

**STUDIES ON YELLOW VEIN MOSAIC DISEASE OF OKRA
(Abelmoschus esculentus (L.) Moench.)**

By

NARESH KUMAR

Dissertation submitted to the Haryana Agricultural University in
partial fulfilment of the requirements for the degree of:

MASTER OF SCIENCE

in

PLANT PATHOLOGY

College of Agriculture

HISAR

1990

DEDICATED


TO

MY RESPECTED PARENTS

CERTIFICATE - I

This is to certify that this dissertation entitled "Studies on yellow vein mosaic disease of Okra (Abelmoschus esculentus (L.) Moench)" submitted for the degree of Master of Science, in the subject of Plant Pathology to the Haryana Agricultural University, is a bonafide research work carried out by Mr.Naresh Kumar under my supervision and that no part of this dissertation has been submitted for any other degree.

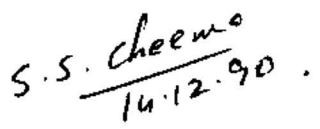
The assistance and help received during the course of investigation have been duly acknowledged.


(Dr.O.P. Kadian) 13/11/91
Associate Professor
Deptt. of Plant Pathology
Haryana Agricultural University
Hisar

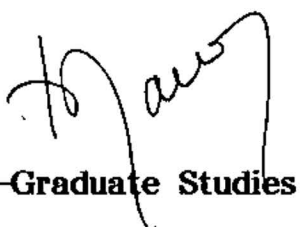
CERTIFICATE-II

This is to certify that the dissertation entitled, "Studies on yellow vein mosaic of Okra (Abelmoschus esculentus (L.) Moench)" submitted by Mr. Naresh Kumar to the Haryana Agricultural University in partial fulfilment of the requirements for the degree of Master of Science in the subject of Plant Pathology has been approved by Student's Advisory Committee after an oral examination on the same, in collaboration with an External Examiner.


14/12/90
Major Advisor


S.S. Cheema
14.12.90
External Examiner


Head of the Department


Dean, Post-Graduate Studies

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Hisar

Nov. 13 ,1990

Nkumar 13/11/90
NARESH KUMAR

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CHAPTER - I

Introduction

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

Abelmoschus esculentus (L) Moench, commonly known as Bhendi, okra or lady finger, is a very popular and extensively cultivated crop in India and abroad. Its tender green fruits are used as vegetable which are rich in vitamins A, B, C and minerals like calcium, iron, potash and others. Okra seeds have 26.0 per cent protein and 18.1 per cent oil contents. Its oil is used for extracting oil for medicinal purposes. Stem and roots of this crop are used for clearing the cane juice in the process of making gur or brown sugar.

Okra crop suffers from several fungal, bacterial and viral diseases. Among those yellow vein mosaic is most severe and destructive and is found in most of the areas in India wherever it is grown. This disease is also observed throughout Haryana.

The casual virus of yellow vein mosaic disease (YVMD) of okra, infects the crop at all stages of plant growth, resulting vein clearing followed by vein chlorosis of leaves and the total chlorosis of plant. When the infection is at early stage, there will be serious adverse affect on quantity and quality of fruits and ultimately results in failure of the crop. The quality of fruit is affected so badly that it fetches very low price in the market.

The extent of losses due to this disease was 49 to 94 per cent at Bangalore in Karnataka (Sastry and Singh, 1974) and 48 to 76 per cent in Tamil Nadu (Chelliah and Murugesan, 1976) depending upon the stage of plant at which the infection takes place. If the infection takes place within

30 days after germination then there is severe reduction in yield. This disease also has the adverse effect on seed production. Earlier the infection, greater is the loss and vice-versa.

Yellow vein mosaic (YVM) of okra has been studied to a limited extent in India and abroad. In Haryana, this disease has not been studied, so far. Information regarding factors affecting disease development, losses caused by the disease and its control measures is scanty.

Considering the growing importance of this disease and lacunae in knowledge about the virus and the disease, it was felt desirable to investigate yellow vein mosaic disease of okra in detail. The studies as presented in this dissertation enabled us to understand the virus and various other aspects of the disease in order to devise the practical methods of control. The present dissertation contains the results of investigations on yellow vein mosaic disease of okra mainly on the following aspects:

1. Symptomatology and evaluation of disease incidence in different areas,
2. Transmission of whitefly,
3. Correlation of meteorological data with whitefly population and disease incidence,
4. Screening of varieties and germplasm and
5. Control measures by using insecticides and oils.

CHAPTER - II
Review of Literature

R E V I E W O F L I T E R A T U R E

A large number of diseases are known to occur on okra crop (Butler and Bisly, 1980; Shamsheer et al., 1988). Important diseases so far recorded in literature are Alternaria leafspot caused by Alternaria spp. (Docea and Coroianu, 1982; Vashney, 1986), capsule rot caused by Ascochyta abelmoschii (Ruscasu, 1985), damping off caused by Rhizoctonia bataticola (Chhabran and Sharma, 1981), dry rot caused by Aspergillus flavus (Jain et al., 1982), fruit rot caused by Cladosporium herbarum (Wadia and Manoharachery, 1979; Kimuro et al., 1982); fusarium wilt caused by Fusarium spp. (Synder and Hansen, 1940); Cercospora leaf spot caused by Cercospora malayensis, C. abelmoschii (Chauhan et al., 1980); powdery mildew caused by Erysiphe cichoroacearum (Sharma et al., 1985; Ganesh and Abdul, 1986); premature fruit abortion caused by Choeneperha cucurbitarum (Adebanjo, 1985); root rot caused by Rhizoctonia bataticola (Wadia and Manoharachery, 1979) and seedling rot caused by Fusarium spp. (Summer et al., 1988). Among bacterial diseases, bacterial blight caused by Xanthomonas compestris pv. esculanti, Pseudomonas syringae (Kimura et al., 1982; Ali and Kulshrestha, 1983; Maharishi and Gupta, 1984) is important disease of okra. Meloidogyne incognita causes root knot in okra (Singh and Sitarmaihi, 1966). Diseases of viral origin viz., yellow vein mosaic (Kulkarni, 1924; Uppal et al., 1940); Kapoor and Verma, 1950), mosaic (Fernado and Udurawana, 1942; Owen, 1946; Lana, 1976; Van Velsen, 1967); leaf curl (Lana, 1976), yellow leaf curl (Shaflik and Asef, 1986); and enation leaf curl (Singh and Dutta, 1986) have also been observed on okra crop.

Among these diseases, okra yellow vein mosaic is most destructive and widespread and was recorded for the first time in 1924 by Kulkarani. He reported that okra crop has been showing typical mosaic symptom with mottling and blistering of leaves, stunting of the plants and caused a great reduction in yield. Uppal et al. (1940) established its viral origin and named it as yellow vein mosaic (YVM). The disease causes clearing of small veins in the beginning and then of the larger ones, followed by extension of yellow to pale yellow areas into mesophyll. The disease was stated to be widespread in Poona district of Maharashtra and Bombay Province, where it causes heavy losses (Uppal et al., 1940; Anonymous, 1942; Capoor and Varma, 1950). This disease was also common and extremely severe in Motel district of Sri Lanka (Newton and Peiris, 1953). The incidence was reported from 50 to 90% in Bihar (Jha and Mishra, 1955). This disease was studied by different workers in other states of India, namely Karnataka (Sastry and Singh, 1973 and Sastry and Singh, 1979), Tamil Nadu (Arumugam et al., 1975), Delhi (Sinha and Chakarbarti, 1978), Punjab (Sharma and Sharma, 1984; 1984) and West Bengal (Khan and Mukhopadhyay, 1985).

Okra yellow vein mosaic virus also infects number of other cultivated and wild crops. A number of workers tried to trace out those hosts. Nariani and Seth (1958) reported that Abelmoschus manihot var. Pungens, A. crinitus, Hibiscus vitifolius, H. panduroformis were immune when eight spp of Abelmoschus and four spp. of Hibiscus were tested for yellow vein mosaic virus reaction. Capoor and Varma (1950) revealed that the host range of this virus is limited to the family Malvaceae, mainly Hibiscus spp. and Althaea rosea. They further explained that a weed namely H. detraphyllus commonly

grown in that locally harbour the yellow vein mosaic virus (YVMV).

Croton sperciflorus and Ageratum conyzoides were found to be additional

host of the virus and A. manihot var. pungens could not be infected but

Zinnia elegans was infected by inoculation (Vasudeva, 1960). Jha and Mishra

(1955) showed that the virus was carried in Malnostrum trichuspidatum.

Civord (1979) inoculated this virus into 287 spp and cvs. belonging to 44 families

and found that 171 plants of 31 families including cotton, coffee, cocae, passion

fruit, groundnut and several market garden plants of the Ivory Coast were

systematically infected. It was also reported by Sharma et al. (1985) that YVM

infected leaves of okra were not susceptible to powdry mildew and if infection

did occur, it was restricted to green areas of okra leaves.

Literature indicates that some attempts were being made on transmission aspect. Capoor and Varma (1950) reported that this virus was transmitted by whitefly (Bemisia tabaci) but the results were found negative when Empoasca devastans, Empoasca spp. and Aphis gossypii were tested. Transmission through sap inoculation, seed and through parasitic activity could not succeed in transmitting the virus. Varma (1952) confirmed the transmission through whitefly and reported that single insect was able to transmit the virus and fifteen whiteflies per plant produced the cent per cent infection. Fasting for over two hours increase the transmission efficiency of the vector (Pruthi and Samual, 1939). The diseased plants were preferred by Aphis gossypii but Amrasea devastans were attracted to the diseased ones (Regupothy and Jayaraj, 1972). Bird and Maramoresch (1977) listed the susceptible cultivated and wild plants in India that serve as food and breeding hosts for the vector Bemisia tabaci.

It can harbour two viruses simultaneously and preserve them for several days without recourse to fresh source of infection (Varma, 1958). Further B. tabaci could carry three different viruses (i.e. yellow vein mosaic of pumpkin and okra, and mosaic virus of Phaseolus luntus), simultaneously for six days, and these findings are similar to earlier findings (Vasudeva, 1960). Haffz et al. (1983) suggested that B. tabaci could be reared from cotton, okra, cabbage and Lantana camera in the field and were released into oviposition cages containing a cut stem of sweet potato in a jar of water. It was reported that visual counting and split cage sampling were more accurate techniques than tap and shake sampling method (Chakarvarthy and Rao, 1985; Horwitz, 1986). Ohnesorge and Rapp (1986) suggested that adults and last two nymphal stages are easy to count than eggs and small larvae. Monitoring of adult was carried out by visual counts or by catches in suction traps or yellow stick traps.

Simple correlation study revealed negative association between disease incidence and relative humidity in all the three periods of crop growth (i.e. 30, 45 and 60 days old crop) and positive association between maximum and minimum temperatures and disease incidence in all three stages (Chelliah and Murugesan, 1975). Further Chelliah and Murugesan (1976) reported a significant increase in the incidence of disease sown in March-May as compared with rest of the year. Singh (1980) revealed the positive correlation between disease incidence and temperature. Shrivanthan (1983) carried out the epidemiological studies of chilli leaf curl disease, mung bean yellow mosaic disease and okra yellow mosaic disease which are transmitted by B. tabaci. The total development of this insect from egg to adult ranged from 65.1 days at 14.9^oC to 16.6 days at 30^oC and average number of eggs laid by one female

was 81 and 72 respectively. The average total life of male was 7.6 and 11.7 days and average total female life was 8.1 and 10.4 days on the corresponding temperatures (Butler et al., 1984). In the gradient study, Khan and Mukhopadhyay (1986) reported that the spread of yellow vein mosaic virus showed a steep rise during the early growth stages of the crop. The extent of final infection dependent on the degree of initial infection.

Losses due to this disease have been recorded in the literature with direct correlation between the age of plant at which infection takes place and losses in yield. Earlier the infection, greater is the loss and vice-versa. Sastry and Singh (1974) observed that the average yield loss due to infection 93.8 per cent when plants were infected within 35 days of germination but reduced to 83.63 per cent and 49.36 per cent in case when the infection occurred 50 and 65 days after germination, respectively. Chelliah and Murugesan (1976) recorded that plants which showed symptoms within 30, 45 and 60 days after sowing, the fruit numbers were reduced to 4, 7 and 8, respectively as compared to healthy plants in which number of fruits were 16. When the plants were infected at various growth stages, highest loss of seed was occurred in the plants showing symptoms on the 33rd days after sowing. The loss was minimum in the plants in which the symptoms appeared after 75th days of sowing and there was no effect on seed germination (Sinha and Chakarabarti, 1978).

Both chlorophyll 'a' and 'b' were destroyed by this disease (Mandhar and Singh, 1971; Ramiah et al., 1972). Mandhar and Singh (1972) reported that the infection reduced the total chlorophyll and total photosynthesis, but there was increase in the respiration rate. They also concluded that carbohydrates are transported from healthy to diseased leaves in which it

accumulated resulting thereby adverse effect on fruit bearing. Singh and Srivastava (1974) reported that the infected fruits contained significantly lower amounts of total reducing sugar and starch and higher amount of protein. Whereas Jamal et al. (1975) found lower total carbohydrates and higher protein. Arumugam and Muthukrishnan (1977) revealed that the phenolic and flavanoids contents in the resistant parents were very high as compared to the susceptible cultivars. Phenolic concentration was higher in the symptomless carrier while flavanoid content was nearly three times higher in the immune but *glycine, histidine and some unidentified amino acids* were present in substantial amounts only in the resistant plants and asparagine only in susceptible ones (Arumugam and Muthukrishnan, 1978). Singh et al. (1985) showed that infection increased the levels of total reducing and non-reducing sugars except in fruits; dextrin, resin, total nitrogen, nitrate in okra leaves, stems and fruits but the total free amino acid is decreased. Johri and Padhi (1985) revealed that level of chlorophyll and carbohydrates in the diseased okra plants declined with the increase in severity of infection and *the nucleic acid levels increased* in the diseased plants. Total protein declined in diseased tissues and its insoluble fraction increased as compared to soluble fraction.

Few attempts have been made in the direction of discovery for source of resistance to this disease by breeding methods and varietal screening for which Shrivanthan (1983) had emphasised its necessity. Nariani and Seth (1958) reported that A. manihot var. pungens and A. crinitus could not be infected by the virus and proved to be highly resistant. Further, study by Singh et al. (1962) found that A. manihot var. pungens could not be used in resistance breeding programme with A. esculentus due to higher sterility in the hybrids.

Arumugam et al. (1975) reported that two accessions of A. (Hibiscus) manihot from Africa and Japan were highly resistant to H. esculentus yellow vein mosaic virus. Thakur (1976) crossed H. esculentus cv. Pusa Sawani and H. manihot spp. manihot grown under condition of natural epiphytotics of this virus and showed that resistance was conditioned by complimentary dominant genes. The crosses between two susceptible cvs of okra and two different wild forms of A. manihot indicated the scope for effective selection because this virus was not associated with any dependent economic character (Arumugam and Muthukrishanan, 1979). Sharma and Dhillon (1983) made hypothesis that resistance is controlled by two complimentary dominant genes with additive effects. Resistance was transferred to Pusa Sawani from A. manihot and resistant segregant from F₂ generation had 71.67 per cent seed fertility and desirable traits (Jambhale and Nerkar, 1983). Madusoodanan and Nazeer (1986) suggested natural hybridization between H. esculentus and H. manihot.

A number of workers screened genotypes of A. esculentus . Swami and Bidari (1976) screened nine genotypes and concluded that two genotypes 15-1-7-4 and 3-1-1-2 were completely resistant to this virus with early and high yielding characters. The selection, 3-2-2-7 and 53-7-9 were found highly susceptible and the rest i.e. 14-2-8-3, 4-1-4-2, 4-3-3-5, 15-1-9-8 and 13-2-11-2 were recorded moderately susceptible. Gunathilagaraj et al. (1977) tabulated thirty seven cvs. out of which none was found resistant to the disease, but three cvs, namely F₃, L.I. Bulk and AE-7 were observed as less susceptible. One resistant accession of A. manihot received from Ghana was shown to be symptomless carrier to this virus (Singh and Thakur, 1979). Chauhan et al. (1981) screened forty six strains and found none of them resistant to this virus with the highest incidence in Pusa long green and lowest incidence in IC 9273.

Sharma and Sharma (1984) identified high degree of symptomless carrier type of resistance in variety EC 31830. They compared Punjab Padmini and found Punjab Padmini superior than others. They also screened seventy four lines and varieties belonging to A. esculentus and related spp. and out of them a line of A. manihot subsp. manihot from Ghana was found resistant, which afterward proved to be symptomless carrier of the virus in grafting test. Salehnaman (1985) screened accessions from twenty nine countries and accessions from Liberia were remained free from symptoms in six sowings, whereas all other accession grown in the same field has 90 to 100 per cent infection. In 1986, Khan and Mukhopadhyay found lowest incidence in SI-1 with highest yield while testing five varieties of okra. Dhankar *et al.* (1989) screened 97 genotypes of okra under natural conditions. Significant differences were observed for disease incidence but none of them was found free from yellow vein mosaic.

Various workers from time to time tried to control the disease by controlling its vector, whitefly (B. tabaci) by using insecticides (Rao, 1959; Anonymous, 1961; Sastry and Singh, 1973; Palaniswamy *et al.*, 1973; Uthamasway *et al.*, 1977; Chakarabarti and Mukhopadhyay, 1977; Khan and Mukhopadhyay, 1985; Singh and Singh, 1989), but none was able to get complete control of the disease. Sastry and Singh (1973) revealed that spraying of insecticides in the initial stages of crop just after germination, is very helpful in reducing the disease incidence. They also reported that four to six sprays of the systemic insecticides not only reduced the whitefly population but also reduced the incidence of this disease to a greater extent. Chakarabarti

and Mukhopadhyay (1977) suggested three applications of Metasystox (0.02%) and recommended sowing of crop in the last week of June. Khan and Mukhopadhyay (1985) recommended soil application of Furatox-10 G @ 15 kg/ha followed by four foliar sprays of Metasystox 25 EC @ 0.03 per cent at an interval of 15 days from date of sowing to reduce the disease incidence and whitefly population. Singh and Singh (1989) found lowest incidence of the disease in phosphamidon (0.02 per cent) followed by methyl demeton (0.025 per cent) and furatox (15 kg/ha). In addition to this they reported that March sown crop escaped the disease for a month over the crop sown in 1st April or 1st May and intercropping with cowpea and mung bean also significantly reduced the whitefly and disease incidence and increased the yield. Khan and Mukhopadhyay (1985) also studied the effect of alternative cultural methods and found yellow mulch polythene significantly delayed the appearance of symptoms. In other experiment they reported that mixed cultivation of pumpkin enhanced both the total incidence of this disease and rate of spread.

Nair (1981) studied the effect of insecticides on cassava mosaic disease and its vector B. tabaci and reported significant decrease in whitefly population, due to insecticide use. For the control of yellow mosaic virus on Vigna radiata, Gupta and Singh (1983) suggested the application of granular insecticides @ 2 kg a.i./ha for both the seasons summer and kharif. Chavan (1984) evaluated the effectiveness of several systemic insecticides for the control of B. tabaci against tobacco leaf curl and spraying of oxydemeton methyl @ 0.2 kg a.i./ha, thiometon @ 0.2 kg/ha and dimethioate @ 0.18 kg/ha, once in the nursery and three times in field, were found effective. Rathor and Agnihotri (1985)

compared five insecticides in field of moth bean (Vigna acontifolia) against yellow mosaic virus and found Rogor as most effective followed by Dimecron, Metasystox, Ekatox and melathion. Horwitz (1986) observed that the repeated use of chemical treatments resulted in creation of resistance in insects and highly fecund strain of the pest is developed in Sudan.

CHAPTER - III
Materials and Methods

M A T E R I A L S A N D M E T H O D S

The present study was conducted during summer and kharif, 1989 and summer, 1990 at the Plant Pathological Research Area, Haryana Agricultural University, Hisar.

3.1 **Symptomatology**

The symptoms were observed in the screen house after inoculation by whiteflies as well as in the field.

3.2 **Disease survey**

Survey of yellow vein mosaic disease of okra was done during summer and kharif, 1989 in ten villages nearby Hisar. In the summer, evaluation of disease incidence was done in May-June and during kharif in the month of September-October. In each village two to three fields were surveyed. In every field, five sites of 25. sq. meter each were selected randomly. The number of plants showing the symptoms of YVMD along with the total number of plants were counted. In addition to this, evaluation of disease incidence in different districts of Haryana was done during kharif, 1989 in the month of September. In all the districts, four to five locations were randomly selected and at every location, four to six sites of 25 sq. meter each were evaluated for disease incidence by counting number of (YVM) diseased plants along with the total number of plants. The per cent disease incidence was calculated by the formula given below:

$$\text{Per cent disease incidence} = \frac{\text{Number of diseases plants}}{\text{Total number of plants observed}} \times 100$$

3.3. Transmission

For transmission test, the okra plants were grown in pots in the screen house. The seeds of okra cultivar 'Pusa Sawani' were sown after treating with Bavistin (2 gm/kg of seed). The plant which found healthy among all the plants in one pot were selected for inoculation while rest of the plants were eradicated. The whiteflies were multiplied on healthy plants of cotton. Whenever needed, the required number of whiteflies were carefully picked from the cotton plants on which they were maintained with the help of Aspirator. Thereafter, they were given pre-acquisition fasting for three to four hours before transferring them to diseased plants of okra to acquire the virus. The virus acquisition feeding was given for 20-24 hours.

3.3.1 Relation to the number of whiteflies to percentage infections

The viruliferous whiteflies already fed on diseased plants were used in this experiment. The required number of viruliferous whiteflies i.e. 1,3,5,10,15 were allowed to fed on healthy test plants of okra cv Pusa Sawani for 12-14 hours. After that a spray of Metasystox 25 EC (0.025 per cent) was done on these plants to kill the vector. The test plants were then removed to another screen house and watched for appearance of the symptoms. Uninoculated checks or control plants were kept along with the test plants used for inoculation test.

3.3.2 Feeding period required by the whiteflies to transmit the virus

Group of fifteen to twenty viruliferous whiteflies that had been fed for 20-24 hours on diseased plants were used. These were allowed to

feed on the test (healthy) okra plants for 1,2 and 3 hours. Thereafter test plants were sprayed with 0.025 per cent Metasystox 25 EC to kill the insects and kept for observation in insect proof screen house.

3.4 EFFECT OF TEMPERATURE AND HUMIDITY ON WHITEFLY POPULATION AND DISEASE INCIDENCE

Okra cultivar 'Pusa Sawani' was sown in summer 1989, kharif 1988 and summer 1990. The effect of temperature and humidity on whitefly population and disease incidence was studied under field conditions. All the agronomical practices for raising a good crop were followed. The whitefly population and disease incidence was recorded at 10 days interval after sowing. Whitefly population was counted directly on five leaves i.e. two at the bottom, two at the middle and one at the top early in the morning when they were less active. The disease incidence was calculated by the formula.

$$\text{Per cent diseased} = \frac{\text{Number of diseased plants}}{\text{Total number of plants observed}} \times 100$$

Data on temperature and humidity were collected from Meteorological Laboratory of Haryana Agricultural University, Hisar and thereafter correlated with whitefly population and disease incidence.

3.5 ESTIMATION OF LOSSES IN YIELD

Okra cultivar 'Pusa Sawani' was sown during kharif, 1989 the plot measuring 200 sq. meter at the Plant Pathology experimental area, H.A.U. Hisar. After 35 days of sowing all the plants showing symptoms of yellow vein mosaic were tagged and properly labelled in the field. In the 2nd stage, the plant showing symptoms after 35 days and before 50 days of sowing were tagged and

properly numbered. Similar tagging was done on the plants showing symptoms between 50 and 65 days of sowing. The plants which remained free from yellow vein mosaic disease symptoms were tagged as healthy plants (control). The following observations were recorded:

- i) Plant height (cms): Plant height was measured from the ground level to the apex of main shoot at the time of last picking.
- ii) Number of leaves: Leaves were counted visually on the last picking.
- iii) Number of fruits per plant: Number of fruits of each picking was added and average per plant worked out.
- iv) Fruit size: Twenty five marketable size fruits ~~per plant~~ were observed for measuring length from the base to the tip by using scale.
- v) Total yield per plant (gms): Weight of fruits per plant was recorded at each picking and average was worked out on the basis of twenty five plants.
- vi) Percentage loss in yield: It was worked out by the formula given below:

$$\text{Percentage loss in yield} = \frac{\text{Yield of healthy plants} - \text{Yield of diseased plants}}{\text{Yield of healthy plants}} \times 100$$

3.6 REACTION OF DIFFERENT GENOTYPE OF OKRA TO YELLOW VEIN MOSAIC VIRUS

For the evaluation of resistance/susceptability against yellow vein mosaic, twenty six genotypes of okra were sown in the field. Sowing was done in completely randomized block design with three replications. Each genotype was sown in three rows of three meter length spaced at 45 cm apart,

Table 1. Determination of resistance/susceptibility of different okra varieties/germplasm to yellow vein mosaic disease

Symptom**	Symptom severity grade	Response value	Co-efficient* of infection (per cent disease development)	Overall reaction	Reaction symbol
1. No symptom	Nil	0.00	0.00	Highly resistant	HR
2. Plants showing mild symptoms- basal half of primary veins green, mild yellowing of arterial half of primary veins, secondary veins and veinlets.	+	0.25	1-25	Resistant	R
3. Veins and veinlets turn completely yellow-Interveinal area green and normal.	++	0.50	25.1-50	Moderately susceptible	Ms
4. Pronounced yellow of veins and veinlets, 50% of the leaf lamina turn yellow, fruits exhibit slight yellowing.	+++	0.75	50.1-75	Susceptible	S
5. Petiols, veins, veinlets, interveinal area turn yellow in colour, leaves, start drying from the margin, fruits turn yellow in colour.	++++	1.00	75.1-100	Highly susceptible	HS

$$\text{Per cent disease} = \frac{\text{Number of diseased plants}}{\text{Total number of plants observed}} \times 100$$

*=Coefficient = Per cent disease x Response value **Fig.1.

accommodating thirty plants in each plot. Infector row of Pusa Mukhmal was planted between the rows of genotypes of okra and around the whole plot. No spraying was done for control of any insect or disease except seed treatment with Bavistin (2 gm/kg of seed) before sowing. In each plot, 10 plants were observed for recording the data for disease incidence. First observation was taken when the plants were 45 days old followed by second observation at 60 days and third observation just before the last picking. The plants which showed disease free reaction in the field were further screened in glass house where sufficient number of whiteflies were released from okra diseased plants with the help of Aspirator. The sowing of germplasm was done in pots accommodating three to four plants in each pot. The pots were placed in glass house at the time of inoculation. Inoculation was done at the stage of 15-20 days after sowing. Plants were observed for symptom expression and disease incidence was estimated. The grading was done as shown in Table 1. Symptom severity grades designed with + sign were given on the basis of visual observations. Each symptom severity grade was given arbitrary value and designed as response value. The co-efficient combined the amount of infection and severity of disease, and was considered as disease development.

3.7 CONTROL OF DISEASE BY INSECTICIDES AND OILS

The experiment was conducted in the simple randomized block design during kharif, 1989 in the research area of Plant Pathology Department, Haryana Agricultural University, Hisar. Certified seeds of variety 'Pusa Sawani' were sown in eleven plots in three replications, each has an area of 9 sq. meter

(3 x 3 meter) consisting of about 40 plants. The insecticides and oils with their concentration tested in this experiment are as under:

Number of Insecticides/oils	Concentration/Quantity applied
1. Phorate (Thimet) 10 G	12.5 kg/ha
2. Carbofuron (Furadan) 3 G	40 kg/ha
3. Dimethoate (Rogor) 30 EC	.03 per cent
4. Oxydemeton methyl (Metasystox) 25 EC	.025 per cent
5. Phosphamidon (Dimecron) 85 WSC	.028 per cent
6. Formathion (Anthio) 25 EC	.025 per cent
7. Fenitrothion (Folithion) 50 EC	.05 per cent
8. Malathion (Malahit) 50 EC	.08 per cent
9. Mustard oil	1.3 per cent
10. Kerosene oil	1.3 per cent
11. Check (Control)	Tap water

Granular insecticides were applied by side dressing while rest of the insecticides were applied as foliar sprays, by mixing them in known quantity of water to obtain the desired concentration. The oil suspension was prepared in 0.15 per cent surf soap solution in water. The first spray was applied 15 days after sowing and the last application was just fifteen days earlier to the first picking. In control plain water was sprayed.

The effect of these insecticides was determined on the basis of per cent disease, whitefly population and fruit yield. The population of whitefly was recorded as per procedure given by earlier workers

(Sastry and Singh, 1973; Chakrabarti and Mukhopadhyay, 1977). Five plants in each replication were selected randomly. Whitefly population was counted on five leaves on each plant (two at the bottom, two at middle and one at top). These were counted directly early in the morning when whiteflies were less active with the help of hand lens.

Disease incidence was determined by the formula given in Disease Survey. Weight of marketable fruits of ten plants was added at each picking and yield per ha. was worked out. All the agronomic practices were followed for raising good crop in all the plots except for the application of insecticides which were different for each set of plot.

CHAPTER - IV

Results

R E S U L T S

The present studies were carried out to get an understanding about the YVM and to design control measures. The results were discussed as under:

4.1 Symptomatology

The symptoms due to infection by YVMV on okra were observed during transmission in the screen house and in the field are as indicated below:

First visible symptoms appeared on the fully developed leaf on second node and developing leaf on third node in the form of clearing of veins which usually starts at various points near the leaf margin and extended into most of the leaf lamina. New leaves emerging thereafter showed a *homogenous interwoven network* of yellow veins enclosing islands of green tissue within. The chlorosis which in the beginning was confined to the veins and then gradually extended into the mesophyll and occasionally a young developing leaf was completely turned chlorotic except for a few patches of green tissue scattered over the leaf surface. The petiole and the stem also became chlorotic. There was general dwarfing due to retardation of growth. The cotyledonary leaves and leaf on first node did not develop any symptom of the disease even if the disease had developed upto chronic stage. The fruits produced by the diseased plants were smaller in size and chlorotic as compared to the healthy ones (Fig.1 Plate 1,2,3).

4.2 Evaluation of disease incidence in different areas

The data on disease incidence of YVM on two popular okra cultivars Pusa Sawani and P-7 nearby Hisar during summer and kharif, 1989 is given

EXPLANATION OF PLATE 1

(on back side)

PLATE 1

Fig. 1.0 = Okra leaf (Healthy) showing no symptoms of yellow vein mosaic disease.

Fig. 1.1 = Okra leaf showing mild symptoms of yellow vein mosaic disease.

PLATE 1



FIG. 1.0



FIG. 1.1

EXPLANATION OF PLATE 2

(on back side)

PLATE 2

Fig. 1.2 = Okra leaf showing moderate symptoms of yellow vein mosaic disease.

Fig. 1.3 = Okra leaf showing severe symptoms of yellow vein mosaic disease.

Fig. 1.4 = Okra leaf showing very severe symptoms of yellow vein mosaic disease.

PLATE 2



FIG.1.2



FIG.1.3



FIG.1.4

PLATE 2

Fig. 1.2 = Okra leaf showing moderate symptoms of yellow vein mosaic disease.

Fig. 1.3 = Okra leaf showing severe symptoms of yellow vein mosaic disease.

Fig. 1.4 = Okra leaf showing very severe symptoms of yellow vein mosaic disease.

EXPLANATION OF PLATE 3

(on back side)

PLATE 3

Fig. 1.5 = Okra fruits from yellow vein mosaic diseased plants (Left) and from healthy plant (Right).

Fig. 1.6 = Plant of okra affected by yellow vein mosaic disease.

PLATE 3



FIG.1.5



FIG.1.6

in Table 2 (Fig.2) whereas the data from different districts of Haryana on this aspect during kharif, 1989 has been given in Table 3 and Fig.3.

It is evident from Table 2 that the disease incidence in different villages nearby Hisar during summer, 1989 was between 10.00 and 28.00 per cent on cv. Pusa Sawani with district average 21.01 per cent. During the same period and locations the disease incidence on cv. P-7 was found upto 1.10 per cent with district average 0.41 per cent. During kharif, 1989, the disease incidence on cv. Pusa Sawani was from 27.00 to 62.33 per cent with district average of 48.05 per cent and on cv. P-7 it was from 0.0 to 2.40 per cent with the district average 1.03 per cent (Table 2). Disease incidence was less in cv. P-7 (1.03 and 0.41 per cent) than Pusa Sawani (48.05 and 21.03 per cent) and it was more in kharif season (1.03 and 48.05 per cent) than summer season (0.41 and 21.03 per cent) during the year 1989 (Table 2).

It is evident from the data in table 3 that during kharif, 1989 on cv. P-7, the disease incidence was maximum in Sonapat district (2.40 per cent) followed by Kurukshetra (2.25 per cent), Ambala (2.10 per cent), Karnal (2.05 per cent), Jind (1.80 per cent), Rohtak (1.75 per cent), Gurgaon (1.20 per cent), Faridabad (1.15 per cent), Sirsa (1.10 per cent), and Hisar (1.0 per cent). The cv. P-7 was free from symptoms of this disease in Bhiwani and Mohindergarh districts.

In cv. Pusa Sawani the disease incidence was maximum in Kurukshetra i.e. upto 68.50 per cent and minimum in Mohindergarh i.e. 26.0 per cent (Table 3, Fig.3). The districts namely Sonapat, Karnal, Ambala, Rohtak, Jind,

Table 2. Incidence of yellow vein mosaic of okra in different villages of Hisar district during summer 1989 and kharif 1989 on cultivars Pusa Sawani and P-7

Sr.No.	Name of villages	*Per cent disease incidence			
		Summer 1989		Kharif 1989	
		Pusa Sawani	P-7	Pusa Sawani	P-7
1.	Anipura	25.6	0.5	58.00	1.0
2.	Dhani	12.37	0.0	50.33	0.33
3.	Dhana Kala	22.00	1.0	62.33	2.40
4.	Dhana Khurd	21.00	0.0	52.50	0.20
5.	Dhiranwas	20.00	0.0	27.00	0.0
6.	Hansi	24.50	0.75	41.00	2.0
7.	Hisar	20.33	0.40	50.33	1.05
8.	Mirka	28.00	0.00	40.00	0.0
9.	Satroad	26.5	1.10	57.00	2.40
10.	Siswala	10.00	0.40	42.00	1.0
Average		21.03	0.41	48.05	1.03

$$\text{*Per cent disease incidence} = \frac{\text{Number of diseased plants}}{\text{Total number of plants observed}} \times 100$$

Table 3. Incidence of yellow vein mosaic disease in different districts of Haryana during kharif, 1989 on okra cvs. Pusa Sawani and P-7

Sr.No.	Name of districts	Per cent disease incidence*	
		Pusa Sawani	P-7
1.	Ambala	64.50	2.10
2.	Bhiwani	30.00	0.0
3.	Faridabad	47.25	1.15
4.	Gurgaon	48.00	1.20
5.	Hisar	47.50	1.00
6.	Jind	58.20	1.80
7.	Karnal	66.25	2.05
8.	Kurukshetra	68.50	2.25
9.	Mohindergarh	26.00	0.0
10.	Rohtak	59.25	1.75
11.	Sirsa	46.50	1.10
12.	Sonepat	68.00	2.40

*Per cent disease incidence = $\frac{\text{Number of diseased plants}}{\text{Total number of plants observed}} \times 100$

FIG:2. OCCURRENCE OF YELLOW VEIN MOSAIC DISEASE ON OKRA CV. PUSA SAWANI AND P-7 IN DIFFERENT VILLAGES OF HISAR DISTRICT DURING THE SUMMER AND KHARIF SEASON OF YEAR 1989

% Disease	SUMMER		KHARIF	
	Pusa Sawani	P-7	Pusa Sawani	P-7
NIL	○	△	□	◇
1-25	○	△	◻	◊
26-50	⊙	△	◻	◊
51-75	⊕	△	◻	◊
76-100	●	▲	◼	◆

Road ———

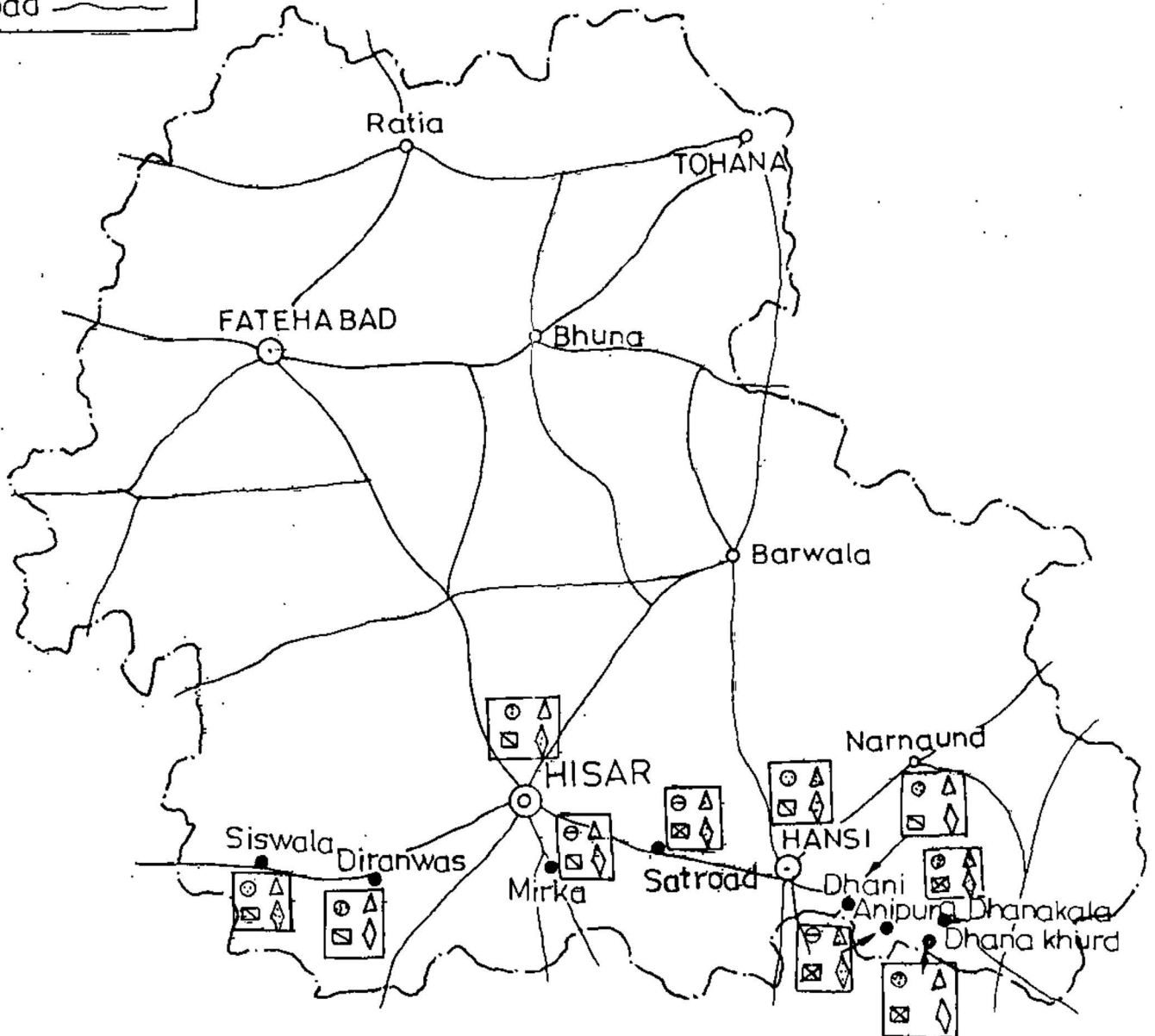
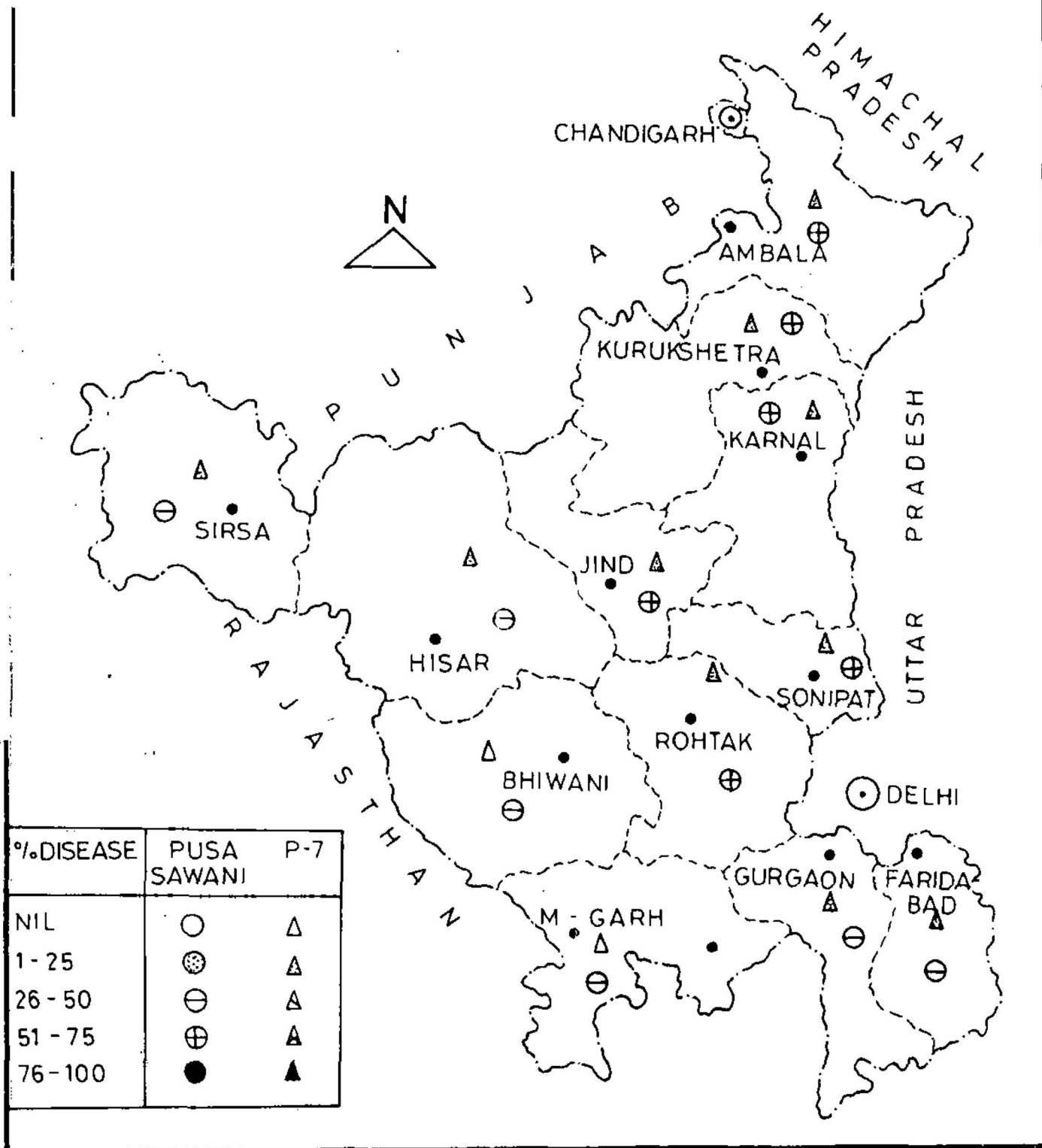


FIG.3. OCCURRENCE OF YELLOW VEIN MOSAIC DISEASE ON OKRA CV. PUSA SAWANI AND P-7 IN HARYANA DURING THE KHARIF SEASON OF THE YEAR 1989



Gurgaon, Hisar, Faridabad, Sirsa and Bhiwani showed the disease incidence to the tune of 68.00, 66.25, 64.50, 59.25, 58.20, 48.00, 47.50, 47.25, 46.50 and 30.00 per cent, respectively. The occurrence of disease in all the districts was more in cv. Pusa Sawani as compared to cv. P-7.

4.3 Transmission

The data on the transmission tests are presented in Tables 4 and 5. Table 4 indicates that single whitefly could transmit the virus upto 20.0 per cent. The highest (70 per cent), transmission was observed in case of the group of fifteen viruliferous whiteflies per plant. The group of ten, five and three viruliferous whiteflies per plant could transmit the virus upto 60.0, 53.33 and 46.66 per cent, respectively. The result of feeding period tests is presented in Table 5 which indicates that feeding period required by whitefly for 70 per cent transmission was three hours. The transmission in one hour and two hours feeding period was 40.0 and 50.0 per cent, respectively.

4.4 Effect of temperature and humidity on whitefly population and disease incidence

Meteorological data on temperature and humidity and the observations on whitefly population and disease incidence during summer, 1989, kharif, 1989 and summer, 1990 on Okra cv. 'Pusa Sawani' under natural conditions are presented in Tables 6, 7 and 8, respectively.

The data indicate that when the average maximum temperature was around $36 \pm 2^{\circ}\text{C}$, average minimum temperature was around $25 \pm 2^{\circ}\text{C}$ and average relative humidity (RH) was more than 40.00 per cent, the whitefly population and disease incidence were highest with more

Table 4. Transmission relationship between whitefly population and yellow vein mosaic disease incidence

Number of whiteflies per plant	Total number of plants inoculated	Number of plants infected	Percentage infection
1	15	3	20.0
3	15	7	46.66
5	15	8	53.33
10	10	6	60.0
15	10	7	70.0

Table 5. Effect of whitefly feeding period in the transmission efficiency of okra yellow vein mosaic disease

Feeding period on test plants (Hrs.)	Total number of plants inoculated	Number of plants infected	Percentage infection
1	10	4	40.0
2	10	5	50.0
3	10	7	70.0

Table 6. Effect of temperature and humidity on whitefly population and disease incidence on okra cv. Pusa Sawani during summer, 1989

Date of observation	Temperature and relative humidity				Average percentage relative humidity	Number of whiteflies per plant	Percentage disease incidence
	Maximum temperature		Minimum temperature				
	Range	Average	Range	Average			
1.3.89 - 10.3.89	24.1-31.2	26.86	4.0-16.7	7.02	59.2	0	0
11.3.89 - 20.3.89	27.2-30.0	28.46	8.9-15.6	12.05	62.7	1.5	0
21.3.89 - 30.3.89	22.5-32.0	28.59	10.5-17.4	13.85	63.05	2.85	1.5
31.3.89 - 9.4.89	27.4-37.6	30.76	7.5-19.0	12.56	53.3	4.05	10.25
10.4.89 - 19.4.89	31.4-38.4	35.13	11.0-16.0	13.55	41.50	6.85	15.75
20.4.89 - 29.4.89	35.7-39.6	37.14	13.3-21.0	16.64	40.25	6.80	20.25
30.4.89 - 9.5.89	36.8-40.0	38.0	13.5-25.0	18.89	35.00	6.50	20.25
10.5.89 - 19.5.89	38.3-46.1	42.97	18.6-27.7	22.29	36.30	6.00	20.25
20.5.89 - 29.5.89	40.1-46.0	43.08	18.9-27.0	21.99	20.75	4.00	20.25

Table 7. Effect of temperature and humidity on whitefly population and disease incidence on okra cv. Pusa Sawani during kharif, 1989

Date of observation	Temperature and relative humidity				Average percentage relative humidity	Number of whiteflies per plant	Percentage disease incidence
	Maximum temperature Range	Average	Minimum temperature Range	Average			
7.7.89-16.7.89	34.0-40.7	38.54	20.5-29.2	25.79	56.2	0	0
17.7.89-26.7.89	35.9-41.9	37.7	25.4-28.4	26.96	55.85	2.4	0
27.7.89- 5.8.89	32.5-38.4	35.69	23.6-28.1	25.42	59.7	5.0	3.5
6.8.89-15.8.89	36.3-39.3	38.0	24.5-27.5	26.08	56.75	5.6	5.4
16.8.89-25.8.89	30.6-37.4	35.04	22.7-27.4	25.32	73.35	7.5	19.76
26.8.89- 4.9.89	28.4-34.1	32.13	22.7-24.8	23.40	76.05	8.75	42.0
5.9.89-14.9.89	36.1-37.1	36.72	19.0-24.0	22.72	53.6	8.7	53.75
15.9.89-24.9.89	31.4-38.3	36.76	19.6-26.0	23.44	58.4	8.54	53.75
25.9.89-4.10.89	32.4-37.6	35.15	17.3-21.1	18.87	52.1	8.0	53.75

Table 8. Effect of temperature and humidity on whitefly population and disease incidence on okra cv. Pusa Sawani during summer, 1990

Date of observation	Temperature and relative humidity				Average percentage relative humidity	Number of whiteflies per plant	Percentage disease incidence
	Maximum temperature		Minimum temperature				
	Range	Average	Range	Average			
1.3.90-10.3.90	17.5-25.4	21.38	7.3-12.9	9.81	78.4	0	0
11.3.90-20.3.90	19.0-30.2	23.78	5.2-10.8	6.94	59.8	1.25	0
21.3.90-30.3.90	26.1-33.6	30.21	9.2-30.0	14.74	54.6	2.65	1.65
31.3.90- 9.4.90	20.4-31.6	26.15	7.2-20.5	12.8	52.35	3.45	2.0
10.4.90-19.4.90	23.6-33.2	29.11	15.2-20.2	18.1	51.65	4.0	7.5
20.4.90-29.4.90	31.4-39.5	35.41	17.5-23.8	19.52	43.15	7.0	26.5
30.4.90- 9.5.90	37.4-41.7	39.51	20.1-25.1	21.9	30.6	6.75	26.5
10.5.90-19.5.90	32.2-39.5	36.71	23.6-29.0	25.79	43.5	6.75	26.5
20.5.90-29.5.90	37.5-42.6	40.77	21.9-29.5	25.89	41.85	5.25	26.5

conspicuous yellow vein mosaic symptoms. Data also indicate that when the plants remained exposed to temperature more than 38°C , the whitefly population started decreasing and also had adverse effect on disease incidence and symptom expression. Higher the increase in maximum temperature (beyond 38°C) more is the adverse effect on whitefly population and disease expression. Higher the whitefly population greater was the disease development and vice-versa.

4.5 Estimation of losses in yield

The data recorded on the height of plants, number of leaves and fruits per plant, average length of and yield of fruits and percentage loss in yield are presented in Table 9. The average height of the plants which developed symptoms within 35 days (1st stage), 50 days (2nd stage) and 65 days (3rd stage) after sowing, was 48.0, 81.25 and 125.5 cms, respectively, whereas it was 152.5 cms in healthy plants. There was gradual increase in the height of the plant with the delay in infection. Similarly, the average number of leaves per plant was lowest in the plants infected within 35 days of sowing (7.01) followed by the plant in which the symptoms appeared within 50 days (10.35) and 65 days (25.0) as compared to healthy plants in which average number of leaves per plant was 32.76.

Average number of fruits per plant was 2.9, 4.58, 8.6 and 17.8 in the 1st, 2nd, 3rd stages of infection and in healthy plants, respectively. Length of the fruit picked from the diseased plants in which symptoms appeared within 35 days, 50 days and 65 days were 4.5, 8.7 and 11.65 cms, respectively, whereas in healthy plants the fruit length was 13.95 cms. Yield data (Table 9) indicate that average yield per plant was 236.5 g in healthy plants, which

Table 9. Effect of yellow vein mosaic disease on plant growth and yield of okra cv. Pusa Sawani

Age of crop when symptoms appeared (days)	Average height of plants (cms)	Average number of leaves per plant	Average number of fruits per plant	Average length of fruits (cms)	Average yield per plant (gm)	Percentage loss in yield in comparison to control (healthy)
1st stage (35)	48.0	7.04	2.9	4.5	7.5	96.82
2nd stage (50)	81.25	10.35	4.58	8.7	36.1	84.73
3rd stage (65)	125.5	25.0	8.6	11.65	114.56	51.56
Control (Healthy)	152.5	32.76	17.8	13.95	236.5	-

decreased to 114.56, 36.1 and 7.5 g in plants showed diseased symptoms within 65, 50 and 35 days after sowing resulted in loss of yield to the tune of 51.56, 84.73 and 96.82 per cent, respectively.

4.6 Screening of genotypes of okra to YVM

Results on varieties/germplasms screening of okra to yellow vein mosaic virus under field and artificial conditions are presented in Table 10 and Table 11. Highly resistant (HR), resistant (R), moderately susceptible (MS), susceptible (S) and highly susceptible (HS) varieties/germplasms were identified on the basis as presented in Table 1.

Out of twenty six okra genotypes which were screened under field conditions, fifteen namely HRB-5, HRB-7, HRB-8, HRB-9, HRB-10, HRB-11, HRB-12, HRB-15, HRB-20, HRB-21, HRB-23, HRB-25, HRB-27, HRB-28 and HRB-41 were highly resistant. One (P-7) was found resistant, five (Ganga cauvery, Parbhani Kranti, PB-57, Punjab Padmini and Sel-10) were moderately susceptible, three (AE-112, HB-57 and Pusa Sawani) were susceptible and two (AE-113, Pusa Mukhmali) were highly susceptible.

The results of fifteen genotypes of okra which were free from disease symptoms in field were further screened in screen house by artificial inoculation are presented in Table 11. None of the genotypes was found to be highly resistant, whereas nine namely HRB-5, HRB-8, HRB-9, HRB-12, HRB-20, HRB-21, HRB-25, HRB-27 and HRB-28 were observed as resistant and six namely HRB-7, HRB-10, HRB-11, HRB-15, HRB-23 and HRB-41 were moderately susceptible.

4.7 Control of YVM by insecticides/oils

The results of the effect of eight insecticides and two oils are presented in Table 12 and 13. A perusal of data (Table 12) indicate that

Table 10. Screening of okra genotypes for resistance/susceptibility to yellow vein mosaic disease under field conditions

Highly resistant (15)	=	HRB-5, HRB-7, HRB-8, HRB-9, HRB-10, HRB-11, HRB-12, HRB-15, HRB-20, HRB-21, HRB-23, HRB-25, HRB-27, HRB-28, HRB-41
Resistant (1)	=	P-7
Moderately susceptible (5)	=	Ganga Cavery, Parbhani Kranti, PB-57, Punjab Padmini, Sel-10.
Susceptible (3)	=	AE-112, HB-57, Pusa Sawani
Highly susceptible (2)	=	AE-113, Pusa makhmali

Table 11. Screening of okra genotypes for resistance/susceptibility to yellow vein mosaic disease under artificial conditions

Highly resistant (0)	=	None
Resistant (9)	=	HRB-5, HRB-8, HRB-9, HRB-12, HRB-20, HRB-21, HRB-25, HRB-27, HRB-28
Moderately susceptible (6)	=	HRB-7, HRB-10, HRB-11, HRB-15, HRB-23, HRB-41
Susceptible (0)	=	-
Highly susceptible (0)	=	-

oxydemeton methyl 25 EC was found the best among all the treatments in reducing the whitefly population and disease incidence followed by dimethoate, malathion, phorate, phosphamidon, Mustard oil, Kerosene oil, fenitrothion, formathion and carbofuran. The effect of all the treatments were statistical significant over control whereas these were at par with each other.

Oxydemeton methyl reduced the whitefly population to the level of 3.66 per plant and disease incidence 21.42 per cent as compared to control in which the whitefly population was 8.0 per plant and disease incidence was 42.77 per cent. It was followed by dimethoate. The maximum number of whitefly i.e. 6.18 per plant with maximum disease incidence i.e. 32.8 per cent was in the treatment of carbofuran. In malathion treated plots, the whitefly population was 4.42 per plant and disease incidence was 22.53 per cent. The data in Table 12 further indicate that oils significantly decreased the whitefly population and disease incidence over check. In check whitefly population and disease incidence was 8.00 per plant and 42.77 per cent which were reduced to 5.25 per plant and 22.57 per cent in the mustard oil treatments and to 5.75 per plant and 23.19 per cent in the kerosene oil treatment.

The data presented in Table 13 indicate that all the insecticides used as spray increased the yield significantly over the check except fenitrothion in which the effect on yield was non-significant over check. The effect of two granular insecticides used as side dressing on the yield was not significant among themselves as well as over the check. Oxydemeton methyl was found to be superior to other insecticides closely followed by dimethoate. The maximum yield (102.6 q/ha) was recorded in plots sprayed with oxydemeton

methyl followed by dimethoate (100.5 q/ha) costing Rs.248.6 and 189.2 per ha, respectively. This gave an increased yield of 16.1 q/ha and 14.0 q/ha over control which in turn gave the net profit of Rs.2971.4 and 2610.8 per ha. The effect of these insecticides closely followed by malathion, phosphamidon, formathion which gave the net profit of Rs.2324, 1908.69 and 1385.6 per ha. Phorate, carbofuran, fenitrothion did not increase the yield significantly over the check (Table 13).

Oil application reduced the yield significantly over control. In other words application of oils was detrimental to yield. The loss of yield was 11.5 q/ha in kerosene applied plots and 6.0 q/ha in Mustard oil treated plots. These caused loss of Rs.1714.8 and 2407.25 per ha, respectively. The maximum return due to application of oxydemeton methyl was Rs.2971.4 per ha, while minimum in the application of fenitrothion (Rs.715.0 per ha) when okra fruits were sold at the rate of Rs.2.0 per kg. There was net loss due to the application of granular formulation. Phorate and carbofuran resulted in a loss of Rs.100 and 1000 per ha, respectively when the cost of insecticides was Rs.500 and Rs.1280/ha, respectively (Table 13).

Table 12. Effect of different insecticides and/oils on the whitefly population and yellow vein mosaic disease incidence on okra cv. Pusa Sawani under field conditions

Sr.No.	Treatments	Concentration per cent or kg/ha	Average whitefly population per plant	Average disease incidence
1.	Dimethoate 30 EC	0.03 per cent	*2.29 (4.28)	** 28.13 (22.23)
2.	Oxydemeton methyl 25 EC	0.025 per cent	2.16 (3.66)	27.56 (21.42)
3.	Phosphamidon 85 WSC	0.028 per cent	2.48 (5.16)	29.13 (23.7)
4.	Formathion 25 EC	0.025 per cent	2.67 (6.18)	33.88 (31.08)
5.	Phorate 10 G	12.5 kg/ha	2.45 (5.05)	29.06 (23.6)
6.	Carbafuran 3 G	40.0 kg/ha	2.67 (6.18)	34.93 (32.8)
7.	Fenitrothion 50 EC	0.05 per cent	2.66 (6.12)	33.08 (29.79)
8.	Malathion 50 EC	0.08 per cent	2.33 (4.42)	28.33 (22.53)
9.	Mustard oil	1.3 per cent	2.5 (5.25)	28.36 (22.57)
10.	Kerosene oil	1.3 per cent	2.61 (5.75)	28.78 (23.19)
11.	Control (check)	-	3.00 (8.00)	40.89 (42.77)

The figures in parentheses are actual value

*Square root transformed value

**Angular root transformed value

C.D. = 0.29
AT 5% LEVEL
SEM± = 0.10

C.D. = 3.22
AT 5% LEVEL
SEM± = 1.09

Table 13. Effect of different insecticides/oils against yellow vein mosaic in relation to yield of okra

Sr.No.	Treatments	Total quantity of insecticides used (kg/ha)	Calculated cost of four sprays (Rs./ha)	Calculated yield (q/ha)	Increase in yield over control (Qtls)	Profit or loss due to insecticides treatments (Rs.)	Net income or less per hectare over control (Rs.)
1.	Dimethoate 30 EC	2.2	189.2	100.5	14.0	2800	2610.8
2.	Oxydemeton methyl 25 EC	2.2	248.6	102.6	16.1	3220	2971.4
3.	Phosphamidon 85 WSC	.73	107.31	96.58	10.08	2016	1908.69
4.	Formathion 25 EC	2.2	334.4	95.1	8.6	1720	1385.6
5.	Phorate 10 G	12.5	500.0	88.5	2.0	400	-100.0
6.	Carbafuran 3 G	.40	1280.0	87.5	1.0	200	-1000.0
7.	Fenitrothion 50 EC	2.2	385.0	92.0	5.5	1100	715.0
8.	Malathion 50 EC	2.2	176.0	99.0	12.5	2500	2324.0
9.	Mustard oil	28.6	514.8	77.5	-6.0	-1200	-1714.8
10.	Kerosene oil	28.6	107.25	75.0	-11.5	-2300	-2407.25
11.	Control	-	-	86.5	-	-	-

C.D. = 7.36
 AT 5% LEVEL
 SEM± = 2.84

CHAPTER - V
Discussion

DISCUSSION

In view of the heavy losses caused by yellow vein mosaic disease to okra crop in Haryana, detailed investigations on various aspects of this disease was carried out. Due to lacunae in information about this disease it was felt desirable to undertake the following studies:

1. Symptomatology and evaluation of disease incidence in different areas
2. Transmission by whitefly
3. Correlation of meteorological data with whitefly population and disease incidence
4. Screening of genotypes
5. Control measures

Symptomatology

The first visible symptoms of YVMD was the clearing of small veins and then larger veins. The chlorosis usually started at various points near the leaf margin. The chlorotic areas were at first confined to the veins, but later they extended into the mesophyll. Occasionally young developing leaves were completely chlorotic except for a few patches of green tissue scattered on the leaf surface. When the disease was severe, the petiole and the stem also became chlorotic and there was general dwarfing of plants. The fruits produced by the diseased plants were smaller in size and chlorotic as compared to the healthy ones. Similar symptoms were observed by Uppal *et al.* (1940) and Capoor and Varma (1950).

Evaluation of disease incidence in different areas

Evaluation of disease incidence in different villages of Hisar district during summer and kharif, 1990 and districts of Haryana during kharif, 1989

revealed that yellow vein mosaic disease of okra was less in summer than kharif. It was less on cv. P-7 than cv. Pusa Sawani. During summer, the disease incidence occurred from 0 to 1.10 per cent on cv. P-7 and 10.0 to 28.0 per cent on cv. Pusa Sawani, whereas it was upto 2.40 per cent in cv. P-7 and 40.00 to 62.33 per cent in cv. Pusa Sawani during kharif, 1989.

In cv. P-7, the disease incidence was maximum in Sonapat district followed by Kurukshetra, Ambala, Karnal, Jind, Rohtak, Gurgaon, Faridabad, Sirsa, Hisar, Bhiwani and Mohindergarh. In cv. 'Pusa Sawani' it was maximum in Kurukshetra followed by Sonapat, Karnal, Ambala, Rohtak, Jind, Gurgaon, Hisar, Faridabad, Sirsa, Bhiwani and Mohindergarh. The disease incidence was more in kharif than summer in both the cultivars. The low disease incidence during summer could be due to very high temperature and very low humidity which may not be congenial for this disease. In addition to this, the possible reason could be that at initial stage of the crop when sowing was done in March, the virus inoculum and whitefly population was very low due to non-availability of suitable hosts and unfavourable climatic conditions during winter season. In the later stages during May-June, the temperature rises very high with very low humidity which may also not be congenial to whitefly survival and virus multiplication. In kharif, the high disease incidence could be due to availability of virus inoculum from summer crop and favourable climatic condition for vector and virus multiplication and spread.

The disease incidence was more in Pusa Sawani than P-7 in all the villages surveyed in Hisar district and other districts of Haryana, which could be due to comparative resistance/susceptibility at the varietal level.

The observations on disease incidence also indicate that Haryana state has two different regions where intensity of yellow vein mosaic differed within the same cultivar. The districts located in North region i.e. Ambala, Kurukshetra, Karnal, Jind, Rohtak and Sonapat showed more disease as compared to the districts situated in South region i.e. Mohindergarh, Bhiwani, Hisar, Sirsa, Gurgaon and Faridabad. The high incidence of disease in Northern region of Haryana may mainly be attributed to comparatively favourable temperature and humidity as compared to districts in South region (Kadian, 1983).

Transmission by whitefly

The data on transmission tests indicate that single viruliferous whitefly per plant could transmit the virus. The highest transmission was found in group of fifteen viruliferous whiteflies per plant followed by ten, five and three whiteflies. The data obtained with okra yellow vein mosaic virus seem to be strikingly similar to that of Orlando and Silbreschmidt (1946) who concluded that transmission of Abuliton virus-I by individual specimens of B. tabaci was low and improved quickly by increasing the number of insects and reached the maximum by using ten insects per plant. Costa and Bennet (1950) have further confirmed these results in the case of Euphorbia mosaic virus.

In another series of experiment, the effect of feeding period on transmission was investigated. The transmission increases with an increase in feeding period on test plants. In three hours feeding period, the transmission was 70 per cent. As the feeding period was reduced to two hours and one hour, the transmission efficiency was decreased to the level of 50.0 and

40.0 per cent, respectively. This confirms the findings of Varma (1952) where he observed that during feeding period of 30 minutes, some insects could transmit the virus but the transmission efficiency was very low, which was further reduced with the decrease in feeding period.

Effect of temperature and humidity on whitefly population and disease incidence

During summer season, the whitefly population and yellow vein mosaic symptoms on okra crop was negligible in the month of March which slightly increased in the first fortnight of April. The highest whitefly population and disease incidence in summer crop was recorded in second fortnight of April. In the months of May-June, whitefly population decreased, whereas the disease incidence remained constant but there was masking in symptom expression and decrease in disease severity. In the kharif season, the pattern of temperature, whitefly population, disease incidence and severity were similar to that of second fortnight of April but the relative humidity was slightly higher. The highest whitefly population and disease incidence during second fortnight of April and in kharif season might be due to congenial maximum temperature $36 \pm 2^{\circ}\text{C}$, congenial minimum temperature $25 \pm 2^{\circ}\text{C}$ and congenial relative humidity more than 40 per cent. The negligible disease incidence in the month of March was due to very less availability of whitefly population and virus inoculum at the initial stage in summer because of very low temperature and non-availability of suitable host for the vector and pathogen in the month of January and February. Khan and Mukhopadhyay (1985) while studying the role of initial inoculum in disease development found the

similar correlation. The decrease in whitefly population and symptom expression in the month of *May-June* might be due to higher maximum temperature above 38°C . The increase in maximum temperature beyond 38°C greater was the adverse effect on whitefly population and disease development. Very low and very high temperatures might have adverse effect on the rate of development and reproduction of whitefly and virus multiplication. This confirms the findings of Trechan (1944) and Butter *et al.* (1983). There was a positive correlation between whitefly population and disease incidence. Higher disease incidence might be due to fast spread of the virus by the vector. Similar reports were also recorded by Chelliah *et al.* (1975).

Estimation of losses in yield

The losses in the yield due to yellow vein mosaic of okra under this study were ranged from 51.56 to 96.82 per cent. Similarly, heavy losses in yield were also observed from other places. Sastry and Singh (1974) recorded the losses in yield from 49.36 to 93.8 per cent in Karnataka while Sinha and Chakarbarty (1978) recorded the loss from 32.85 to 86.13 per cent in the seed production at IARI Regional Station, Karnal. This wide range in the yield losses in the present investigation were due to the effect of the stage of plant growth at which infection occurred. The infection of this disease had also affect on the number of fruits per plant, size of the fruits and other plant parameters like plant height and leaves. Earlier the infection, greater was the reduction in plant height, leaves and fruits per plant, length of fruits and finally loss in yield, which decreased with an

increase in the age of the plant at the time of infection. In this study, it was observed that when the plants were infected within 35 days of sowing the losses were 96.82 per cent. When infection was delayed by next fifteen days, losses were also reduced to 84.73 per cent and when the infection was further delayed by fifteen days (i.e. between 51 to 65 days after sowing) the losses were reduced to 51.56 per cent. These findings are similar with that of by Chelliah and Murugesan (1976).

Screening of genotypes

Out of twenty six genotypes of okra screened under field conditions for susceptibility/resistance to okra yellow vein mosaic virus, fifteen genotypes of okra viz., HRB-5, HRB-7, HRB-8, HRB-9, HRB-10, HRB-11, HRB-12, HRB-15, HRB-12, HRB-15, HRB-20, HRB-21, HRB-23, HRB-25, HRB-27, HRB-28 and HRB-41 were highly resistant, one (P-7) was resistant, five viz., Ganga Cauvery, Parbhani Kranti, PB-57, Punjab Padmini and Sel-10 were moderately susceptible, three viz., AE-112, HB-57 and Pusa Sawani were susceptible and two viz., AE-113 and Pusa Mukhmali were highly susceptible

The highly resistant material under field conditions when inoculated by the vector in screen house, none of the genotypes of okra was found highly resistant and nine genotype viz., HRB-5, HRB-8, HRB-9, HRB-12, HRB-20, HRB-21, HRB-25, HRB-27 and HRB-28 were found resistant and six varieties/germplasms viz., HRB-7, HRB-10, HRB-11, HRB-15, HRB-23 and HRB-41 were moderately susceptible. Some attempts have been made under natural conditions for screening of genotype of okra against yellow vein mosaic by Chauhan et al. (1981) and Dhankar et al. (1989).

Control of disease by insecticides and oils

The spread of virus diseases can be checked by controlling its insect vectors through the application of insecticides either as soil or foliar application (Gates, 1958; Broadbent *et al.*, 1964). Okra yellow vein mosaic can be controlled by soil or foliar application of insecticides and/or oils. Among the insecticides tested in this study, oxydemeton methyl (0.025 per cent) was found most effective with maximum control of disease spread with maximum net profit. It was followed by dimethoate. The present investigations confirms the findings of Sastry and Singh (1973). They also found oxydemeton methyl as second best chemical after Ekatox. The present results corroborate the findings of Chakrabarti and Mukhopadhyay (1977). The net loss in case of granular insecticides was due to their high cost and high application rate. The kerosene and mustard oils were found very effective in reducing the whitefly population and disease incidence and their effect was significant superior over the check. But they also reduced the yield significantly due to their phytotoxic effects. Phytotoxic effects were also reported by previous workers (Peters, 1978 on potato and Walkey and Dancer, 1979 on mustard, broad bean and french bean).

CHAPTER - VI

Summary

S U M M A R Y

Studies on yellow vein mosaic of okra were undertaken with respect to disease evaluation in different areas, transmission, epidemiology, losses, screening of disease resistance and control measures.

The cotyledonary leaves and leaf of first node did not develop any symptoms of this disease. The disease appears in the form of clearing of veins which usually starts near the leaf margin. In severe form, the chlorosis extended into the mesophyll and young developing leaves became completely chlorotic. The petioles, stems and fruits also became chlorotic.

A survey of all districts of Haryana with main emphasis of Hisar district revealed that the disease incidence was less in summer seasons as compared to kharif seasons. It was more in cv. Pusa Sawani than P-7. The districts located in Northern part of Haryana showed more disease incidence in comparison to districts located in Southern part.

A single whitefly per plant could be able to transmit the virus. A group of fifteen whiteflies per plant was found able to cause 70 per cent infection. The transmission efficiency was affected by the feeding period of viruliferous whitefly on healthy test plant. The highest transmission was recorded in three hours feeding period.

Studies on the effect of temperature and humidity in relation to whitefly population and disease incidence showed that maximum temperature around $36 \pm 2^{\circ}\text{C}$, minimum temperature around $25 \pm 2^{\circ}\text{C}$ and relative humidity more than 40.00 per cent were found most congenial for B. tabaci and yellow vein

mosaic development. Higher temperature beyond 38°C in May-June, low temperature in January-February, low vector population and initial virus inoculum in the month of March had adverse effect on B. tabaci population, virus multiplication, disease incidence. B. tabaci population and disease incidence was positively correlated.

The losses in yield due to this disease on okra ranged from 51.56 to 96.82 per cent depending upon the age of the plant at which infection occurred. Earlier the infection, greater was the loss in yield. The affected plants produced poor quality fruits.

Field evaluation of twenty six promising genotypes of okra for resistance to yellow vein mosaic virus indicated that fifteen were highly resistant, one was resistant, four were moderately susceptible and three were highly susceptible. The highly resistant material under field conditions did not behave as highly resistant under artificial conditions. Nine germplasm viz., HRB-5, HRB-8, HRB-9, HRB-12, HRB-20, HRB-21, HRB-25, HRB-27 and HRB-28 were found resistant. Rest of the genotypes appeared susceptible in varying degrees.

Out of eight insecticides and two oils tested in this study either as soil application or foliar application in the field, oxydemeton methyl 25 EC (0.025 per cent) was found most effective with maximum net profit. It was followed by dimethoate 30 EC (0.03 per cent). The granular application was found unprofitable. Though, application of oils decreased the whitefly population and disease incidence but were phytotoxic to plants resulting into significant decrease in the yield.

From the above findings it is concluded that the yellow vein mosaic of okra caused chlorosis of leaves and fruits found in whole Haryana state in both the season i.e. summer and kharif is transmitted by whitefly B. tabaci. The weather conditions during kharif season were favourable for disease development which could cause loss of 51.56 to 96.82 per cent in yield in cv. Pusa Sawani. The disease can be controlled by growing resistant cultivars like P-7 and application of insecticides like oxydemeton methyl 25 EC and/dimethoate 30 EC at an interval of ten days and last application was just fifteen days earlier to the first picking.

CHAPTER - VII

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* Original not seen.

