STUDIES ON THE MOSAIC DISEASE OF SNAKE GOURD (Trichosanthes anguina L.)

By

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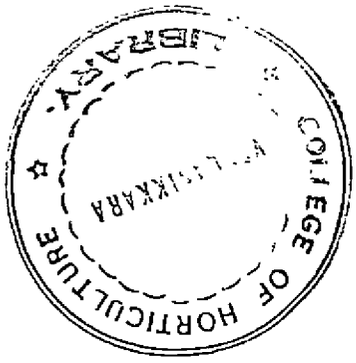

THESIS

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D E C L A R A T I O N

I hereby declare that this thesis entitled "STUDIES ON THE MOSAIC DISEASE OF SNAKE GOURD (*Trichosanthes anguina* L.)" is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship, or other similar title, of any other University or Society.

Vellayani,

January, 1978.

P. J. J O S E P H

C E R T I F I C A T E

Certified that this thesis, entitled "Studies on the mosaic disease of snake gourd (*Trichosanthes anguina* L.)" is a record of research work done independently by Shri. P.J. J O S E P H, under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

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C O N T E N T S

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INTRODUCTION

I N T R O D U C T I O N

Snake gourd (*Trichosanthes anguina* L.) is one of the major Cucurbitaceous vegetable crops of Kerala State and is extensively cultivated in summer months. It is also an important vegetable crop cultivated throughout India.

A severe disease on snake gourd characterised by mosaic like symptoms was observed in the Instructional Farm, Vellayani and adjoining localities. Further survey revealed that the disease is prevelant throughout Kerala State. The infected plants blossom sparingly and set only very few fruits, thus causing severe reduction in yield.

A perusal of the literature revealed that mosaic disease of snake gourd has been reported from Kerala (Pillai, 1971) caused by a strain of CMV and at Delhi (Dubey *et al.*, 1974) caused by *Cucumis Virus* 1.

Other viruses infecting snake gourd include *Cucumis Virus* 2B (Verma *et al.*, 1970), Melon mosaic virus (Jaganathan and Ramakrishnan, 1971) Watermelon mosaic virus (Nagarajan and Ramakrishnan, 1971 b) and Pumpkin mosaic virus (Shankar *et al.*, 1972). Since different viruses infecting snake gourd have been reported by various

workers from different parts of India, an attempt was made to identify and characterise the virus causing mosaic disease of snake gourd prevalent in Kerala. The information will also be useful in formulating measures of control of the virus. The nature and extent of loss due to the disease is not definitely known. In view of the serious nature and widespread occurrence of the disease the following aspects were investigated.

1. Symptomatology.
2. Transmission studies.
3. Physical properties of the virus.
4. Host range of the virus.
5. Estimation of loss.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Cucurbit plant was observed to be affected by mosaic disease in Ohio, Massachusetts and Connecticut during the first decade of this century, but delineation of a distinct infectious disease was not recorded until 1916. The first thorough comprehensive study of cucumber mosaic was carried out by Doolittle (1920). Since then a number of different virus diseases of Cucurbitaceous crops have been reported from various parts of the world.

Bewley (1923) reported the incidence of cucumber mosaic in Britain. He demonstrated two types: one, characterised by yellow patches and the other marked by mottling and blistering of leaves and stunting of the plants.

Walker (1933) was the first to record the spontaneous occurrence of a mosaic disease on water melon in Florida. The affected plants were stunted and the leaves were mottled accompanied with severe malformations of floral parts and mottling and distortion of fruits. The affected plants produced a 'Petunia' like appearance.

Ainsworth (1934; 1935) named the cucumber mosaic viruses as common cucumber mosaic virus, cucumber yellow mosaic virus and cucumber yellow mottle mosaic virus.

The occurrence of CMV was first suspected in India by Uppal (1934). Vasudeva and Lal (1943) reported that bottle gourd (Lagenaria vulgaris) was affected by a systemic mosaic disease. The symptoms were chlorotic streaks and dark green blisters appearing as small convex areas on the upper leaf surface and wavy with irregular outlines. Plants affected in the early stages remain stunted, sparsely flower and set few fruits. The virus was identified as Cucumis Virus 3.

Vasudeva and Pavgi (1945) first reported a Water melon mosaic virus from contaminated melon seeds. Later, Capoor and Varma (1948) studied bottle gourd mosaic virus and identified as Cucumis Virus 2 C.

Vasudeva et al. (1949) reported the virus disease of bottle gourd which caused general stunting and reduction in flower and fruit production. Young leaves exhibited dark green blisters on a crinkled pale green surface and identified as Cucumis Virus 2 C.

Bhargava (1951) isolated four strains of cucumber mosaic virus, a spinach strain which caused severe chlorosis and isolated from local lesion in tobacco leaf; a primula strain obtained from Primula obconica and two strains derived from a naturally infected turnip, one of which was a derivative of Price's yellow strain.

Bhargava and Joshi (1960) detected Water melon mosaic virus from vegetable marrow in U.P., with mosaic symptoms which differed from other previously described viruses.

Joshi (1962) identified a strain of Water melon mosaic virus infecting vegetable marrow and a number of other Cucurbitaceous crops.

Reddy and Mariani (1963) reported the occurrence of CMV on vegetable marrow (Cucurbita pepo) and identified that there are 3 types of viruses, a mosaic type, filiform type and witches' broom type. Vegetable marrow infected with mosaic type virus showed typical mosaic pattern of light and deep green areas on leaf and slight reduction in leaf size. In filiform type, the symptoms were distortion of lamina, filiformy of the leaves, vein-clearing in younger leaves, and development of dark green blisters on older leaves. Flowering was delayed, size of flowers was reduced and in severe cases there will not be any normal setting of fruits. Witches' broom type was seen in the later stages and characterised by a dense tuft of irregularly bent, stunted branches producing severely reduced and malformed leaves. The petioles and internodes were very severely reduced resulting in a witches' broom like appearance. The mosaic

type was identified as Cucumis Virus 1 and filiform type as Melon Mosaic Virus.

Hariharasubramanian and Badami (1964) reported the widespread occurrence of a mosaic virus disease on pumpkin, causing severe blistering, distortion and reduction in size of leaves. He concluded that the causal virus resembled the bottle gourd mosaic virus and filiform type of vegetable marrow mosaic.

Mitra and Nariani (1965) reported a mosaic disease in Tori (Luffa acutangula) which is characterised by light and dark green mosaic mottling, downward curling of leaf margins and general stunting in the plant growth. The causal virus was identified as Cucumis Virus 3.

Janardhan et al. (1969) studied a mosaic disease of bottle gourd from Mysore State and reported that the causal virus though resembled Cucumis Virus 2 B in symptomatology and certain other characteristics, it was assumed to be a new virus or a new strain of Lagenaria vulgaris virus.

Shankar et al. (1969) identified a mosaic disease of snake gourd which is characterised by a mosaic pattern of irregular dark green and yellow chlorotic patches on the lamina. The affected plants were stunted, produced fewer

flowers and showed leaf crinkling. The causal virus was identified as Cucumis Virus 1.

Verma et al. (1970) investigated the virus disease of snake gourd, bottle gourd and pumpkin in U.P. The leaves of the infected snake gourd were variously malformed, reduced in size and showed dark green mosaic mottling symptoms. In bottle gourd, leaves showed mosaic mottling and blistering symptoms. Leaves were smaller and deformed and plants stunted. Pumpkin showed mosaic mottling, blistering and deformity in leaves. The viruses of snake gourd and bottle gourd were identified as Cucumis Virus 2 B, and that of pumpkin as Cucumis Virus 1.

Jaganathan and Ramakrishnan (1971) isolated viruses from musk melon and pumpkin and identified as strains of Melon Mosaic virus.

Nagarajan and Ramakrishnan (1971 b) reported the occurrence of Water melon mosaic virus on snake gourd. The plants were stunted and leaves were affected by prominent mosaic mottling with considerable reduction in leaf size. In advanced stages, the leaves were crowded together to give a bushy appearance. When young plants were infected, considerable malformation were seen in leaves. The outer edge of lamina became serrated. In severely infected plants, the

leaves showed blistering and inward cupping of leaf margins. The symptoms appeared within 7 to 9 days. The causal virus was identified as Water Melon Mosaic virus.

Pillai (1971) was the first to record a mosaic disease of snake gourd in Kerala. Symptoms were a distinct mosaic of leaves with crinkling and reduction in leaf size. Affected plants were stunted and produced fewer flowers and fruits. The causal virus was reported to be due to a strain of CMV, probably by the spinach strain (Bhargava, 1951).

Goel and Varma (1973) isolated a mosaic disease of ridge gourd (Luffa acutangula) from Haryana. The causal virus was identified as a new strain of CMV designated as luffa strain.

Dubey et al. (1974) identified the snake gourd mosaic virus in New Delhi, the symptoms included mosaic mottling accompanied by chlorosis, vein banding and blistering of leaf lamina. Diseased plants produced very few branches and plants affected in the early stages blossom sparingly and set few fruits. The causal virus was identified as Cucumis Virus 1.

HISTOPATHOLOGY

Gigante (1934) reported the occurrence of a mosaic disease on vegetable marrow in Rome. The chlorotic areas

of the infected leaves were thinner and almost all the palisade cells were isodimetric with few or no chloroplast. The mesophyll cells were closely packed with much reduced intracellular spaces.

In the histological studies of infected leaves of cucumber with CMV, Cook (1938) found that the mosaic affected parts of leaves were thinner than normal parts of leaves. The palisade cells in the mosaic areas were invariably shorter than normal and occasionally they remained undeveloped and cuboidal. The chloroplasts were more numerous and larger in the normal areas than in the mosaic areas. There was little difference in the size of palisade cells in the affected and healthy portion of fruits, but fewer chloroplasts were noted in the chlorotic cells.

Porter (1954) conducted histological and cytological studies and the changes induced by CMV. He studied the leaves of naturally and artificially infected cucumber and showed the initial changes originated in the mesophyll of the affected leaves in mottled portions but necrosis in other species eventually involved other tissues. The yellow areas appear hyperplastic in young leaves and hypoplastic in mature ones. The palisade in both are reduced in size and aggregated to form granular masses. The dark green raised areas of mottled leaves resulted from hyperplasia in mesophyll cells.

Joshi and Dubey (1975) studied the histopathology of virus infected chilli leaves caused by CMV. A clear change in the length and breadth of palisade cells was noted. A clear distinction between palisade and spongy parenchyma was noted.

TRANSMISSION:

a) Mechanical transmission:

Doolittle (1920) had shown that the transmission of CMV in the field was effected by mechanical means during the training and thinning of plants and plucking of fruits. He also demonstrated the sap transmission of the virus.

b) Insect transmission:

The aphid transmission of cucumber mosaic virus was first demonstrated by Doolittle (1920) by Aphis gossypii Glov. in the field conditions.

It was reported that cucumber aphids (Aphis gossypii) after feeding on a mosaic affected plant can transmit the disease within 5 minutes to a healthy plant (Anon., 1926).

Hoggan (1930) proved on the basis of green house trials that Myzus pseudosolani, Myzus circumflexus and Macrosiphum solanifolii were able to transmit CMV from tobacco and tomato.

Hoggan (1933) showed that single individuals of green peach aphid (Myzus persicae) were able to transmit CMV to tobacco, but the per cent infection increased with the number of aphids. The entire process of picking up the virus and transmitting it to healthy plants required only 30 minutes. No incubation period was noticed and the viruliferous aphids found to loose their infectivity after feeding for 2 hours on healthy plants or after starvation for 18 to 27 hours.

Doncaster and Kassanis (1946) found that the Shallot aphid, Myzus ascalonicus (onion aphid) was able to transmit Cucumis Virus 1.

Severin (1947) found that CMV was transmitted by Aphis gossypii, the bean (dock) aphid, Aphis rumicis and Myzus persicae.

Simons (1955) studied the host-vector-virus-relationships of southern cucumber mosaic virus. It was transmitted by Aphis gossypii, Myzus persicae and Aphis fabae in the order of efficiency. The acquisition threshold of the first two vectors ranged from 5 - 10 seconds.

Joshi (1962) reported a strain of Water melon mosaic virus infecting vegetable marrow which was transmitted by Aphis gossypii and Myzus persicae.

Coudriet (1962) reported that rusty plum aphid, Hysteroneura setariae is a vector of CMV.

Reddy and Mariani (1963) found that the mosaic disease of vegetable marrow caused by CMV was transmitted by Aphis craccivora which is the first report of that insect to be a vector of CMV. In addition, Aphis gossypii, Aphis evonymi and Myzus persicae were identified as vectors. The per cent transmission increased when inoculation feeding was given on cotyledons of healthy seedlings.

Hariharasubramanian and Badami (1964) reported a virus disease of pumpkin which was found to be transmitted to Cucurbitaceae only by Aphis laburni.

Mitra and Mariani (1965) identified a mosaic in tori which was not transmitted by any of the insects tested.

Forghani et al. (1966) isolated a CMV strain from oil pumpkin (Cucurbita pepo) transmitted by Aphis gossypii, Aphis fabae, Dysaulacurthium pseudosolani and Macrosiphum solanifolii.

Singh (1970) studied the mosaic of Benincasa and its transmission by Myzus persicae. It is a stylet-borne virus and preliminary fasting upto 4 hours followed by 1 - 2 minutes acquisition feeding increased vector efficiency whereas prolonged starvation reduced it. In serial transfers, aphids soon ceased to be infective.

Verma *et al.* (1970) studied a snake gourd mosaic disease, a virus disease on bottle gourd and a mosaic virus on pumpkin. The diseases were transmitted by *Myzus persicae* and *Aphis gossypii*.

Jaganathan and Ramakrishnan (1971) isolated a mosaic disease on muskmelon and another from pumpkin. In aphid transmission tests, *Myzus persicae* alone transmitted the muskmelon isolate while *Myzus persicae* and *Aphis gossypii* transmitted the pumpkin isolate. They found that a minimum number of 5 aphids are required to transmit the viruses. The vector-virus relationships were studied in detail.

Kaiser and Danesh (1971) studied the biology of CMV on chickpea (*Cicer arietinum*) and reported the transmission of CMV by *Aphis craccivora* to be stylet-borne. The vector acquired the virus with brief probes in less than two minutes.

Nagarajan and Ramakrishnan (1971 a) studied the vector-virus relationships of bitter gourd mosaic virus using *Myzus persicae*, *Aphis gossypii* and *Aphis malvae*.

Nagarajan and Ramakrishnan (1971 b) reported a Water melon mosaic virus on snake gourd and found to be transmitted by *Myzus persicae* and *Aphis gossypii*, out of 8 species of aphids tested, including *Aphis craccivora*.

Detailed studies of the vector-virus relationships were made using Myzus persicae.

Pillai (1971) attempted to transmit the mosaic disease of snake gourd (CMV) by Aphis craccivora and Myzus persicae but was not successful.

Goel and Varma (1973) isolated a new strain of CMV (Luffa strain) and the virus was found to be transmitted by Myzus persicae, Aphis gossypii and Brevicoryne brassicae.

Dubey et al. (1974) identified a snake gourd mosaic virus and designated as Cucumis Virus 1 which was found to be transmitted by Aphis gossypii, Myzus persicae and not by Aphis craccivora and 3 other aphid species.

c) Seed Transmission:

Bewley (1923) reported that an acuba type of CMV is transmitted through seeds of cucurbits. Bewley (1925) reported that out of 2000 seeds of cucumber taken from infected plants, 9 were found to be infected at the pot stage and further studies indicated strong circumstantial evidence of seed transmission.

Doolittle and Walker (1925) showed that CMV is not transmitted through seeds of cucumber. But seeds of certain

wild plants were reported to be an important agency in the over-wintering of the disease. The wild cucumber (Macrompelis lobata) transmit the virus through seeds.

Bewley and Corbett (1930) observed that CMV is seed transmitted in cucumber and showed that 17 out of 100 cucumber plants carried the infection.

Chamberlain (1939) reported CMV is seed transmitted in vegetable marrow.

Rainio (1943) studied the causal virus of crinkle disease of cucumber which was found to be seed transmitted.

Vasudeva and Pavgi (1945) reported the seed transmission of Melon mosaic virus which is reported to be a strain of CMV.

Radar et al. (1947) reported a seed-borne mosaic virus on muskmelon which differed from CMV by its restricted host range and much higher per cent seed transmission.

Joshi (1962) isolated a strain of Water melon mosaic virus infecting vegetable marrow which is reported to be seed transmitted in that host.

Reddy and Mariani (1963) isolated a mosaic type of virus from vegetable marrow and differentiated 3 types of viruses. They reported that out of 1308 seeds germinated, only one seedling showed mosaic symptoms.

Mukhopadhyay and Saha (1968) reported that cucumber mosaic virus is transmitted through seeds of Cucurbita maxima.

Tomlinson and Carter (1970) showed that CMV is seed transmitted in chick weed (Stellaria media), the per cent infection varied from 3 - 20 in naturally infected plants and 20 - 40 in plants raised from seeds obtained from artificially infected plants. CMV was also seed transmitted in Lamium purpureum (4%), Cerastium holosteoides (2%) and Spergula arvensis (2%) but not through seeds of other weeds.

Nagarajan and Ramakrishnan (1971b) reported a water melon mosaic virus on snake gourd and was shown to be transmitted through seeds to a little extent.

Goel and Varma (1973) isolated a new strain of CMV from luffa which was tested for seed transmission but none was found to be seed transmitted.

Sharma and Chohan (1973) reported the transmission of Cucumis Virus 1 and 3 through seeds of cucurbits. These viruses have been found seed-borne in vegetable marrow, ash gourd and pumpkin (Cucurbita moschata) and identified as Cucumis Virus 1. Cucumis Virus 3 was found to be seed-borne in bottle gourd and ridge gourd.

Dubey et al. (1974) tested the seed transmission of snake gourd mosaic virus. Out of 432 seedlings raised from seeds from infected snake gourd plants, none was found infected.

Kemp et al. (1974) reported the occurrence of squash mosaic virus in musk melon seeds. The virus was detected in seeds of 3 out of 29 musk melon. The per cent seed transmission was 6.3.

PHYSICAL PROPERTIES

Doolittle (1920) showed that CMV was never infectious after 3 - 5 days and lost its virulence within 24 to 48 hours.

Johnson (1926) showed that the infectivity of CMV was lost in 2 days in liquid tobacco extract and in 2 - 3 weeks in slowly drying leaves.

Walker (1926) compared the properties of cucumber mosaic, tomato mosaic and physalis mosaic. The expressed juice of CMV is incapable of withstanding aging, drying and heating.

McKinney (1927) reported that the thermal death point depends on the concentration of the virus in the sap and on the nature of plant extract.

Fajardo (1930) compared the properties of CMV and bean mosaic virus and showed that the thermal death point, dilution end point and resistance to aging of CMV are, 75°C, 1:10000 and 24 - 28 hours respectively. It remains infective in dried leaves for less than 10 days.

Chamberlain (1939) described the physical properties of CMV, viz., thermal inactivation point - (62-66°C), dilution end point - (1:1000) and survival *in vitro* (4 days).

Vasudeva and Lal (1943) observed that T.I.P., D.E.P. and resistance to aging of cucumber mosaic virus 3 on bottle gourd as 60°C, 1:500, and 6 hours at room temperature respectively.

Vasudeva *et al.* (1949) showed the T.I.P., D.E.P. and longevity *in vitro* of Cucumis Virus 2 C from bottle gourd as 86 to 88°C, 1:1000 in standard sap and 1:10000 in pure sap, more than 90 days respectively.

Bhargava (1951) studied the physical properties of 4 viruses occuring on cucurbits. The T.I.P. was 55 - 60°C for yellow strain and 65 - 70°C for others.

Reddy and Nariani (1963) isolated three types of viruses from vegetable marrow. The mosaic type had a T.I.P. of 52 - 55°C, D.E.P. of 1:200 to 1:300 and longevity *in vitro* of 12 - 16 hours at room temperature (32 - 35°C) and 8 days at 7 - 9°C. The filiform type had a T.I.P. of 55 - 60°C,

D.E.P. of 1:500 - 1:1000 and longevity *in vitro* of 24 hours at 32 to 35°C and 9 days at 7 - 9°C.

Verma *et al.* (1970) isolated viruses from snake gourd, bottle gourd and pumpkin. The snake gourd mosaic and bottle gourd mosaic had a T.I.P. of 97.5°C, D.E.P. of 10^{-6} to 10^{-7} and longevity *in vitro* for 9 to 10 days at 30°C. The virus isolated from pumpkin had a T.I.P. of 60 - 70°C, D.E.P. of 10^{-3} to 10^{-4}, and longevity *in vitro* of 7 - 9 days.

Jaganathan and Ramakrishnan (1971) isolated a virus from musk melon and another from pumpkin. The musk melon isolate had a T.I.P. of 60 - 63°C, D.E.P. of 1:5000 to 1:7500 and longevity *in vitro* of 8 - 10 days at 5°C. The pumpkin isolate had a T.I.P. of 50 - 53°C, D.E.P. of 1:2500 to 1:5000 and longevity *in vitro* of 4 to 6 days at 32°C and 8 - 10 days at 5°C. Both the isolates were identified as strains of Melon mosaic virus.

Nagarajan and Ramakrishnan (1971 b) isolated a Water melon mosaic virus form snake gourd. It had a T.I.P. of 52 - 54°C, D.E.P. of 1:200 - 1:500 and longevity *in vitro* at 32°C was 4 - 6 days and at 5°C was 4 to 8 days.

Pillai (1971) identified a mosaic disease of snake gourd, which had a D.E.P. of 1:10000, T.I.P. 60°C and longevity *in vitro* was up to 72 hours at room temperature.

Shankar *et al.* (1972) identified a pumpkin mosaic virus which was inactivated at 50°C, at a dilution of 1:500 and in 8 hours at room temperature and 26 hours at 8°C.

Dubey *et al.* (1974) isolated *Cucumis Virus* 1 from mosaic infected snake gourd. Its D.E.P. was between 1:1000 - 1:5000, T.I.P. between 65 to 70°C and longevity *in vitro* in M/30 phosphate buffer (pH 7.6) was 16 - 18 hours at 34.6° - 39°C and 8 days at 8°C.

Shankar and Mariani (1974) reported a Water melon vein-banding mosaic which was identified as *Cucumis Virus* 2. It had a T.I.P. of 95 - 98°C, D.E.P. of 1:1000000 - 1:5000000 and a longevity *in vitro* of more than one year at room temperature.

HOST RANGE

Doolittle (1921) mentioned the role of host plants such as *Asclepias syriaca*, *Martynia louisiana* as over wintering hosts of CMV. After that, large number of plant

species in different families were recorded as hosts or carriers of CMV. Doolittle and Walker (1923) showed in cross-inoculation studies with CMV that a large number of plants are susceptable to infection by the virus and some serve as host of the virus. It was also found that CMV is transmissible to tobacco through pepper as an intermediate host.

Johnson (1926) reported that CMV is transferable to tobacco, Nicotiana glutinosa and a number of other plants.

Samuel (1931) showed that CMV produces local lesions on tobacco. Hoggan (1933 b) reported the formation of local lesions on sugar beets due to CMV.

Ainsworth (1934) found yellow cucumber mosaic infecting vegetable marrow, gherkin, tomato and Datura stramonium.

Price (1935) reported the occurrence of CMV in Zinnia elegens. Smith (1936) reported the occurrence of Cucumis Virus 1 on a number of wild and cultivated plants.

Vasudeva and Lal (1943) reported a mosaic disease of bottle gourd which was transmitted to bitter gourd, melon, water melon, and vegetable marrow and identified as Cucumber Virus 3.

Vasudeva and Pavgi (1945) showed a melon mosaic virus, identified as CMV, infecting cucumber and to be transmitted to a number of Solanaceous plants.

Vasudeva et al. (1949) reported a virus disease in bottle gourd caused by Cucumis Virus 2 c which differed from the early mentioned strain of cucumber green mottle mosaic virus by its ability to produce symptoms on water melon and Datura stramonium. The virus was carried symptomlessly on bitter gourd and Luffa acutangula.

Freitag (1952) isolated seven virus diseases of cucurbits, viz, CMV, squash mosaic, musk melon mosaic, wild cucumber mosaic, cantaloup mosaic, water melon mosaic and musk melon vein necrosis. The host range of all the viruses except CMV is very much restricted.

Vasudeva and Mariani (1952) studied the host range of bottle gourd mosaic virus and found that it caused localized infection on inoculated leaves of tobacco, Solanum nigrum and Solanum nodiflorum.

Anderson (1954) isolated Water melon mosaic virus strains from Florida (the type and the yellow strains) inducing systemic symptoms in 12 Cucurbitaceous species including cannon ball, Water melon, squash, vegetable marrow,

cantaloup, citron, Melothria pendula, bottle gourd Luffa cylindrica and Luffa aegyptiaca.

Van Velson (1960) identified a strain of melon mosaic virus on Cucurbita moschata which had a limited host range causing systemic infection in Cucurbitaceous plants and local lesions on Chenopodium amaranticolor.

Prasad and Raychaudhari (1961) identified a mosaic disease of Zinnia elegans caused by Cucumis Virus 1 with an extensive host range.

Joshi (1962) detected a strain of Water melon mosaic virus infecting vegetable marrow, Cyclanthera pedata, squash and cucumber.

Reddy and Mariani (1963) isolated 3 types of viruses from vegetable marrow. The mosaic type(Cucumis Virus 1) had an extensive host range. It caused systemic infection in snake gourd. The filiform type (Cucumis Virus 3) also caused mosaic on snake gourd but the host range was restricted to Cucurbitaceae.

Hariharasubramanian and Badami (1964) recorded a mosaic on pumpkin which was transmissible only to members of Cucurbitaceae and identified as a new virus though resembled bottle gourd mosaic virus.

Allam (1965) isolated a mosaic from vegetable marrow, the host range being restricted to Cucurbitaceae and the virus was identified as squash mosaic virus.

Mitra and Nariani (1965) isolated a mosaic from tori (Cucumis Virus 3). The host range was restricted to Cucurbitaceae and it involves Lagenaria siceraria, Cucurbita moschata, Cucurbita pepo, Momordica charantia, Trichosanthes anguina and Citrullus vulgaris.

Anjaneyulu and Apparao (1967) reported the natural occurrence of CMV on chilli in India.

Inouque et al. (1967) studied a mosaic disease in Japan on cucumber and identified as a cucumber green-mottle mosaic virus and found that many cucurbit hosts are systemically affected; while tobacco was infected locally without lesions and local lesions were formed on Datura stramonium and Petunia hybrida.

Shankar et al.(1969) reported a mosaic disease in snake gourd (CMV) having an extensive host range which included Nicotiana glutinosa, Chenopodium amaranticolor and Cucurbita pepo.

Verma et al. (1970) recorded a severe mosaic disease on snake gourd and the host range was restricted

to members of Cucurbitaceae. But it produced local lesions on Chenopodium amaranticolor.

Jaganathan and Ramakrishnan (1971) showed that the host range of viruses isolated from musk melon and pumpkin are confined to Cucurbitaceae. Among different species of cucurbits tested, snake gourds and bitter gourds were also found to be the hosts. The viruses were identified as strains of Melon Mosaic virus.

Nagarajan and Ramakrishnan (1971b) reported the occurrence of Water melon mosaic virus on snake gourd, the host range being restricted to Cucurbitaceae.

Pillai (1971) identified a mosaic disease of snake gourd in Kerala. Out of 31 species of plants in 7 families tested 15 species in 6 families were found to be hosts of the virus. The causal virus was identified as a strain of CMV.

Shankar et al. (1972) reported the natural occurrence of pumpkin mosaic in India, the host range being restricted to Cucurbitaceae viz. snake gourd, bitter gourd, bottle gourd, Water melon, Luffa acutangula, Cucurbita pepo and Cucumis melo.

Dubey et al. (1974) identified a mosaic disease of snake gourd in India caused by Cucumis Virus 1. The host range includes numerous plants in different families.

Shankar and Narieni (1974) identified a melon vein banding mosaic in India. Host range was limited to Cucurbitaceae which included melon, cucumber, Cucumis melo, Cucumis anguria, bottle gourd, snake gourd, Cucurbita pepo and bitter gourd.

Nagarajan and Ramakrishnan (1975) reported a strain of melon mosaic virus on Cucurbita lundelliana. The virus was transmitted to snake gourd.

Chung et al. (1975) recorded 145 plants belonging to 43 families which act as hosts of CMV in Korea, of which 24 are previously unreported.

ESTIMATION OF LOSS:

Doolittle (1924) studied the loss due to cucumber mosaic virus in U.S.A. and estimated a loss of $ 75,000 in a single locality. There are several reports regarding the loss of different Cucurbitaceous crops infected by CMV from different parts of the world. But a systematic study to understand the extent and the manner in which loss occur due to infection by CMV has been conducted only in a few cases.

Middleton and Whitaker (1942) reported a lethal virus disease of cantaloup occurring in Imperial Valley which is caused by a strain of CMV and recorded a loss of about 75 per cent of the crop.

Vasudeva and Lal (1943) observed that bottle gourd affected by a mosaic when attacked in early seasons, remained stunted, sparsly flowered, and set only few fruits.

Hills et al. (1961) studied the effect of CMV on cantaloup and recorded that inoculation of melon plants at the 6th leaf stage caused 40 per cent reduction in yield.

Fletcher et al. (1969) found that cucumber plants affected by cucumber green-mottle mosaic virus caused 15 per cent loss in yield following early infection. later infection had little effect on yield.

Demski and Chalkley (1972) showed that when summer squash plants at different developmental stages were inoculated with Water melon mosaic virus, yield loss averaged 43 per cent, 38 per cent, and 9 per cent from early, mid-term and late inoculations respectively.

Kazda et al. (1975) showed that yield of green-house cucumber plants, inoculated with CMV, at the

cotyledonary stage, was reduced by 89 per cent in summer and by 96 per cent in winter and 80 per cent and 94 per cent, respectively, when inoculated at the 5 and 7 leaf stages.

Alvarez and Campbell (1976) analysed the yield factors of cantaloup infected by squash mosaic virus and recorded significant reduction in the number of fruits per plant but had no influence on size, weight, or edible quality (total soluble solids) of the fruits. However, retardation in fruit maturity was observed.

MATERIALS AND METHODS

MATERIALS AND METHODS

1. SEED MATERIAL:

Seeds of snake gourd, obtained from the Instructional Farm, Vellayani, were used for the study. The seeds were sown in pots containing the standard potting mixture (Sand, Soil, Cowdung in the ratio of 1:1:2).

2. CULTURE OF THE VIRUS:

The culture of the virus causing snake gourd mosaic was obtained from naturally infected snake gourd plants of the locality. The virus was maintained on young snake gourd plants by sap inoculation. Two to three seeds were sown in each pot (size 20 cms x 20 cms) but only one vigorously growing plant per pot was retained, and used for the studies. The plants were inoculated in all cases, except otherwise stated, at 3 - 4 leaf stage.

3. SYMPTOMATOLOGY:

a) External Symptoms:

The symptoms produced by the plants artificially inoculated with the virus, were studied. The changes observed on the vegetative parts and floral parts were recorded.

b) Histopathology:

The transverse sections of the infected and healthy leaves were taken using a sharp blade and examined after staining with safranin under the microscope to study the changes and damages on the different tissues caused by the infection. The observations on the histological changes were recorded.

4. TRANSMISSION OF THE VIRUS:

a) Sap transmission:

Inoculation of snake gourd plants were conducted using concentrated sap, standard sap and sap extracted with phosphate buffer. Inoculations were also made with and without carborundum powder as an abrasive.

Young leaves of infected plants showing clear symptoms of mosaic were collected and ground in a mortar and pestle to a fine pulp. Then it was filtered through fine muslin cloth and the filterate was used for inoculation (concentrated sap).

The standard sap was prepared by adding 1 ml. of sterilised distilled water to every gram of infected tissue used for extraction of sap.

Phosphate buffer was prepared (Sorensen's buffer) and the pH was adjusted to 7.0. The buffer was used as an

extraction medium of the sap. (1 ml of buffer/g. of infected tissue).

The effectiveness of inoculation by using carborundum as an abrasive was tested. A pinch of finely powdered carborundum (500 mesh) was added to the sap and the plants were inoculated.

Inoculations were carried out by gently rubbing the upper surface of the fully formed young leaves of the test plants with a swab of absorbent cotton moistened with the sap. Inoculations were also made by rubbing the leaves with the forefinger dipped in the sap. Care was taken not to injure the leaf tissues during inoculation. After inoculations, the excess sap on the leaves was washed off using sterilised distilled water. Ten plants each were inoculated for each experiment and they were kept under insect-proof conditions. Observations were recorded on the number of plants infected.

b) Insect transmission:

Insect transmission studies were carried out using two species of aphids: (a) Aphis gossypii Glov., (b) Aphis craccivora Koch.

Healthy colonies of Aphis craccivora were maintained on cowpea and Aphis gossypii on bhindi under insect proof conditions.

Healthy insects were collected and transferred to petriplates. They were starved for a period of 2 hours (pre-acquisition fasting period) and then were allowed to feed on detached young leaves of infected snake gourd plants, so as to give them an acquisition feeding period of 24 hours. After the acquisition feeding period, the insects were starved for an hour (pre-infection starvation). The viruliferous aphids were then released on young healthy test plants, at the 3 - 4 leaf stage, for an infection feeding period of 24 hours using glass chimneys. In every case 30 adult insects were used for inoculation. After the infection feeding period, they were killed by spraying 0.1% Ekalux solution. The inoculated plants were kept for observation under insect-proof conditions.

To study the vector - virus relationships, the following experiments were conducted using the aphid, Aphis craccivora, as the vector.

1) Acquisition feeding period:

In order to determine the minimum acquisition feeding period of the vector, the aphids were allowed to acquire virus for different periods of time: 5, 10, 15 and 30 minutes, 1, 2, 4, 8, 16 and 24 hours. The pre-acquisition and pre-infection starvation periods were 2 hours and 1 hour

respectively. The aphids (twenty each) were allowed to feed on healthy test plants for 24 hours. After the infection feeding the plants were sprayed with the insecticide and kept under observation for the development of symptoms.

ii) Minimum number of aphids required for transmission of the virus:

Aphids in groups of 3, 5, 10, 15, 20, 25 and 30 were released on each test plant after allowing a pre-acquisition starvation period of two hours, an acquisition feeding period of 30 minutes, pre-infection starvation period of 1 hour and an infection feeding time of 24 hours to determine the minimum number of aphids required for the transmission of the virus. After infection feeding, insects were killed by spraying 0.1% Ekalux solution and the plants kept under observation.

iii) Infection feeding period:

The minimum infection feeding period of the insect to transmit the virus was determined. Non-viruliferous aphids were given 2 hours pre-acquisition starvation, an acquisition feeding period of 30 minutes and a pre-infection starvation of 1 hour. Different batches of 20 aphids each were given different infection feeding periods - 5, 10, 15 and 30 minutes; 1, 2, 4, 8, 16, 22 and 24 hours. Observations were recorded on the number of plants infected.

iv) Effect of pre-acquisition fasting of aphids in the efficiency to acquire the virus:

In order to estimate the effect of pre-acquisition starvation on the efficiency of the vector to acquire the virus, the insects were starved for different periods of time and allowed an acquisition feeding period of 30 minutes. After the acquisition feeding, the insects were again starved for 1 hour and released on test plants and allowed to feed for 24 hours. After the infection feeding period, the insects were killed by sparying Ekalux 0.1% solution and the plants were kept under observation. The pre-acquisition starvation period tried were 30 minutes, 1, 2, 3, 4 and 5 hours.

v) Effect of pre-infection starvation on the efficiency of the vector to transmit the virus:

Aphids, starved for 2 hours, were allowed an acquisition feeding period of 30 minutes and starved for different periods of time. They were afterwards transferred to healthy test plants and allowed to feed for 24 hours. Plants inoculated with viruliferous aphids without pre-infection starvation were maintained as control. In every case 20 insects were used. The pre-infection starvation time were 30 minutes.

1, 2, 3, 4 and 5 hours. Observations were recorded on the number of plants infected.

vi) Persistence of the virus in the insect vector:

Experiments were conducted to determine how long the insect could retain the virus without further access to a fresh virus source. The insects, starved for 2 hours, were allowed to feed on infected leaf to acquire the virus for 30 minutes. After giving a pre-infection starvation of 1 hour, the viruliferous aphids were fed on test plants by transferring them in succession to a series of test plants, allowing an infection feeding time of 1 hour on each test plant. The inoculated plants were sprayed with 0.1% Ekalux solution. Observations were recorded on the number of plants infected.

c) Seed transmission:

Seeds collected from mechanically inoculated plants showing clear symptoms of the disease were sown in sterilized soil in pots and kept under insect-proof conditions. The seedlings were kept under observations for a period of 30 days to see whether they develop the symptoms.

d) Soil transmission:

Snake gourd plants were raised in pots and inoculated mechanically. When the plants were 30 days old, the

infected plants were pulled out from the pots and the roots were cut into small bits, and incorporated into the soil in the same pots. Seeds of snake gourd were sown in those pots (5 seeds per pot) at intervals of 5 days each. The plants were maintained under insect-proof conditions and kept under observations.

5. PHYSICAL PROPERTIES OF THE VIRUS:

a) Thermal inactivation point:

The sap was extracted from infected leaves of snake gourd plants using a mortar and pestle. Five ml. each of the extracted (concentrated) sap was pipetted into thin walled glass test tube. Care was taken not to pour the sap on the sides of the tubes. The tube was then kept in thermostatically controlled water bath for 10 minutes at the required temperature in such a way that the level of the water in the water bath was just above the level of sap in the tube. After the heat treatment the tube was suddenly cooled by dipping in cold water. The sap was treated at different temperatures ranging from 40°C to 90°C at ranges of 5°C. The treated sap was inoculated on young vigorously growing test plants of the same age. The inoculation of plants was first done with sap treated at the highest temperature and then the other plants were inoculated with sap treated at low temperatures. Ten plants were inoculated in

each set of treatment. Observations were recorded on the number of plants infected.

b) Dilution end point:

The sap was prepared as in the previous case, and was diluted with sterilised distilled water in the ratio of 1:100, 1:500, 1:1000, 1:5000, 1:10000, 1:50000 and 1:100000.

The diluted sap was inoculated separately on test plants (10 for each treatment) starting from the highest dilution. The inoculated plants were kept under insect-proof conditions and observed for the development of symptoms.

c) Longevity in vitro:

The inoculum was prepared as in the previous tests and kept in tubes at room temperature ($32^{\circ}C$ - $35^{\circ}C$) and also at $10^{\circ}C$ (stored in a refrigerator). One tube containing the sap from each treatment was taken after every 24 hours and inoculated on the test plants. The inoculated plants were kept under insect-proof conditions and observed for the development of symptoms.

6. HOST RANGE:

Thirtythree different plant species belonging to 10 families were tested to study the host range of the virus by mechanical inoculation using concentrated sap extracted

from the leaves of the infected plants.

Amaranthaceae

Gomphrena globosa L.

Balsaminaceae

Impatiens balsamina L.

Caricaceae:

Carica papaya L.

Chenopodiaceae

Chenopodium amerantlcolor Coste and Reyn.

Compositae

Helianthus annuus L.

Zinnia elegans Jacq.

Cucurbitaceae

Benincasa hispida Cogn.

Citrullus vulgaris Schrad.

Cucurbita maxima L.

Cucurbita pepo var *condensa*

Cucumis sativus L.

Lagenaria siceraria (Mol.) Standl.

Luffa acutangula Roxb.

Momordica charantia L.

Euphorbiaceae

Manihot esculenta Crantz.

Leguminosae

Crotalaria juncea L.

Cyamopsis tetragonoloba (L.) Taub.

Glycine max (L.) Merr.

Phaseolus lunatus L.

Phaseolus mungo L.

Phaseolus vulgaris L.

Pisum sativum L.

Vigna sinensis Torner.

Malvaceae:

Abelmoschus esculentus (L.) Moench.

Solanaceae:

Capsicum annuum L.

Datura stramonium L.

Lycopersicon esculentum Mill.

Nicotiana glutinosa L.

Nicotiana longsdorfii L.

Nicotiana tabacum L.

Petunia hybrida Vilm.

Solanum melongena L.

Solanum nigrum L.

7. ESTIMATION OF LOSS

Experiments were conducted to estimate the effect of infection at different stages of growth of the plants, on the vegetative growth and loss in the yield.

The experiment was laid out in Completely Randomised Design. Snake gourd plants were grown individually in pots. The following treatments were fixed to estimate the effects of infection at different growth stages of the plants. Five plants each were inoculated in each treatment.

Treatment - 1	(T_1)	Inoculation of 15 day old plants.
Treatment - 2	(T_2)	Inoculation of 30 day old plants.
Treatment - 3	(T_3)	Inoculation of 45 day old plants.
Treatment - 4	(T_4)	Inoculation of 60 day old plants.
Treatment - 5	(T_5)	Inoculation of 75 day old plants.
Treatment - 6	(T_0)	Control plants were maintained without inoculation.

All the plants were periodically sprayed with 0.1% Ekalux solution to keep the plants free of insect infestation.

Observations were recorded on the following aspects at an interval of 10 days till the plants were 110 days old.

(a) Number of leaves formed.

(b) Length of leaves.

(c) Width of leaves.

(d) Internodal length.

(e) Number of branches developed.

(f) Total length of the vine.

(g) Number of flowers formed.

(h) Per cent of fruits set.

(i) Number of fruits formed.

8. STATISTICAL ANALYSIS:

The data were analysed statistically by applying the technique of analysis of variance for Completely Randomised Design with more replications for control and the significance was tested by 'F' test (Cochran and Cox 1965). The data on the number of leaves, number of branches, number of flowers and number of fruits formed were analysed after square root transformations for counts (Snedecor and Cochran, 1967).

RESULTS

R E S U L T S

I. SYMPTOMATOLOGY:

a) External Symptoms:

The symptoms first manifested as a transitory vein clearing on the young leaves followed by the appearance of small greenish-yellow areas, which are transparent than the healthy areas of the leaf. Later, clear mosaic symptoms consisting of irregular dark green and yellow chlorotic patches developed. The small chlorotic areas coalesced to form bigger patches. In advanced stages of infection, dark green raised blisters of varying size and shape developed on the lamina. The leaves were markedly reduced in size and variously crinkled and deformed. In older leaves, the symptoms of mosaic mottling was less pronounced and only chlorotic areas were seen. The growth of infected plants was severely retarded and the internodes were shortened. The plants infected at very early stages, blossom sparingly and set only very few or no fruits. The development of auxillary branches were also induced. No visible symptoms were observed on flowers and fruits, but excessive shedding of flowers was noticed. (Plate-1)

Plate-1

Leaf of snake gourd showing the symptoms of the mosaic disease.

b) Internal Symptoms:

Histopathological studies showed a reduction in thickness of the infected portions (lighter areas) of the leaf i.e. thinner than healthy green areas. The palisade cells in the affected portions were shortened and loosely packed. In the mesophyll cells chloroplasts were fewer in number and showed some kind of degeneration. Since these cells contained only very few chloroplasts, they were more transparent than healthy tissues.

II. TRANSMISSION OF THE VIRUS:

a) Sap Transmission:

The snake gourd plants were inoculated with concentrated sap extracted from leaves of severely infected plants and observed for the development of symptoms. The symptoms of infection appeared on the newly formed leaves, 6 - 8 days after inoculation.

When plants were inoculated using standard sap, the symptom expression was delayed, symptoms appeared in 7 - 10 days. Use of phosphate buffer did not show any significant increase in the infectivity of the virus. When carborundum (500 mesh) was used as an abrasive during inoculations no significant increase of infectivity was noticed.

b) Insect Transmission:

The results of the studies of insect transmission are given in Table 1. Transmission of the virus with Aphis gossypii Glov. and Aphis craccivora Koch. were tried. Though both the species of aphids transmitted the virus, Aphis gossypii was more efficient in transmitting the virus and the symptoms appeared earlier.

Vector-virus relationships:

The vector-virus relationships were studied using Aphis craccivora Koch. as the vector.

1. Determination of the minimum acquisition feeding time:

The results are given in Table 2. It was observed that the aphid can acquire the virus with a minimum feeding time of 5 minutes on the infected leaves. The infectivity of the virus was found to be increased when the acquisition feeding time was increased upto a period of 30 minutes; but further increase in the acquisition feeding time only reduced the per cent of infected plants and no infection could be obtained after 8 hours of acquisition feeding time. A period of 6 to 9 days was taken after infection feeding for the manifestation of symptoms.

TABLE I

Results of the experiment to determine transmission of snake gourd mosaic virus by aphid vectors.

Test insect	Number of aphids	Pre-acquisition fasting time (hours)	Acquisition feeding time. (hours)	Pre-infection fasting time. (hours)	Infection feeding time. (hours)	Number of plants infected.	Number/ per cent of plants infected	Time taken for appearance of symptoms (days)
Aphis gossypii	30	2	24	2	24	15	5/33.3	8 - 9
Aphis craccivora	30	2	24	2	24	20	6/30.0	10 - 12

TABLE 2

Results of the experiment to determine the minimum acquisition feeding time.

Number of aphids	Pre-acquisition fasting time (hours)	Acquisition feeding time.	Pre-infection starvation time. (hour)	Infection feeding time. (hours)	Number of plants inoculated.	Number/percent of plants infected.	Time taken for the appearance of symptoms (days)
20	2	5 minutes	1	24	15	4/26.6	8
20	2	10 "	1	24	15	4/26.6	7
20	2	15 "	1	24	15	5/33.3	6
20	2	30 "	1	24	15	9/60.0	7
20	2	1 hour	1	24	15	6/40.0	6
20	2	2 "	1	24	15	3/20.0	9
20	2	4 "	1	24	15	3/20.0	9
20	2	8 "	1	24	15	0	Not infected
20	2	16 "	1	24	15	0	"
20	2	24 "	1	24	15	0	"

ii. Minimum number of aphids required for infection:

The results of the studies are presented in Table 3. No infection could be obtained when a minimum number of 3 aphids were used for the trial. Infection was noticed when 5 infective insects or more were allowed to feed on the test plants. Maximum infection was obtained when 20 insects were used per plant. The symptoms were manifested within 7 to 9 days. It was found that when the number of aphids were increased per plant, the per cent of transmission was also increased.

iii. Determination of the minimum infection feeding period:

It was found that the aphids can transmit the virus with a minimum infection feeding period of 5 minutes on the test plants (Table 4). The per cent of infected plants was increased by increasing the infection feeding period upto 2 hours. The per cent of infected plants was found to be reduced when the infection feeding time was further increased. The plants exhibited the symptoms within 5 to 9 days. When a maximum infection feeding time of 24 hours was given, the development of symptoms was found to be delayed.

iv. Effect of pre-acquisition starvation of aphids on the efficiency to acquire and transmit the virus:

The data are presented in Table 5. Pre-acquisition fasting of aphids increased the efficiency of the vector to

TABLE 3

Results of the experiment on the minimum number of aphids required for infection.

Number of aphids	Pre-acquisition fasting time. (hours)	Acquisition feeding time. (minutes)	Pre-infection fasting time. (hours)	Infection feeding time. (hours)	Number of plants inoculated.	Number/per cent of plants infected.	Time taken for appearence of symptoms. (days)
3	2	30	1	24	10	0	Not infected.
5	2	30	1	24	10	4/40.0	8
10	2	30	1	24	10	8/80.0	6
15	2	30	1	24	10	8/80.0	8
20	2	30	1	24	10	10/100.0	9
25	2	30	1	24	10	8/80.0	8
30	2	30	1	24	10	3/30.0	7

TABLE 4

Results of the experiment to determine the minimum infection feeding period.

Number of aphids	Pre-acquisition fasting time (hours)	Acquisition feeding time (minutes)	Pre-infection fasting time. (hours)	Infection feeding time.	Number of plants inoculated.	Number/per cent of plants infected.	Time taken for appearence of symptoms. (days)
20	2	30	1	5 minutes	10	2/20.0	7
20	2	30	1	10 "	10	6/60.0	7
20	2	30	1	15 "	10	6/60.0	6
20	2	30	1	30 "	10	8/80.0	5
20	2	30	1	1 hour	10	8/80.0	5
20	2	30	1	2 "	10	10/100.0	5
20	2	30	1	4 "	10	8/80.0	6
20	2	30	1	8 "	10	6/60.0	7
20	2	30	1	16 "	10	8/80.0	6
20	2	30	1	22 "	10	4/40.0	6
20	2	30	1	24 "	10	4/40.0	9

TABLE 5

Effect of pre-acquisition starvation of aphid on the efficiency to acquire and transmit the virus.

Number of aphids.	Pre-acquisition starvation time.	Acquisition feeding time. (minutes	Pre-infection starvation time. (hours)	Infection feeding time. (hours)	Number of plants inoculated.	Number/per cent of plants infected	Time taken for appearance of symptoms (days)
20	30 minutes	30	1	24	10	4/40.0	9
20	1 hour	30	1	24	10	8/80.0	8
20	2 "	30	1	24	10	8/80.0	7
20	3 "	30	1	24	10	6/60.0	8
20	4 "	30	1	24	10	2/20.0	8
20	5 "	30	1	24	10	0	Not infected

acquire and transmit the virus. Maximum efficiency was noted when insects were starved for a period of 2 hours. Further increase of fasting time did not appreciably increase the per cent of infected plants and so also the efficiency of the vector to transmit the virus. The plants developed the symptoms within 7 to 9 days.

v. Effect of pre-infection starvation on the efficiency of the vector to transmit the virus:

The efficiency of transmission was maximum when the viruliferous vectors were starved for 30 minutes before infection feeding. The data also revealed that fasting of aphids before infection feeding upto a period of 1 hour did not severely affect the efficiency of the vector to transmit the virus (Table 6). But further increase of pre-infection fasting showed that the efficiency was declined and after 4 hours fasting the insects were unable to transmit the virus to healthy plants. The symptoms manifested within a period of 9 to 10 days.

vi. Persistence of the virus in the insect-vector:

The results are tabulated in Table 7. The insects were transferred from plant to plant after an infection feeding of 1 hour. It was observed that the virus could be transmitted to the first test plant only; where as the subsequent plants remained healthy when the same insects were

TABLE 6

Effect of pre-infection starvation on the ability of the vector to transmit the virus.

Number of aphids	Pre-acquisition starvation time. (hours)	Acquisition feeding time. (minutes)	Pre-infection starvation time.	Infection feeding time. (hours)	Number of plants inoculated.	Number/percent of plants infected.	Time taken for appearence of symptoms. (days)
20	2	30	0	24	10	8/80.00	8
20	2	30	30 minutes	24	10	8/80.0	9
20	2	30	1 Hour	24	10	7/70.0	9
20	2	30	2 "	24	10	4/40.0	10
20	2	30	3 "	24	10	4/40.0	10
20	2	30	4 "	24	10	0	Not infected
20	2	30	5 "	24	10	0	"

TABLE 7

Results of the experiment to determine the persistence of the virus in the insect vector.

Number of aphids	Pre-acquisition fasting time (hours)	Acquisition feeding time (minutes)	Pre-infection fasting time. (hours)	Infection feeding time. (hours)	Number of plants inoculated.	Number of plants infected.				
						1	2	3	4	5
20	2	30	0	1	5	+	-	-	-	-
20	2	30	1	1	5	+	-	-	-	-
20	2	30	1	1	5	+	-	-	-	-
20	2	30	1	1	5	+	-	-	-	-
20	2	30	1	1	5	+	-	-	-	-

\+ Infected.

\- Not infected.

transferred serially from plant to plant. The insects could not retain the virus and lost its virulence immediately after feeding on a test plant.

c) Seed Transmission:

Seeds from severely infected plants were collected and grown in pots to assess whether the virus is seed-borne. Out of the 67 seeds sown, 52 were germinated. The plants did not show any symptoms of the disease upto 30 days of growth, indicating that the virus is not transmitted through seeds.

d) Soil Transmission:

An experiment was conducted to determine whether the virus is soil-borne. The infected plant materials were incorporated with the soil in pots and after one week, healthy seeds of snake gourd were sown. Observations made for a period of 30 days revealed that the virus is not soil-borne as the plants remained healthy.

III. PHYSICAL PROPERTIES OF THE VIRUS:

a) Thermal Inactivation point:

The results are given in Table 8. The virus was inactivated between temperatures of 70° to 75°C. The test plants, inoculated with sap treated at 75°C remained healthy without exhibiting any symptoms, whereas 20 per cent

TABIE 8

Results of the experiment to determine the thermal inactivation point of the virus.

Treatment (Temperature $^{o}_{C}$)	Number of plants inoculated.	Number of plants infected.	Per cent infection.	Time taken for appearence of symptoms. (days)
50	10	10	100	9
55	10	8	80	10
60	10	9	90	12
65	10	6	60	12
70	10	2	20	11
75	10	0	0	Not infected.
80	10	0	0	"
85	10	0	0	"

test plants developed symptoms when inoculated with sap treated at 70°C.

The plants inoculated with sap treated at lower temperature developed symptoms within 8 to 9 days while there was a delay in the symptom expression when sap was treated at 65°C and 70°C.

b) Dilution end point:

The data are given in Table 9. It was found that the virus was inactivated at a dilution of 1:10000. But 60 per cent of the plants inoculated with the sap at a dilution of 1:5000 developed the symptoms. So it was concluded that the dilution end point of the virus is between 1:5000 - 1:10000. It was also observed that at dilutions above 1:500, the infectivity was reduced and the expression of symptoms was delayed.

c) Longevity in vitro:

The results are given in Table 10 and 11. When the sap extracted from infected plants was stored at room temperature for a period of 96 hours, its infectivity was completely lost. About 40 per cent of the plants, inoculated with sap kept for 72 hours at room temperature developed symptoms. So the longevity in vitro of the virus stored at room temperature is between 72 and 96 hours. The

TABLE 9

Results of the experiment to determine the dilution end-point of the virus

Concentration of sap.	Number of plants inoculated.	Number of plants infected.	Per cent infection	Time taken for appearance of symptoms. (days)
1:100	10	10	100	8
1:500	10	10	100	9
1:1000	10	8	80	11
1:5000	10	6	60	11
1:10000	10	0	0	Not infected
1:50000	10	0	0	"
1:100000	10	0	0	"

TABLE 10

Result of the experiment to determine the longevity *in vitro* of the virus at room temperature ($32^{o}C$ - $35^{o}C$)

Time interval	Number of plants inoculated.	Number of plants infected.	Percent infection.	Time taken for appearence of symptoms (days)
Freshly extracted sap.	5	5	100	9
After 24 hours	5	5	100	12
After 48 hours	5	4	80	11
After 72 hours	5	2	40	14
After 96 hours	5	0	0	Not infected
After 120 hours	5	0	0	"
After 144 hours	5	0	0	"

TABLE 11

Results of the experiment to determine the longevity *in vitro* of the virus stored under refrigerator (10°C)

Time interval	Number of plants inoculated	Number of plants infected.	Percent infection	Time taken for appearance of symptoms. (days)
Freshly extracted sap.	5	5	100	9
After 24 hours	5	5	100	9
After 48 hours	5	4	80	12
After 72 hours	5	3	60	11
After 96 hours	5	2	40	15
After 120 hours	5	1	20	15
After 144 hours	5	1	20	14
After 168 hours	5	0	0	Not infected.

per cent of infected plants was decreased as the storage time of sap was prolonged and at the 72 hours it was only 40 per cent. The plants exhibited the symptoms of the disease within 9 to 11 days when inoculated with sap stored for 24 hours, but the symptom expression was delayed for 14 days in those inoculated with sap stored for 72 hours.

When the inoculum was stored in a refrigerator, the infectivity was retained upto 144 hours, but the per cent of infected plants was considerably decreased. After 168 hours of storage of the inoculum the infectivity was completely lost. So the longevity in vitro of the virus is between 144 to 168 hours, when the sap was stored under refrigerated conditions. When the inoculum was stored for 72 hours the symptoms manifested within 9-12 days but when it was stored for 144 hours the expression of symptoms was delayed upto 14-15 days.

IV. HOST RANGE:

Out of 33 plant species belonging to 10 different families tested, 8 species of plants belonging to 3 families were found to be susceptible to infection by the virus and produced distinct symptoms.

a) Cucurbitaceae:

i. Cucumis sativus L.

The symptoms were first noticed on the developing young leaves as small greenish-yellow areas which was more lighter than healthy areas. Later, characteristic yellow mottle appeared. The leaves were reduced in size, distorted, crinkled and in advanced stages there was downward curling of the edges of the lamina. The plants were stunted and the internodes were shortened.

ii. Cucurbita maxima L.

Yellowish-green chlorotic patches were developed on the lamina. The greenish darker portions of the leaf produced raised blisters. The chlorotic patches coalesced together to form larger patches of infected tissues. The size of leaves were reduced considerably, internodes were shortened and plants were stunted. (Plate-2).

iii. Cucurbita pepo var condensa L.

The infected plants showed the symptoms within 5 to 7 days. The leaves exhibited pale green spots which enlarged in size and developed into yellowish-green patches. Reduction in size of leaves, crinkling and distortion of lamina, were also observed. The infected plants were stunted.

Plate-2

Cucurbita maxima showing symptoms of infection by the snake gourd mosaic virus

b) Solanaceae:

i. Nicotiana tabacum L.

The plants inoculated with concentrated sap did not show any symptoms of the diesease. But when the sap, extracted with phosphate buffer, was inoculated on the plants, the vein clearing symptoms developed within 8 -10 days. The symptoms were mild and only pale green areas were developed on the leaf lamina. (Plate-3)

ii. Nicotiana glutinosa L.

The affected plants showed mottling within 3 to 6 days. A large number of dark green raised blisters were produced on the lamina. The newly emerged leaves were highly reduced in size and plants showed severe stunting. The internodes were shortened. As the plant matured the severity of the symptoms was reduced. (Plate-4)

iii. Capsicum annuum L.

The plants showed the symptoms within 6 to 7 days. The main symptoms were the development of light green mottling and curling of the lamina. The size of leaves were severely reduced and stunting of the plants was also evident.

Plate-3

Symptoms developed on tobacco (*Nicotiana tabacum*) by inoculating snake gourd mosaic virus.

Plate-4.

iv. Petunia hybrida Vilm.

The inoculated plants exhibited symptoms within 8 days. Young leaves showed pale green areas and later developed dark green raised blisters. The size of leaves were reduced and in severe cases internodes were shortened. No symptoms could be observed on flowers.

c) Compositae:

Zinnia elegans Jacq.

Clearing of veins usually developed within 8 to 10 days after inoculation and the plants showed systemic mottling. The symptoms produced were similar to those produced on Petunia hybrida. No visible symptoms were observed on flowers.

V. ESTIMATION OF LOSS:

a) Effect of infection on number of leaves:

The observations of the experiment to find out the effect of inoculation of 15, 30, 45, 60 and 75 day old seedlings, on the number of leaves formed are given in Table 12. The results showed that inoculation of the plants with the virus at the different stages of growth, did not affect the production of leaves significantly as compared with untreated plants.

TABLE 12

Effect of infection at different stages of growth of the plant on the number of leaves

(Means after $\sqrt{X}$ transformation)

Treatments	Time of observation (days)								
	20	30	40	50	60	70	80	90	100
T_1	4.240	5.006	6.050	6.910	7.720	8.516	9.286	9.852	10.330
T_2	-	-	5.674	6.420	7.230	8.116	8.808	9.402	9.950
T_3	-	-	-	6.680	7.520	8.156	8.736	9.260	9.718
T_4	-	-	-	-	-	8.228	8.912	9.506	9.930
T_5	-	-	-	-	-	-	9.028	9.548	9.980
T_0	4.170	4.969	6.020	6.920	7.760	8.602	9.472	10.024	10.522
C.D. for comparison with control.	-	-	-	-	-	-	-	-	-
C.D. for comparison between treatments.	-	-	-	-	-	-	-	-	-

b) Effect of infection on the length of leaves:

The results are given in Table 13. The data showed that the inoculation of 15 day old plants reduced the length of leaves significantly as compared to control and other treatments. It was observed that the length of leaves was reduced significantly over control, when 30 and 60 day old plants were inoculated. When the plants were inoculated on the 75th day the reduction in the length of leaves was not significant. The reduction in the length of leaves depend on the time of inoculation i.e. if the plants were inoculated at early stages of growth, the length of leaves reduced significantly.

c) Effect of infection on the width of leaves:

The effect of inoculation of plants at different growth stages on the width of leaves was recorded and presented in Table 14. The results showed that the width of leaves was reduced significantly when 15 day old plants were inoculated. There was significant reduction in the width of leaves when 30 and 45 day old plants were inoculated. There was no significant reduction in the width of leaves when inoculated on the 60th day and thereafter.

As in the case of length, the reduction in the width of leaves also depends on the time of inoculation. Late inoculations did not appreciably reduce the width of the leaves.

TABLE 13

Effect of infection at different stages of growth of the plant on the length of leaves

(Mean length in Centimetres)

Treatment	Time of observation (days)								
	20	30	40	50	60	70	80	90	100
T_1	4.680	3.920	3.580	3.140	3.000	2.880	2.460	2.520	2.200
T_2	-	-	4.820	4.200	4.140	3.920	3.400	3.200	3.220
T_3	-	-	-	4.320	3.900	3.400	3.300	3.040	2.920
T_4	-	-	-	-	-	5.000	4.780	4.540	4.440
T_5	-	-	-	-	-	-	4.940	4.740	4.540
T_0	7.516	6.244	6.235	5.987	5.840	5.670	5.400	5.160	4.760
C.D. for comparison with control.	0.905	1.131	0.724	0.521	0.497	0.395	0.402	0.469	0.511
C.D. for comparison between treatments.	--	--	0.915	0.638	0.608	0.456	0.402	0.469	0.511

TABLE 14

Effect of infection at different growth stages of the plant on the width of leaves.
(Mean width in centimetres)

Treatment	Time of observation (days)								
	20	30	40	50	60	70	80	90	100
T_1	5.860	4.720	4.180	4.000	3.860	3.560	3.160	2.900	2.800
T_2	-	-	5.120	5.100	4.800	4.580	4.340	3.640	3.740
T_3	-	-	-	4.180	4.660	4.200	4.100	3.780	3.560
T_4	-	-	-	-	-	5.800	5.480	5.480	5.160
T_5	-	-	-	-	-	-	5.260	5.000	4.980
T_0	7.804	6.492	6.350	6.493	6.460	5.870	5.840	5.500	5.220
C.D. for comparison with control.	1.056	1.215	0.968	0.662	0.563	0.571	0.501	0.461	0.489
C.D. for comparison between treatments.	-	-	1.224	0.811	0.689	0.659	0.501	0.461	0.489

d) Effect of infection on the internodal length:

The data on the internodal length are given in Table 15. It was observed that the internodal length is reduced significantly over all other treatments when the 15 day old plants were inoculated. The results also revealed that the inoculation at different stages of plant growth reduced the internodal length significantly over control. The degree of reduction in the internodal length depends on the time of inoculation.

e) Effect of infection on the total length of vine:

The data are presented in Table 16. It was observed that inoculation of 15 day old plants affected the length of vine significantly as compared to the other treatments. Subsequent inoculation of plants did not exert any significant effect in the length of the vine as compared to the control.

f) Effect of infection on the number of branches:

The data on the effect of inoculation at different growth stages of the plants on the number of branches are given in Table 17. The results showed that the number of branches was increased significantly when 15 day old plants were inoculated. The differences in the number of branches

TABLE 15

Effect of infection at different stages of growth of the plant on internodal length

(Mean length in Centimetres)

Treatments	Time of observation (days)								
	20	30	40	50	60	70	80	90	100
T_1	8.780	7.940	7.340	7.100	6.000	5.460	5.120	4.760	4.560
T_2	-	-	10.740	10.500	10.360	9.040	7.060	6.380	5.720
T_3	-	-	-	10.420	7.960	7.620	7.020	6.400	5.860
T_4	-	-	-	-	-	10.040	8.760	7.760	7.300
T_5	-	-	-	-	-	-	8.180	7.840	7.440
T_0	14.470	13.020	13.640	12.573	11.087	9.530	9.800	9.280	8.500
C.D. for comparison with control.	3.384	2.581	1.918	1.438	1.301	0.937	1.325	0.993	0.868
C.D. for comparison between treatments.	-	-	2.426	1.762	1.593	1.082	1.325	0.993	0.868

TABLE 16

Effect of infection at different stages of growth of the plant on the total length of the vine.

(Mean length in centimetres)

Treatment	Time of observation (days)								
	20	30	40	50	60	70	80	90	100
T_1	146.20	182.20	202.20	218.00	234.00	248.20	258.20	265.60	273.40
T_2	--	--	267.80	302.20	330.40	349.00	363.20	374.80	383.20
T_3	--	--	--	330.00	350.60	367.60	385.00	399.40	411.20
T_4	--	--	--	--	--	329.40	340.40	351.00	358.20
T_5	--	--	--	--	--	--	386.80	399.80	408.20
T_0	179.00	238.72	292.30	327.53	355.60	399.20	440.20	452.80	462.20
C.D. for comparison with control.	--	--	68.511	68.314	68.400	68.640	76.390	74.800	73.420
C.D. for comparison between treatments.	--	--	79.110	83.662	83.250	79.270	76.390	74.800	73.420

TABLE 17

Effect of infection at different stages of growth of the plant on the number of branches produced.
(Means after $\sqrt{x+1}$ transformation)

Treatment	Time of observation(days)								
	20	30	40	50	60	70	80	90	100
T_1	1.460	1.820	2.313	2.470	2.561	2.603	2.820	2.890	2.960
T_2	--	--	1.946	1.994	2.364	2.485	2.520	2.640	2.680
T_3	--	--	--	2.088	2.359	2.359	2.410	2.480	2.530
T_4	--	--	--	--	--	2.485	2.530	2.600	2.720
T_5	--	--	--	--	--	--	2.360	2.520	2.600
T_0	0.710	1.340	1.788	2.082	2.372	2.358	2.490	2.680	2.720
C.D. for comparison with control.	0.643	0.643	0.362	--	--	--	0.220	0.244	0.242
C.D. for comparison between treatments.	--	--	0.458	--	--	--	0.220	0.244	0.242

was not significant over control in the case of other treatments.

g) Effect of infection on the number of flowers:

The results are given in Table 18. It was observed that the number of flowers produced was significantly reduced over control by those plants inoculated on the 15th day. Inoculation of 30, 45 and 60 day old plants also reduced the number of flowers significantly over control. It is observed that the reduction in the number of flowers depend on the time of inoculation. Earlier inoculations severely affected flower formation.

h) Effect of infection on the per cent fruit set:

It was observed from the data presented in Table 19 that there was no fruit set when 15 day old plants were inoculated. When plants were inoculated on 30th day of growth, the per cent of fruit set was significantly reduced over control. There was no significant reduction in the fruit set on other treated plants when compared with control. So early infection resulted in significant reduction of fruit set whereas later infections did not exert significant effect on fruit set.

TABLE 18

Effect of infection at different stages of growth of the plant on the number of flowers formed.

(Means after $\sqrt{x+1}$ transformation)

Treatments.	Time of observation (days)								
	20	30	40	50	60	70	80	90	100
T_1	1.219	2.494	2.138	2.341	2.479	2.679	2.083	2.020	1.890
T_2	--	--	2.839	2.303	2.231	2.364	2.169	1.890	1.720
T_3	--	--	--	2.839	2.485	2.556	2.173	1.830	1.710
T_4	--	--	--	--	--	2.738	2.927	2.630	2.480
T_5	--	--	--	--	--	--	2.964	2.570	2.640
T_0	1.885	2.737	2.912	3.052	3.126	3.184	2.925	2.950	2.820
C.D. for comparison with control.	1.051	--	0.353	0.349	0.354	0.292	0.431	0.359	0.312
C.D. for comparison between treatments.	--	--	0.441	0.428	0.434	0.337	0.431	0.359	0.312

TABLE 19.

Effect of infection at different stages of growth of the plant on the per cent of fruit set.

(Means after angular transformations)

Treatment	Mean
T_1	0
T_2	9.950
T_3	11.256
T_4	11.362
T_5	11.250
T_0	13.192
C.D.	4.034

i. Effect of infection on the number of fruits:

The data are given in Table 20. It was observed that when 15 day old plants were inoculated no fruits were formed. When plants were inoculated at subsequent stages of growth, there was significant reduction of fruit formation over control. The effect of the time of infection of the plants was evident on the fruit production.

TABLE 20.

Effect of infection at different stages of growth of the plant on the number of fruits formed.

(Means after $\sqrt{x + 1}$ transformation)

Treatment	Mean
T_1	1.000
T_2	1.512
T_3	1.605
T_4	1.829
T_5	1.839
T_0	2.184
C.D.	0.287

DISCUSSION

D I S C U S S I O N

The virus causing mosaic disease of snake gourd (*Trichosanthes anguina* L.) was investigated. The disease was found to be widespread in Kerala. Symptomatological studies showed that the symptoms produced on snake gourd were similar to CMV reported by Smith (1937). It also resembled the symptoms produced on snake gourd by a strain of CMV reported by Pillai (1971) and *Cucumis Virus* 1 reported by Dubey *et al.* (1974).

The histopathological studies revealed that the palisade cells were shortened in the affected portions and loosely packed. The chloroplasts were fewer in number. The affected portions were found to be thinner than the normal areas. Such an effect have been reported by Gigante (1934) on vegetable marrow due to a mosaic virus. Cook (1938) had also reported similar histological changes due to CMV on cucumber.

The virus was found to be transmitted by sap inoculation. Attempts to transmit the virus by the aphid vectors, *Aphis gossypii* Glov. and *Aphis craccivora* Koch, were successful. But the virus was not transmitted through seed or soil.

Sap inoculation with carborundum as an abrasive did not increase the infectivity of the virus. But when carborundum was used as abrasive, for inoculating, with standard sap, the infectivity was increased as compared to the inoculation without abrasive. Inoculation with sap extracted with phosphate buffer did not increase the infectivity of the virus. This is not in agreement with the observations of the earlier workers (Thornberry, 1935; Costa, 1944). They reported several fold increase of infectivity of CMV with the use of phosphate buffer.

Transmission of snake gourd mosaic by the aphid, Aphis gossypii was reported by earlier workers (Dubey et al., 1974). Reddy and Narlani (1963) reported that cucumber mosaic virus infecting vegetable marrow was transmitted by Aphis gossypii and Aphis craccivora. Kaiser and Danesh (1971) also could obtain transmission of CMV infecting Cicer arietinum (chickpea) by Aphis craccivora in a stylet-borne manner. However, failure to transmit viruses infecting cucurbits by Aphis craccivora has been reported by Verma et al. (1970), Pillai (1971) and Dubey et al. (1974).

Studies on the vector-virus relationships showed that the vector, Aphis craccivora could acquire the virus with a minimum acquisition feeding period of 5 minutes. It has been reported that the vector can pick up the Melon

mosaic virus with an acquisition feeding of less than 30 seconds (Jaganathan and Ramakrishnan, 1971). As the acquisition feeding period was increased, the efficiency of transmission of the virus was reduced and after long acquisition feeding period, the vector completely lost its ability to transmit the virus (Table 2). The reason for the reduction in the efficiency of transmission after longer acquisition feeding periods had been explained by Watson and Roberts (1939). Day and Irzykiewicz (1954) supported the inhibitor hypothesis. Bradley (1954) reported the formation of salivary sheath during longer feeding intervals which prevented the aphids from becoming infective.

The optimum number of aphids which produced maximum infection was twenty. Further increase of the number of aphids did not increase the per cent infectivity. The data also revealed that a minimum number of five aphids were required for successful transmission. The failure to get infections with single aphids or with groups of 3 is not in agreement with the observations of earlier workers. Hoggan (1933a) mentioned that single aphid (Myzus persicae) could transmit CMV in a non-persistent manner. Kaiser and Danesh (1971) found that single aphid was able to transmit CMV but greater transmissions were obtained only by using comparatively larger number of aphids. The failure to transmit

the virus by a group of three aphids may be due to the fact that snake gourd is not the natural host of the vector or the virus concentration is low to be infective.

The aphids were found to transmit the virus with a minimum infection feeding period of 5 minutes. Jaganathan and Ramakrishnan (1971) observed that the viruliferous aphids can transmit the virus of Melon mosaic with a short infection feeding of less than 30 seconds. The per cent transmission increased with increase in infection feeding period upto 2 hours. But as the time of infection feeding was increased a reduction in the per cent transmission was noticed (Table 4). The possible explanation for this is that most of the aphids that can cause infections do so within the first hour and the increase in the duration of infection feeding reduced the ability of the vector (Nariani and Sastry, 1962).

The efficiency of the vector to acquire and transmit the virus was increased when they were starved prior to acquisition feeding. It was found that starving the aphids for a period of 2 hours increased the efficiency of transmission (Table 5). Fasting for longer periods did not increase the efficiency of transmission. Similar results were reported by Reddy and Nariani (1963) in the case of mosaic disease of vegetable marrow.

Pre-infection fasting of the vector for a period of 30 minutes increased the efficiency of transmission than other periods tried, but prolonged starvation for a period of 3 hours affected the efficiency of transmission and the per cent of infected plants was reduced considerably. The efficiency of the vector to transmit the virus was lost when starved for 4 hours. It was reported that pre-infection fasting of aphids decreased the transmissibility of non-persistent virus of chilli mosaic and longer fasting resulted in considerable reduction in the ability of the vector to transmit the virus (Mariani and Sastry, 1962). Jaganathan and Ramakrishnan (1971) reported that when aphids were starved post-acquisitionally for more than five minutes, there was decrease in the infectivity of Melon mosaic virus but after a period of one hour the viruliferous aphids ceased to transmit the virus.

It was found that the aphids could not retain the virus for longer periods and lost its virulence immediately after feeding on a test plant (Table 7). Mariani and Sastry (1962) observed that single viruliferous aphid lost its infectivity after feeding on a test plant for 10 minutes and could not transmit the virus to the subsequent test plants when serial transfers were made. So the virus is non-persistent and stylet-borne type.

It was observed that the virus was not seed transmitted. Reddy and Nariani (1963) isolated a strain of CMV from vegetable marrow and showed a very low per cent of seed transmission of the virus. It was reported that CMV was transmitted through seeds of Cucurbita maxima (Mukhopadhyay and Saha, 1968). Nagarajan and Ramakrishnan (1971b) reported a Watermelon mosaic virus infecting snake gourd which was seed transmitted to a little extent. However, Verma et al. (1970), Pillai (1971) and Dubey et al. (1974) could not obtain seed transmission of the viruses, Cucumis Virus 2B, a strain of CMV and Cucumis Virus 1 respectively, in snake gourd.

The virus was not transmitted through soil. There are no reports of soil transmission of the viruses affecting cucurbits.

Study of the physical properties showed that the virus has a thermal inactivation point between 70° - 75°C. It was observed that as the sap was treated at higher temperatures, the per cent infection was decreased and the development of symptoms was delayed (Table 8). This may be due to the partial inactivation of the virus by heat treatment. The delay in the development of symptoms may

be due to the time taken by the virus to multiply in the host and manifest the symptoms.

Pillai (1971) reported the T.I.P. of a strain of CMV as 60°C. However, Dubey et al. (1974) found it to be 65 - 70°C for Cucumis Virus 1 and the T.I.P. of Melon mosaic virus was reported as 60 - 63°C by Jaganathan and Ramakrishnan (1971).

The dilution end point of the virus was between 1:5000 and 1:10000. With regard to the D.E.P. the virus resembled the Melon mosaic virus (Jaganathan and Ramakrishnan 1971) having a D.E.P. of 1:5000 to 1:75000.

The longevity in vitro of the virus was 72 - 96 hours at room temperature (32 - 35°C) and 144 - 168 hours at refrigerated conditions (10°C). The virus resembled CMV described by Pillai(1971) in having a longevity in vitro of 72 hours at room temperature. Dubey et al.(1974) reported that the longevity in vitro of Cucumis Virus 1 at 5°C was 8 days. However, Jaganathan and Ramakrishnan (1971) found that the Melon mosaic virus retained infectivity for 8 - 10 days at 5°C.

Out of 33 plant species belonging to 10 families, 8 species of plants in 3 families were found to be susceptible to the virus under study. The virus produced systemic infection of Nicotiana tabucum, Nicotiana glutinosa, Capsicum annuum, Petunia hybrida and Zinnia elegans. Thus,

the host range of the virus is restricted to the plants belonging to Solanaceae and Compositae, in addition to plants of Cucurbitaceae. Chenopodium amarenticolor and Vigna sinensis have been reported as local lesion hosts of Cucumis Virus 1 and its strains. The failure of the virus under study to produce local lesions on such hosts may be due to the strain variation of the virus infecting snake gourd.

It is reported that cucumber mosaic virus can cause mosaic like symptoms on snake gourd (Reddy and Nariani, 1963; Pillai, 1971 and Dubey et al. 1974). Other viruses like Watermelon mosaic virus (Nagarajan and Ramakrishnan, 1971b), Cucumis Virus 3 (Mitra and Nariani, 1965), Cucumis Virus 2B (Verma et al., 1970); Melon mosaic virus (Jaganathan and Ramakrishnan, 1971); Pumpkin mosaic virus (Shanker et al., 1972); Melon Vein-banding mosaic virus (Shankar and Nariani, 1974) and Melon mosaic virus (Nagarajan and Ramakrishnan 1975) have been reported on snake gourd.

Inoculation of snake gourd plants with virus at different developmental stages showed that the vegetative growth and yield are affected severely when infected at an early stage while the effect of virus inoculation was not significant at later stages of infection.

There was no significant effect on the production of leaves when the plants were inoculated at different stages of growth. But there was severe reduction of leaf size with regard to length and width when the plants were inoculated at an early stage. The effect was not pronounced when the plants get infected at later stages of growth. The internodal length was also considerably reduced following earlier infection. The total length of the vine was also reduced when 15 day old plants were inoculated and resulted in stunting of the plant. The plants showed a tendency to produce more branches when inoculated at an early stage (15 day old plants). However, there was no significant difference in the number of branches formed in other cases.

It was observed that early inoculation of plants resulted in complete failure of fruit set due to the excessive shedding of flowers and resulted in complete loss in yield. Subsequent inoculations reduced significantly the number of fruits formed. Vasudeva and Lal (1943) reported that bottle gourd plants infected by Cucumis Virus 3 at an early stage, remained stunted, sparcely flowered and set only few fruits. Similar results have also been reported by Kazda et al. (1975) in the case of green house cucumbers when inoculated with CMV at the cotyledonary stage. It has been reported that the per cent crop loss

varied depending on the growth stage of plants (Hills et al., 1961; Fletcher et al., 1969 and Demski and Chalkley, 1972).

The virus resembled those reported by Pillai (1971) and Dubey et al. (1974) with regard to the symptoms produced on snake gourd.

The virus was transmitted by Aphis gossypii and Aphis craccivora. The latter vector has been reported to transmit the mosaic diseases of vegetable marrow caused by CMV (Reddy and Mariani, 1963). Kaiser and Danesh (1971) found that CMV was transmitted in chickpea by Aphis craccivora. So studies on insect transmission also revealed that the virus occuring in snake gourd is related to CMV.

Studies on the physical properties revealed that the virus is more closely related to Cucumis Virus 1 reported by Dubey et al. (1974) with regard to thermal inactivation point. The dilution end point of the virus closely resembled that of Melon mosaic virus (Jaganathan and Ramakrishnan, 1971). With regard to the longevity in vitro it resembled a strain of CMV (Pillai, 1971).

In the host range studies, it was observed that plants belonging to Solanaceae and Compositae also are susceptable to infection by the virus. These plants were

reported to be susceptible to CMV (Smith, 1937). Other cucurbit viruses have not been reported to infect Solanaceous plants except bottle gourd mosaic virus which caused local lesions on tobacco, Solanum nigrum and S. nodiflorum (Vasudeva and Mariani, 1952). But the bottle gourd mosaic virus differed from the virus under study since the former virus has not been reported to infect Solanaceous plants in a systemic manner.

The virus failed to produce local lesions on Chenopodium amaranticolor or on Vigna sinensis. However C. amaranticolor has been reported as the local lesion host of cucumber mosaic virus and its strains.

The physical properties of the virus indicate that the virus infecting snake gourd is a strain of Cucumis Virus 1. This view is further strengthened by the fact that the virus can infect plants belonging to Solanaceae and Compositae. The findings of Dubey and Mariani (1975) that snake gourd mosaic virus and Melon mosaic virus are serologically related and formed one group i.e. Cucumis Virus 1, also strongly support the view that the virus is a strain of Cucumis Virus 1.

SUMMARY

S U M M A R Y

A mosaic disease of snake gourd (*Trichosanthes anguina* L.) caused by a virus was studied. The disease is widespread in Kerala and is observed in a severe form in Vellayani and nearby localities.

The symptoms of the disease developed as mosaic with irregular chlorotic patches and formation of dark-green blisters on the leaves of infected plants. The size of the leaves were reduced considerably and variously crinkled and deformed. The growth of the plant was retarded and internodes were shortened. The infected plants blossom sparingly and set only few fruits.

The affected portions of the leaf were thinner than healthy areas and the palisade cells in the affected tissues were shortened and loosely packed. Mesophyll cells contain only very few chloroplasts.

The virus was sap transmissible. Use of carborundum as an abrasive along with concentrated sap did not increase infectivity but with standard sap, the infectivity was increased. Use of phosphate buffer did not show any effect on the infectivity of the virus.

The aphids, Aphis gossypii Glov. and Aphis craccivora Koch. were found to be vectors of the virus. The vector-virus relationship using the vector, Aphis craccivora, was studied. It was found that the vector could acquire the virus with a minimum feeding time of 5 minutes and also transmit it with a minimum infection feeding of 5 minutes. Infection was obtained when 5 or more viruliferous insects were used per plant. Starving the aphids before acquisition feeding increased the efficiency of the vector to acquire the virus whereas pre-infection starvation resulted in reduced infection. The virus was not retained by the vector for longer periods and ceased to be infective after one hour feeding on a test plant. So the nature of relationship was found to be of the non-persistent type.

The virus was not transmitted through seed and soil.

The thermal inactivation point of the virus was between 70 - 75°C, the dilution end point between 1:5000 to 1:10000 and longevity in vitro at room temperature (32 - 35°C) between 72 - 96 hours and at refrigerated conditions (10°C) between 144 - 168 hours.

In the host range studies, it was found that the plants Cucumis sativus L., Cucurbita maxima L. Cucurbita

pepo var condensa L., Nicotiana tabacum L., Nicotiana glutinosa L., Capsicum annuum L., Petunia hybrida Vilm. and Zinnia elegans Jacq. were susceptible to infection by the virus. The virus failed to produce local lesions on Chenopodium amaranticolor or Vigna sinensis.

It was found that inoculation of plants at early stages of growth induced reduction in size of leaves, internodal length and total length of the vine, and also resulted in complete loss of yield. There was a tendency to produce more branches following early inoculation. At later stages of infection the effect was not pronounced on yield but leaf size and internodal length reduced. The loss depend on the stage at which the plants are infected.

The virus was identified as a strain of Cucumis Virus 1 (Smith, 1937) based on the studies on symptomatology, mode of transmission, physical properties and host range.

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* Originals not seen. Informations taken from Review of Applied Mycology/Review of Plant Pathology/Other sources.

APPENDICES

A P P E N D

Abstract of

Effect of infection on

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	0.02	1	0.005	2	0.261	3	0.351
Error	28	0.16	28	0.152	27	0.258	26	0.385

I X I

ANOVA

number of leaves.

60th day		70th day		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	0.393	4	0.324	5	0.409	5	0.409	5	0.438
26	0.408	25	0.399	24	0.388	24	0.288	24	0.236

A P P E N D I X

Abstract of

Effect of infection on

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	33.512**	1	22.504**	2	15.726**	3	12.431**
Error	28	0.815	28	1.270	27	0.497	26	0.241

** Significant at 0.01 level.

II

ANOVA

lengths of leaves

60thday		70thday		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	12.868**	4	8.904**	5	6.661**	5	5.830**	5	5.463**
26	0.219	25	0.122	24	0.095	24	0.129	24	0.153

A P P E N D

Abstract of

Effect of infection on

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	15.746**	1	13.083**	2	10.738**	3	11.838**
Error	28	1.107	28	1.465	27	0.894	26	0.388

** - Significant at 0.01 level.

I X III

ANOVA

the width of leaves

60th day		70th day		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	11.060**	4	6.302**	5	5.083**	5	5.947**	5	5.140**
26	0.281	25	0.256	24	0.147	24	0.125	24	0.141

A P P E N D I X

Abstract of

Effect of infection on

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	134.996**	1	107.866**	2	84.983**	3	38.686**
Error	28	11.378	28	6.618	27	3.495	26	1.835

** Significant at 0.01 level.

IV

ANOVA

the internodal length.

60th day		70th day		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	38.096**	4	18.491**	5	13.281**	5	12.214**	5	10.281**
26	1.501	25	0.689	24	1.030	24	0.579	24	0.442

A P P E N D I X

Abstract of

Effect of infection on the

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	4482.67	1	13310.46	2	16323.50*	3	16206.31*
Error	28	3505.60	28	3638.42	27	3715.77	26	4139.56

* Significant at 0.05 level.

** Significant at 0.01 level.

V

ANOVA

total length of the vine.

60th day		70th day		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	19343.43**	4	19913.14**	5	18501.18**	5	19800.38**	5	20329.73**
26	4099.08	25	3700.99	24	3423.85	25	3284.20	24	3163.05

A P P E N D I X

Abstract of

Effect of infection on the

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	2.280*	1	0.970*	2	0.558*	3	0.239
Error	28	0.410	28	0.170	27	0.125	26	0.106

* - Significant at 0.05 level.

** - Significant at 0.01 level.

VI

ANOVA.

number of branches.

60th day		70th day		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	0.052	4	0.064	5	0.133**	5	0.106*	5	0.109*
26	0.042	25	0.039	24	0.029	24	0.035	24	0.035

A P P E N D I XX

Abstract of

Effect of infection on the

Source	20th day		30th day		40th day		50th day	
	df	M.S.	df	M.S.	df	M.S.	df	M.S.
Treat	1	11.842**	1	0.246	2	1.214**	3	1.259**
Error	28	1.098	28	0.343	27	0.118	26	0.108

** - Significant at 0.01 level.

VII

ANOVA

number of flowers.

60th day		70th day		80th day		90th day		100th day	
df	M.S.	df	M.S.	df	M.S.	df	M.S.	df	M.S.
3	1.395**	4	0.702**	5	0.957**	5	1.081**	5	1.218**
26	0.111	25	0.067	24	0.109	24	0.075	24	0.057

APPENDIX VIII

Anova

Effect of infection on the per cent fruit set.

Source	S.S.	df	M.S.	F
Total	797.611	29		
Treat	568.482	5	113.696	11.909**
Error	229.129	24	9.547	

** Significant at 0.01 level.

APPENDIX IX

Anova

Effect of infection on the number of fruits

Source	S.S.	df	M.S.	F
Total:	5.176	29		
Treat	4.020	5	0.804	16.681**
Error	1.156	24	0.048	

** Significant at 0.01 level.

STUDIES ON THE MOSAIC DISEASE OF SNAKE GOURD (Trichosanthes anguina L.)

By

P. J. JOSEPH

ABSTRACT OF THE THESIS

Submitted in partial fulfilment of the

requirement for the degree

MASTER OF SCIENCE IN AGRICULTURE

Faculty of Agriculture

Kerala Agricultural University

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1978

A B S T R A C T

A mosaic disease of snake gourd (Trichosanthes anguina L.) caused by a virus, was investigated which was prevalent at Vellayani and nearby localities.

The symptoms manifested as mosaic and formation of dark green blisters on the lamina was also observed. The affected leaves were severely reduced in size, variously crinkled and malformed. Growth was retarded and internodes were shortened. The infected plants blossom sparingly and set only very few fruits.

The affected portions of the leaf were thinner than healthy areas, palisade cells reduced in size and loosely packed. The mesophyll cells contain only few chloroplasts.

The virus is sap-transmissible and Aphis gossypii Glov. and Aphis craccivora Koch. were identified as vectors of the virus. The virus is neither seed nor soil-borne.

The vector-virus relationship showed that the acquisition feeding and infection feeding were five minutes each, a minimum of five infective aphids were required for successful inoculation, the pre-acquisition fasting increased the efficiency of the vector, the pre-infection starvation

reduced infectivity and the aphids retained the virus only for a short period. The virus is of non-persistent nature.

The thermal inactivation point of the virus was between 70 - 75°C, the dilution end point between 1:5000 to 1:10000 and longevity *in vitro* at room temperature (32 - 35°C) between 72 - 96 hours and 144 - 168 hours under refrigerated conditions (10°C).

Studies on the host range of the virus revealed that eight species of plants belonging to three families were susceptible to infection by the virus. The virus failed to produce local lesions on *Chenopodium amaranticolor* or *Vigna sinensis*.

Early inoculation of the plants resulted in severe reduction in vegetative growth and complete loss of yield whereas late inoculations did not exert significant effect on growth and yield of the plants.

Based on the symptomatology, mode of transmission, physical properties and host range of the virus, the causal virus infecting snake gourd in Kerala is identified as a strain of *Cucumis Virus 1* (Smith, 1937).