accession feeding period.

Control

Control measures include planting seed free of CMV and crop rotation to reduce the number of hosts for the virus and its insect vector. Chemical control may be used to reduce aphid populations in other host crops. Bean cultivars differ in their resistance, but none are highly resistant.

Soybean Mosaic Virus

The rapid expansion of soybean plantings in traditional common-bean-producing areas has increased the frequency of soybean mosaic virus infection of susceptible bean cultivars (Costa et al., 1978; Provvidenti et al., 1982).

Soybean mosaic virus is another potyvirus widely distributed because it is easily transmitted by seed and aphids (Bos, 1972). Bean cultivars can be systemically infected, showing local lesions only or systemic mosaic or necrosis. Black-seeded cultivars usually exhibit local or systemic hypersensitivity (Costa et al., 1978). Systemic symptoms in beans are usually more severe than those induced by bean common mosaic virus.

Soybean mosaic virus is mechanically transmissible and can be transmitted by several aphid species, notably *Acyrthosiphon pisum*, *Aphis fabae*, and *Myzus persicae*. The thermal inactivation point is between 55-60 ^{0}C, its dilution end point around 10^{-3}, and sap may still be infectious after three days at room temperature (Bos, 1972). The virus can be seed-transmitted in *Phaseolus vulgaris* (Castaño-J. and Morales, 1983; Provvidenti et al., 1982).

Soybean mosaic virus is best propagated in susceptible soybean (*Glycine max*) cultivars. It can be isolated by using the purification methods used for bean common or yellow mosaic viruses. Some bean cultivars such as Top Crop and Monroe, are local-lesion assay hosts (Castaño-J. et al., 1982).

Because of the lack of information on the present distribution and incidence of SMV in the main bean-growing areas, the epidemiology and control of this virus have not been investigated. However, genetic resistance will be the main control measure in the future, using the resistant bean genotypes identified so far (Costa et al., 1978; Provvidenti et al., 1982).

Alfalfa Mosaic Virus

Alfalfa mosaic virus (AMV) is an aphid-transmitted virus that was first detected on beans in United States (Zaumeyer and Thomas, 1957). The virus consists of various strains, including yellow dot, alfalfa yellow mosaic, vein necrosis, and spot mosaic (Zaumeyer, 1963; Zaumeyer and Goth, 1963; Zaumeyer and Patiño, 1960; Zaumeyer and Thomas, 1957). None of these strains of AMV is economically important (Zaumeyer and Thomas, 1957).

Alfalfa mosaic virus has been known as lucerne mosaic virus, alfalfa virus 1, alfalfa virus 2, Medicago virus 2, and *Marmor medicaginis* Holmes (Bos and Jaspars, 1971; Zaumeyer and Thomas, 1957). Although it occurs on other legumes, alfalfa mosaic virus has not been found on beans in Latin America. In Spanish, the virus and its strains are called "mosaico de la alfalfa," "punto amarillo," "mosaico amarillo de la alfalfa," "necrosis venal," "mosaico de la mancha," and "calico."

The virus and its strains produce a systemic mottling of leaves, necrosis of leaves or stems, and dieback of the growing point (Costa et al., 1971b). However, the most common symptom consists of local necrotic lesions which have a diameter of 0.5-3.0 mm (Zaumeyer and Thomas, 1957).

The alfalfa mosaic virus is transmitted mechanically, but apparently not in bean seed. However, it is transmitted in the seed of alfalfa (6%) and pepper (1%-5%). The virus is a bacilliform, multicomponent RNA virus (Bos and Jaspars, 1971).

Because AMV is not an economically important virus disease of beans, there are no specific control measures.

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