

**STUDIES ON THE DEVELOPMENT OF F₁ HYBRIDS IN TOMATO
(*Lycopersicon esculentum* Mill.) WITH COMBINED RESISTANCE TO TOMATO
LEAF CURL VIRUS (TLCV) AND A TOSPOVIRUS (T_v) INFECTING TOMATO**

Thesis submitted in part fulfillment of the requirements for the degree of **DOCTOR OF
PHILOSOPHY IN HORTICULTURE** to the
Tamil Nadu Agricultural University, Coimbatore

By

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COIMBATORE -641 003**

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CERTIFICATE

This is to certify that the thesis entitled “**STUDIES ON THE DEVELOPMENT OF F₁ HYBRIDS IN TOMATO (*Lycopersicon esculentum* Mill.) WITH COMBINED RESISTANCE TO TOMATO LEAF CURL VIRUS (TLCV) AND A TOSPOVIRUS (Tv) INFECTING TOMATO**” submitted in part fulfillment of the requirements for the award of the degree of **DOCTOR OF PHILOSOPHY IN HORTICULTURE** to the Tamil Nadu Agricultural University, Coimbatore is a **bonafide** record of research work carried out by **Mr. K. PRADHEEP** under my supervision and guidance and that no part of this thesis has been submitted for the award of any degree, diploma, fellowship or other similar titles or prizes and that the work has not been published in part or full in any scientific or popular journal or magazine.

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Date :

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(K.PRADHEEP)

ABSTRACT

STUDIES ON THE DEVELOPMENT OF F₁ HYBRIDS IN TOMATO (*Lycopersicon esculentum* Mill.) WITH COMBINED RESISTANCE TO TOMATO LEAF CURL VIRUS (TLCV) AND A TOSPOVIRUS (Tv) INFECTING TOMATO

By

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Degree : **DOCTOR OF PHILOSOPHY IN HORTICULTURE**

Chairman : **Dr. D. VEERARAGAVATHATHAM**
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A study was undertaken to develop F₁ hybrids in tomato with combined resistance to Tomato Leaf Curl Virus (TLCV) and the Tospovirus (Tv) prevailing in south India. A total of 30 F₁ hybrids were developed and studied for resistance to both the viruses along with yield and yield contributing traits during January –April 2002 and March –June 2002 under unprotected condition *viz.* without spraying any pesticides. From them eight hybrids were short listed and raised during July -October 2002. The resultant two best performed hybrids were raised again for further confirmation.

Tv incidence was persisting in all the seasons studied while TLCV incidence was maximum in summer season. More than half the hybrids tested showed Highly Resistant reaction for TLCV incidence at 75 DAT. The hybrid H 24 x CLN 2123A exhibited high level of resistance to TLCV (PDI of less than 5.00 per cent) along with delayed expression of symptom. For Tv, almost all the hybrids exhibited first symptom expression at within 45 DAT and none was exhibiting zero per cent disease infection in all the seasons studied. The hybrid combinations H 24 x LCR 1 and H 24 x CLN 2123A

recorded the Tv incidence of 16.90 and 22.66 per cent with fairly high yield suggesting these two are tolerant to Tv in addition to their high level of resistance to TLCV.

Among the parents, H 24 was adjudged as the best general combiner for plant height, number of branches per plant, days to 50 per cent flowering, number of fruits per plant, yield, lycopene, total and OD phenol, TLCV and Tv resistance. This was followed by CLN 2123A which proved as best combiner for six out of 14 characters under study.

The hybrids H 24 x LCR 1, H 24 x CLN 2123A, H 24 x LCR 9, LCR 1 x H 24, LCR 1 x LE 415, LCR 1 x CLN 2123A, CLN 2123A x LCR 9, LE 415 x LCR 1 exhibited a mean total PDI of less than 28 over three different seasons with the mean yield of more than 2.30 kg per plant. In contrast, the susceptible check recorded the PDI of 70 per cent with fruit yield of only 0.45 kg per plant.

Additive gene action was observed to be controlling the traits *viz.* plant height, number of branches per plant, days to 50 per cent flowering, number of fruits per plant, fruit weight, lycopene, acidity, total and OD phenol and TLCV incidence at 75 DAT while non additive gene action was observed in yield, TSS and ascorbic acid. Possible role of modifiers in the Tv incidence was also observed.

Correlation studies indicated that disease incidence (TLCV /Tv/ Total) was negatively correlated with yield, total and OD phenol. Number of fruits per plant and fruit weight exhibited significant positive correlation as well as high direct effect on fruit yield per plant.

Enzyme activity studies revealed clear cut increase in PO and PPO activity upto 96 hours of graft-inoculation. The SDS- PAGE analysis indicated the differences in band intensity of the resistant hybrids with their parents and susceptible check. Isozyme analysis revealed differences in number of isoforms in the resistant hybrids with their parents and susceptible check.

In the confirmatory trial, the hybrids H 24 x LCR 1 and H 24 x CLN 2123A recorded total PDI of 45.71 and 37.50 respectively while the susceptible check recorded

98.15 per cent infection. But the yield of the above said hybrids was almost comparable with that of previous seasons indicating tolerance mechanism operating in them.

The cross combinations *viz.* H 24 x LCR 1 and H 24 x CLN 2123A have been identified as potential F₁ hybrids with field resistance to TLCV and Tv along with high yield. Keeping two flowers per truss in the H 24 x CLN 2123A had resulted in significant improvement in the fruit weight (53.55 Vs. 40.60g) with less reduction in fruit yield (2.55 Vs. 3 .11kg) per plant.

LIST OF ABBREVIATIONS USED

AVRDC	-	Asian Vegetable Research and Development Centre
AOAC	-	Association of official analytical chemists
di	-	Relative heterosis
dii	-	Heterobeltiosis
diii	-	Heterosis over best parent
<i>gca</i>	-	Effect due to general combining ability
GCA	-	Variance due to general combining ability
IAA	-	Indole Acetic Acid
PO	-	Peroxidase
PPO	-	Polyphenol oxidase
PAGE	-	Polyacrylamide Gel Electrophoresis
<i>sca</i>	-	Effect due to specific combining ability
SCA	-	Variance due to specific combining ability
SDS	-	Sodium Dodecyl Sulphate
TSS	-	Total Soluble Solids
°C	-	Degree Centigrade

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CHAPTER I

INTRODUCTION

Tomato (*Lycopersicon esculentum* Mill.) is the second most consumed vegetable in the world behind only potato. It is an excellent cash-earning crop for small farmers (Villareal, 1980). It is eaten fresh or processed and can be stewed, fried, baked or used as juice. It is a good source of iron, vitamin C, vitamin A and vitamin B. It has almost become the part of our daily diet.

Tomato has gained considerable importance especially during the past 50 years in India. The crop is found cultivated throughout the year except during peak summer. It is estimated to occupy an area of 0.52 million ha. with an annual production of 7.42 million tones (Anon., 2004). Though the area under the crop is increasing steadily in this country as a result of increased value of the crop, the productivity is quite low as compared to world figures. Among the constraints on tomato production, those diseases caused by viruses transmitted through insects are the most difficult ones in pest management, affecting the yield drastically.

In south India, it is a common observation that tomato plants are frequently challenged with two serologically different viruses namely Tomato Leaf Curl Virus (TLCV) and a Tospovirus leaving no saleable produce to farmers especially during January to July. Tospoviruses rank one among the ten economically most important plant viruses causing crop losses worldwide to the tune of more than 1 billion dollars (Goldbach and Peters, 1994). There is no remedial measure for a plant once it has become infected unlike for other pathogens where a particular chemical treatment may lower the infection. Their broad host range, efficient transmissibility by vectors, pathogenic variability caused by different strains help them to prove as successful pathogens. Effective control of TLCV and Tospovirus through vector control using with

insecticides is often difficult or quite impossible because their vectors whitefly (*Bemisia tabaci* Genn.) and thrips (*Frankliniella* spp.) respectively are highly mobile and rapidly colonize in fields without the knowledge of tomato cultivators.

Almost all the varieties recommended for cultivation are found to be susceptible or only tolerant to some extent to leaf curl virus. Whenever a cultivar resistant to one virus is introduced, reduction in yield, quality etc. often continues to occur due to the severity of other virus. Only a few tomato lines are available as tolerant ones to each of the viruses combined with good horticultural traits. This tolerant nature is acquired from different wild relatives after the painstaking works of early tomato breeders. Well-known hazards of commonly used pesticides to environment, prohibitory cost and cumbersome methods of their application will adequately prove the worth of resistant breeding in tomato for the control of viral diseases and recognize the same as a core item in the integrated pest and disease management strategies.

In tomato most of the resistant traits are controlled by single dominant genes and it is possible to develop F₁ hybrids accumulating several genes (Rick and Butler, 1956; Kalloo, 1986). In most cases, those interested resistant genes are linked with defects that are masked in the F₁ hybrids. Combining several genes in open pollinated varieties requires more time and breeders need to preserve the exclusivity of their variety. In the F₁ hybrids, the effect of phenomenon of heterosis are expected to show increased vigour, size, early and total yield over better parent helps in securing favourable price of the produce. An appreciable improvement in this direction can be achieved when donor for resistance with good combining ability for yield is identified.

Combining ability analysis following the diallel technique is frequently used for testing the performance of the lines in hybrid condition and also for characterizing the nature and magnitude of gene action involved in controlling a quantitative trait. The array means and the *per se* information being direct estimates based on no assumptions, provide more accurate information regarding GCA and SCA (Kalloo *et al.* 1974). Apart

from this, knowledge on source of resistance, their biochemical background, breeding behaviour, inheritance of disease resistance etc. is of paramount importance in the development of F₁ hybrids. Efficient screening strategies pave way for identifying potential sources of resistance.

Understanding the biochemical basis of resistance to these viral diseases will help the breeder to select different parents and in this connection isozyme variations are proved to be a powerful tool to complement conventional biochemical studies. Genotypic differences that are not always expressed at gross phenotypic level can be suitably interpreted by isozyme variations (Sindhu *et al.* 1984). Hence these markers will serve as a tool for rapid screening in tomato disease resistance breeding programmes.

An eye on yield and horticulturally important traits including quality attributes is a must for a breeder while aiming for disease resistant varieties. This is particularly true in case of F₁ hybrids that should meet out at least acceptable fruit size though not excel with pericarp thickness, good processing attributes etc. Yield being a complex character, is a result of interaction between a number of plant characters among themselves as well as the environment. Hence knowledge on their associations would help in efficient evaluation of genotypes. Correlation and path coefficient analysis would adequately meet out the above objective.

In the light of the above text, an attempt was made to develop F₁ hybrids with combined resistance to TLCV and Tospovirus taking into consideration the following objectives:

1. To evaluate the *per se* performance of parents and hybrids for their combined resistance to TLCV and Tospovirus along with high yield.
2. To estimate the magnitude of heterosis in hybrids
3. To estimate the donor value for the specific traits through the estimation of general combining ability of parents and to estimate specific combining ability of hybrids

4. To identify the superior F₁ hybrids in combination with high field resistance to both TLCV and Tospovirus for commercial exploitation
5. To elicit the nature of gene action for yield, its components and resistance to both TLCV and Tospovirus and
6. To understand the biochemical basis of resistance to both TLCV and Tospovirus.

CHAPTER II

REVIEW OF LITERATURE

Among the viral diseases affecting tomato, TLCV and Tospoviruses particularly Tomato Spotted Wilt Virus (TSWV) are the important disease causing severe losses in the world. In south India, TLCV and a Tospovirus more often confused with TSWV, causing symptoms similar to that of latter damage the crop especially if grown in early spring and summer months, thereby reducing the area under cultivation in recent times. In India, the occurrence of leaf curl disease was reported by Pal and Tandon (1937) and Pruthi and Samuel (1937) whereas TSWV a member of genus *Tospovirus* in 1964 in Nilgiris hills (Todd *et al.*, 1975).

Both TSWV and TLCV have broad host range infecting several other crop species and weeds. Resurgence of TSWV is quite a recent one mainly due to the efficient transmission by the vector western flower thrips *Frankliniella occidentalis* Pergande (Wijkamp *et al.*, 1995). Peculiarity of these viruses is that even a very low number of their vectors may transmit the same to cause economic damage (Reddy *et al.*, 1991; Berlinger *et al.*, 1988 and Makkout and Laterrot, 1983). Moreover these viruses multiply and are retained in the vector for a long time even after moulting and their vectors quickly develop resistance to insecticides (Stansly *et al.*, 1991; Denholm *et al.*, 1996). Hence genetic resistance is one of the few practical strategies to control infection by these viruses since chemical and cultural controls are either non effective or costly and mostly leads to substantial increase in the level of residual toxicity of insecticides used to control them.

From the time when Hedrick and Booth (1908) observed the phenomenon of hybrid vigour in tomato, several investigators have shown that vegetative growth, earliness, more number of fruits per plant, fruit size, fruit quality, resistance to diseases

and unfavourable conditions, yield etc. are possible with F₁ hybrids. The ability of the hybrids to resist diseases will depend upon the degree of resistance found in either one or both the parents. Number of evidences showed that F₁ hybrids have higher degree of resistance to bacterial wilt, early blight, TSWV, TLCV etc. along with other desirable attributes.

In India, lot of confusion exists in adapting the nomenclature of these economically important viruses i.e. TLCV with TYLCV and TSWV with Groundnut Bud Necrosis Virus (GBNV). Of course in breeders' point of view, the symptoms caused by these viruses are more important for genetic study rather than their taxonomy. Hence for practical purposes, these diseases are reviewed wherever applicable. Prerequisite knowledge on importance, distribution, ecology, epidemiology of the viruses, virus - vector - plant relationship, interaction with other viruses, their survival on alternative hosts, related viruses, biochemical basis of host resistance and the inheritance of resistance is inevitable for breeding a variety for multiple virus resistance (Walkey, 1991; Scully and Federer, 1993). Moreover knowledge on clear symptom exhibited due to virus infection including effect of environment, virus isolate, host genotype on that symptom expression and variation in symptomatology is must. Since literature on combined resistance to these viruses for above aspects is not available, these two diseases are reviewed separately.

2.1. TLCV

2.1.1. Importance

The occurrence of tomato leaf curl disease has been reported from East Africa (Storey, 1932), East Indies (Thung, 1932), Sudan (Cowland, 1932), India (Pal and Tandon, 1937; Pruthi and Samuel, 1937), South America (Wolf *et al.*, 1949), Somalia (Cartellani *et al.*, 1981) Jordan Valley (Hassan *et al.*, 1982), Florida (Kring *et al.*, 1991), northern parts of Australia (Condes and Connelly, 1994) and Pakistan (Mansoor *et al.*, 1997). The disease is serious throughout India and yield losses may be as high as 100 per cent (Lal and Sastry, 1961; Sastry and Singh 1973; Datar, 1984; Kalloo, 1988 and Green and Kalloo, 1994). In certain places of north India, tomato cultivation in autumn season has been dispensed with due to high occurrence of this disease (Banerjee and Kalloo, 1987a).

The virus belongs to a genus Begomovirus (family: Geminiviridae) (Chakraborty *et al.*, 2003) and is transmitted by tobacco whitefly *Bemisia tabaci* Genn. which affects the crop during summer (Martelli and Quacquarelli, 1982; Chowfla *et al.*, 1999) and autumn (Martelli and Quacquarelli, 1982 and Kalloo *et al.*, 2003). This virus is highly 'plastic' being able to adapt to a large number of different cultivated plant species, following their transmission from wild or other cultivated hosts (Padidam *et al.*, 1999). Apart from tomato, cassava, tobacco, cotton, pulses and vegetables are the major crops affected by TLCV (Brown and Bird, 1992). Weeds including those belonging to the common genera *Euphorbia*, *Acanthospermum*, *Ageratum* and *Parthenium* were found to be an important reservoir of TLCV (Saikia and Muniyappa, 1989 and Muniyappa *et al.*, 1991).

2.1.2. Symptomatology

According to Raychaudhari and Nariani (1989), affected plants showed stunted appearance and leaves and internodes were greatly reduced in size. Leaves were curled and crowded together. Leaflets were deformed and margins were curled upwards or

downwards. Infected plants were pale and tend to produce stunted lateral branches which resulted in bushy growth of plant. Partial or complete sterility was also observed by them. In case of early infection, no fruit formation took place. Similar observations were made by Ragupathi (1995). Mansoor *et al.* (1997) noticed an additional symptom of vein thickening caused by TLCV transmitted by *Bemisia tabaci* (non-B, biotype K) in Pakistan. Tomato plants infected with the south Indian strains of tomato leaf curl virus exhibit a range of symptoms including leaf curling, vein clearing, stunting of vines, and partial or complete sterility (Anon., 2001).

2.1.3. Epidemiology

Storey (1935) reported that the leaf curl virus was transmitted naturally through whitefly and not carried through seed. Yassin and Nour (1965) reported that maximum leaf curl infection occurred during hottest months i.e. March to June in Sudan. Singh and Sastry (1976) in a periodical survey during 1970 – 71 observed that the disease progressed from February to June when the dry hot weather prevailed (temperature range between 30° and 35°C and relative humidity between 60 and 65 per cent). It was comparatively much less during rainy and winter seasons because of low vector incidence. Butter and Rataul (1978) observed that the virus was transmitted to tomato with 100 per cent efficiency at 33 – 39° C, compared to 30 per cent at 44° C and 10 per cent at 21° C. The percentage of infection reached a peak of 58 and 78 in August and September planting while it was 6 and 8 per cent with respect to planting during December and January respectively (Mazyad *et al.*, 1979). Significant positive correlation between spread of TLCV and population size of vector was noted by Mazyad *et al.* (1979), Ioannou and Iordanou, (1985), Cohen *et al.* (1988), Verma *et al.* (1989), Saikia and Muniyappa (1989), Zouba *et al.* (1993), Borah and Bordoloi (1998) and Kulkarni and Madalageri (2002).

Vijayakumar (1987) observed that the TLCV incidence was high during summer months in crops planted in January, February and March. Saikia and Muniyappa (1989) observed that the incidence of TLCV in tomato ranged from 17.53 per cent during July to November and up to 100 per cent during February to March. According to Nainar (1996) incidence of TLCV disease was highest when tomato was transplanted during February.

Singh *et al.* (1999) noted that maximum and minimum temperature were 28.7 to 30.8 and 15.1 to 22.3° C respectively and those of relative humidity of 88 to 91.3 and 44.6 to 69.6 per cent respectively, no rainfall favored disease spread under Chattisgarh conditions. Holt *et al.* (1999) reported that the incidence of disease in the crop depended primarily on the immigration of vectors from alternative hosts which acted as reservoir of both virus and vector.

2.1.4. Interaction between the strains and with other viruses

Srivastava *et al.* (1975) reported that tomato seedlings when infected by tomato leaf curl and cucumber mosaic virus (CMV) simultaneously developed fern-leaf and leaf curl, extreme stunting and death of plants suggesting a synergistic action. Dhanju and Varma (1986) also reported a complex disease with symptoms of crinkling, dwarfing, yellowing and premature withering of leaves together with stunting and profuse branching of plant widely spread in Haryana and considered that it was due to combined infection by TMV and TLCV.

Singh and Lal (1964) reported the existence of three strains of tomato leaf curl virus based on the symptomatology. Sasikumaran *et al.*, (1979) observed the existence of two distinct isolates of virus, the first one causing only crinkling and curling of leaves and the second one causing curling coupled with vein enation. Condes and Conelly (1994) observed that infection of tomato plants with the TLCV-Au strain or the TLCV-Au D₂ strain induced relatively mild symptoms.

2.1.5. Screening for resistance

2.1.5.1. Screening under field conditions

Many successful resistance screening programmes have been carried out (Yassin and Nour, 1965; Joshi and Choudhary, 1981; Geneif, 1984; Banerjee and Kalloo, 1987a; Saikia *et al.*, 1989 and Singh *et al.*, 1994) in the field relying upon natural virus infection.

Yassin (1985) studied the segregating progenies of tomato lines for TLCV resistance under field conditions by growing susceptible tomato cultivars nearby. Similarly Shoba (1990) screened segregating progenies of different crosses of tomato under field condition by planting a highly susceptible accession LE 125 on all the four sides of the plot. Nainar (1996) and Elakkuvan (1997) evaluated certain interspecific crosses of tomato along with their parents for TLCV resistance. Some of the somaclonal variants were screened for TLCV resistance under field condition by Mala (1999) by interplanting with susceptible variety CO 2 as an inoculum for viral infection. Sankari (2000) screened 36 hybrids along with their parents during summer and *kharif*.

2.1.5.2. Screening under glasshouse conditions

Insect proof glasshouses are preferred wherever there is no possibility of an isolated site distant from possible sources of contamination.

Pilowsky and Cohen (1974) suggested the artificial inoculation of viruliferous whiteflies maintained on *Datura stramonium* plants for testing resistance to TLCV. Hayati (1978) reported on the individual plant inoculation techniques, in which whiteflies were reared on eggplant which were starved for one hour before, allowed to feed on fully infected plants followed by allowing them to feed on test plants. Joshi and Choudhary (1981) screened tomato lines using graft inoculation technique. Kalloo and Banerjee (1990a) screened F₁ hybrids and back cross progenies under screen house, wherein three weeks old plants were artificially inoculated with viruliferous whiteflies. Shoba (1990) screened F₁ and F₂ progenies of tomato by grafting technique. Banerjee and Kalloo

(1991) proposed a method for large scale inoculation and screening for TLCV resistance utilizing an insect proof double door screen house in which whitefly population was continuously maintained by rearing them on brinjal plants. Ragupathi (1995) screened cultivars field-resistant to TLCV by using viruliferous whiteflies at the rate of three insects per plant. He reported that the particular vector number was optimum for 100 per cent infection. Nainar (1996) screened seven accessions of three wild species of tomato by artificial vector inoculation technique. Twenty seedling progenies of somoclonal variants were screened by Mala (1999) for TLCV resistance by grafting method. Thirty six hybrids and 13 parents were screened for resistance to TLCV resistance by graft inoculation method (Sankari, 2000).

2.1.6. Assessment of disease

Since there is a continuous gradient from severe infection to extreme resistance for TLCV disease reaction, different scoring systems were used to measure or estimate intensity of the disease.

Joshi and Choudhary (1981) scored the disease reaction on graft-inoculated plants into four grades *viz.* severe symptoms (Score-4), less severe symptoms (score-3), mild symptoms (score-2) and no symptoms (score-1). Under field conditions, these cultivars were classified into ten classes ranging from 0 – 100 per cent. Geneiff (1984) assigned the scores 0, 1, 2 and 3 for measuring the degree of infection. Hassan *et al.* (1984) assigned disease scoring *viz.* 1 – no visible symptoms, 2 – slight, 3 – moderate and 4 – severe symptoms. Plants assigned score – 1 could be resistant or symptomless virus carrier or susceptible escapes.

Banerjee and Kalloo (1987a) introduced a disease scoring method by combining the amount of disease infection with its severity grade. Five severity grades were assigned based on the intensity of the symptom and for each grade a response value was given. The coefficient of infection was calculated by multiplying the PDI by response

value, then arrived at 0 - 4 as highly resistant, 5 – 9 as resistant, 10 – 19 as moderately resistant, 20 – 39 as moderately susceptible, 40 – 69 as susceptible and 70 – 100 as highly susceptible.

Saikia *et al.* (1989) employed a scale for scoring disease reaction of TLCV in which, the plants which didn't show any symptoms were scored as resistant (F), the plants with yellowing along the margin but no curling as mildly infected (M), the plants with light yellowing on the margins but slight curling and stunting as moderately susceptible (MS) and the leaves with severe curling, puckering and reduced size as susceptible (S). Shoba (1990) followed simple assessment that plants which didn't exhibit any symptoms of TLCV after 40 days of inoculation as resistant. Singh *et al.*, (1994) classified the tomato cultivars following a disease scale based on the percentage of plants infected with TLCV. The grades were highly resistant (0 % infection), resistant (1 – 5 %), moderately susceptible (5.1 – 10 %), susceptible (10.1 – 50 %) and highly susceptible (> 50 %). While Ragupathi and Narayanaswamy (2001) classified the same into six classes *viz.* 0 per cent (Highly Resistant) , 1 – 10 per cent (Resistant), 11 – 20 per cent (Moderately Resistant), 21 – 30 per cent (Moderately Susceptible), 31 – 50 per cent (Susceptible) and 51 - 100 per cent (Highly Susceptible). Since growth conditions have a strong effect on severity of disease symptoms by TYLCV, assessment of the resistance level played by the plant was often misleading. Hence Lapidot *et al.* (2001) developed a scale of differential hosts, which enabled the determination and comparison of level of resistance to TYLCV expressed by resistant tomato plants. The scale comprised seven different homozygous tomato genotypes that exhibited differential levels of TYLCV resistance, ranging from fully susceptible to highly resistant. While the score of each individual resistant genotype indeed changed under different environment conditions, its position on the scale didn't. Thus genotype in question is being inoculated alongside the differential hosts comprising the scale, and within four weeks one can determine relative level of resistance of tested genotype related to its position on the resistance scale.

2.1.7. Sources and inheritance of resistance

Information on genetic resistance to a particular disease is necessary in any disease resistance breeding programme to know whether the resistance is dominant or recessive and controlled by one or a few or many genes. Many viral diseases have been studied sufficiently to show that useful resistance does exist among varieties of host plants or in close relatives of them. A substantial beginning has been made in learning how some of these resistances are inherited in successive generations after hybridization.

Clayberg and Kring (1974) observed that TLCV resistance was controlled by relatively a few genes. From the genetic data obtained from F₁, F₂, F₃ and backcross generations, Pilowsky and Cohen (1974) indicated that resistance was controlled by simple incompletely dominant gene. Joshi and Choudhary (1981) observed susceptibility of F₁ to TLCV hence indicated that resistance in tolerant lines was a recessive character. Mazyad *et al.* (1982) reported resistance to TYLCV in *Lycopersicon hirsutum* was dominant and controlled by more than one gene. Berlinger *et al.* (1983) reported that resistance to TLCV appeared to be polygenic.

Studies by Geneiff (1984) revealed that a single gene controlled TLCV resistance since the reaction of the heterozygous F₁ population to the disease was very similar to that of homozygous resistant parent. The gene action was inferred as complete dominance and the F₂ progenies segregated in a ratio of 3 (resistant): 1 (susceptible) and the backcross progenies segregated in a ratio of 1(resistant): 1 (susceptible).

Hassan *et al.* (1984) studied the inheritance of resistance in interspecific crosses using *L. chilense* and *L. hirsutum* as resistant parents and *L. esculentum* as susceptible parent. The results indicated that resistance for TLCV derived from *L. chilense* seemed to be recessive whereas that from *L. hirsutum* was by more than one gene. Yassin (1985) studied resistance of TLCV in F₁, F₂, F₃ generations of a cross between a cultivated tomato variety and *L. pimpinellifolium* and concluded that the wild relative carried a

dominant factor for resistance. Green (1986) observed that the resistance was partially dominant and controlled by more than one gene.

According to Banerjee and Kalloo (1987b) resistance for TLCV in *L. hirsutum* f *glabratum* B 6013 and *L. hirsutum* was based on epistatic genes, the gene with major effect from *L. hirsutum* f *glabratum* B 6013 and *L. hirsutum* and minor effect from susceptible *L. esculentum* cultivar. Kasrawi (1989) observed that the resistance to TLCV in *L. pimpinellifolium* was controlled by single dominant gene. Muniyappa *et al.*, (1989) reported that F₁ generation of the cross between *L. esculentum* and *L. hirsutum* was susceptible to TLCV indicating dominance of susceptibility.

Shoba (1990) studied the inheritance of F₁ plants from six crosses and found susceptible symptom similar to susceptible parents indicating dominance of susceptibility. Pilowsky and Cohen (1990) opined that tolerance was found to be controlled by five recessive genes in a cross made between susceptible tomato cultivar and a tolerant line M – 60. Wild species *L. chilense* and *L. hirsutum* were found to be remained symptomless and with low levels of viral DNA after 85 days of inoculation while all the other inoculated species were infected and had detectable levels of viral DNA (Zakay *et al.*, 1991). Kasrawi and Mansour (1994) after analyzing F₁, F₂ and backcross populations from the lines derived from *L. pimpinellifolium* showed that resistance to TLCV in these lines was a quantitative trait with some level of dominance. Kegler (1994) reported that the resistance or tolerance to TLCV in several lines of wild species of tomato was controlled by mono- and oligo- or polygenes.

Michelson *et al.* (1994) indicated that tolerance to TLCV was controlled by single major gene *Ty – 1* in wild *L. chilense* which was associated with inhibition of disease symptoms and viral accumulation in inoculated tissues. Nagaraja *et al.* (1996) opined an additive dominant model for variation among the parents, F₁, F₂, F₃ generations for days to TLCV symptom expression in crosses involving Arka Vikas and Arka Saurabh with TLCV resistant *L. hirsutum*.

Friedmann *et al.* (1998) crossed resistant TY – 172 with susceptible lines. The hybrids exhibited milder symptoms and lower virus content than the susceptible parent, yet higher than TY – 172 suggesting a partial dominance of TY – 172 resistance. Upon inoculation of F₂ populations, the number of symptomless individuals appeared in a ratio of ~ 7: 64 suggesting that at least 3 genes *viz.* one partially dominant (AA), the other recessive (bb) and both of them being controlled dominantly and epistatically by a third recessive gene (cc). Vidavsky and Czosnek (1998) analyzed segregation pattern of susceptible, tolerant and resistant of plants of generations from BC₁F₁ to BC₁F₄ of two TYLCV resistant *L. hirsutum* accessions (LA 1777 and LA 386) crossed with *L. esculentum* and suggested that tolerance was controlled by a dominant major gene and resistance by 2 – 3 additive resistance genes.

From the results obtained with the F₂ segregating populations of three wild tomato species *L. chilense*, (LA 1969), *L. peruvianum* (EC – 104395) and *L. pimpinellifolium* (Hirsute) tolerant to TYLCV through a diallel F₁ cross, Vidavsky *et al.* (1998) showed that tolerance from *L. pimpinellifolium* was controlled by one major gene, that from *L. chilense* by two genes and that from *L. peruvianum* three genes with no dominant effect. They suggested that a maximum level of tolerance can be obtained by the additive effect of partly dominant genes from *L. chilense* and *L. pimpinellifolium*. Dharmatti *et al.* (1999) reported non additive gene action for TLCV resistance.

Sources of resistance to TLCV/TYLVCV around world

Sl.No	Source of resistance	SN *	T/R*	Inheritance	N/C*	Reference (s)
1	<i>L. pimpinellifolium</i>					
	LA 121	-	T	Single incompletely dominant gene	-	Pilowsky and Cohen (1974)
	A 1921	A	R	Single incompletely dominant gene (<i>Tlc</i>)	-	Banerjee and Kalloo (1987a)
	LA 121, LA 1582	-	R	Single dominant gene	-	Yassin (1987)
	LA 1342	-	R	-	-	Giordano <i>et al.</i> (1999)
	XXXII-354-a-Silvestra, SI-496, PRS	B	R	-	-	Som (1973)
	P13 (2247), XXII-354-A-Silvestra	B	T	-	-	Joshi and Choudhary (1981)
	IIHR 1942	A	R	-	N,C	Nainar and Pappiah (2002b)
2	<i>L. esculentum</i>					
	EC - 104395	B	R	-	N	Varma <i>et al.</i> (1980)
	Nova, Nematex, HS 110, TI, HS 102	B	T	-	N	Hayati and Varma (1984)
	H 2,11,17, 23, 24	B	R	-	N	Kaloo and Banerjee (1990a,b)
	E 445, DRW 8001, 8006, 8003, 8009, 8005, Saria, W 322F ₁	V	R	-	N	Hussein and Mansour (2001)
	TLB 111, 130, 182	B	R	-	N	Muniyappa <i>et al.</i> (2002)
	H 36, H 86, NDT-VR-60	B	R	-	N	Kulkarni and Madalageri (2002)
	TY - 172, 197, 198, 536	B	R/T	-	-	Lapidot <i>et al.</i> (1997); Friedmann <i>et al.</i> (1998)
	FI 901	B	R	-	-	Vidavsky and Czosnek (1998)
	Multichiltyle 95, Chiltyle 93 - 3	B	R	-	C	Giordano <i>et al.</i> (1999)
	Ty - 52	-	R	Carrying <i>Ty-1</i> gene	-	Anon. (1999)
	CLN 2116A	B	R	Introgression from <i>L.</i>	-	Anon. (1999)

				<i>hirsutum</i>		
	L 5178, 5198, 6802-1, 6803	A	R	-	N	Singh <i>et al.</i> (1994)
	H 24, 36	V/B	R	-	-	Singh <i>et al.</i> (1999)
	BT-3, Arka Alok, Arka Abha	V/B	R	-	-	Mahanta <i>et al.</i> (1998)
	LE-812, LE-376	B	R	-	N	Shoba (1990)
	UPV TY 1, 3, 6, 9, 17, 53	B	R	-	N,C	Pico <i>et al.</i> (1999)
	TY-20	-	R	-	-	Pilowsky and Cohen (1990)
	M-60	-	R	Five recessive genes	-	Pilowsky and Cohen (1990)
3.	<i>L. glandulosum</i>					
	EC- 66002, 66003	A	R	-	-	Varma <i>et al.</i> (1980)
	EC- 68003	-	R	-	-	Saikia <i>et al.</i> (1986)
4.	<i>L. hirsutum</i>					
	PI 127826	A	R	-	C	Banerjee and Kalloo (1987a)
	PI 390658, 390658, 390658	A	R	-	N	Saikia and Muniyappa (1989)
	LA 386, LA 1777	A	R	More than one dominant gene in LA 386		Hassan <i>et al.</i> (1984); Saikia <i>et al.</i> (1986); Muniyappa <i>et al.</i> (1991)
	UPV - 16910	B	R	-	-	Pico <i>et al.</i> (2001)
	LA 1353	A	R	-	N	Ragupathi and Narayanaswamy (2001)
5.	<i>L. hirsutum</i> f <i>glabratum</i>					
	B 6013	A	R	Two epistatic genes interacting complementarily	C	Banerjee and Kalloo (1987b)
	LA 1223	A	R	-	N	Ragupathi and Narayanaswamy (2001)
	LE 1118	A	R	-	N,C	Nainar and Pappiah (2002b)
6.	<i>L. hirsutum</i> f <i>typicum</i>					
	A 1904	A	R	-	C	Banerjee and Kalloo (1987b)
7.	<i>L. cheesmanii</i>					
	LA 1401	-	R	Monogenic recessive	-	Hassan <i>et al.</i> (1984)

8.	<i>L. chilense</i>					
	Line 414-2 x 414 – 1 SIB, LA 267, LA 458, 63L-Tacna, Peru, Line 986-22 x 986-24, 55 L – Antofagaster, Chile	B	R	-	-	Joshi and Choudhary (1981)
	LA 1932, 1938, 1963	B	R	-	-	Pico <i>et al.</i> (2002)
	LA 1967	B	R	-	-	
	LA 1969, 2731, 2737, 2764, 2765, 1970	A	R	-	-	Anon. (1993)
9.	<i>L. peruvianum</i>					Giordano <i>et al.</i> (1999)
	EC – 65980, 65968, 148898, 148897	A	R	-		Varma <i>et al.</i> (1980)
	B 6002/77	A	R	-	C	Banerjee and Kalloo (1987b)
	PI 127830, 127831	A	R	-	-	Saikia and Muniyappa (1989);
	PI 143679, 126944	B	R	-	-	Pico <i>et al.</i> (2002)
	CNPH 784, 786, 787	-	R	-	C	Giordano <i>et al.</i> (1999)
	LA 996- 22 x 996-24 SIB, LA 444, 63L – Chinchu, Peru	B	R	-	-	Joshi and Choudhary (1981)
10.	<i>L. peruvianum</i> f <i>glandulosum</i>					
	B 6005	A	R	-	C	Banerjee and Kalloo (1987b)
11.	<i>L. peruvianum</i> f <i>typicum</i>					
	B 6002	A	R	-	C	Banerjee and Kalloo (1987b)

A- wild species; B- line; V- variety; R-resistant; T- tolerant; N- natural condition; C- controlled condition; SN- Source nature

Nainar and Pappiah (2002a) studied the inheritance of TLCV resistance in eight crosses involving four susceptible varieties and two wild parents under field condition. The F₁ plants of four crosses involving *L. hirsutum* as male parent exhibited infection similar to the susceptible female parent indicating the dominance of susceptibility. The segregating pattern on the F₂ generation of those crosses showed a good fit for 1:63 ratio for field resistance and susceptibility which indicated that there might be three recessive genes to control field resistance which was further confirmed by the segregating pattern of B₂ generation which showed a good fit for 1:7 ratio in all the four crosses. The F₁ plants of other crosses involving *L. pimpinellifolium* as male parent exhibited an intermediate reaction with very mild symptoms of TLCV incidence indicating the incomplete dominant nature of inheritance. Three out of four crosses in the F₂ generation showed a good fit for 1:2:1 ratio for field resistance, intermediate reaction and susceptibility. However, in the B₁ generation, all the four crosses showed a 1:1 ratio for intermediate and susceptibility. But in the B₂ generation, three crosses showed a good fit for 1:1 ratio for field resistance and intermediate reaction, confirming the role of single incompletely dominant gene action. They reported that the genetics of resistance with reference to a particular wild parent was not constant in certain crosses of the same generation indicating that genetics of resistance could vary with the parents hence resulted in variation in the inheritance of genes.

2.2. Tospovirus

2.2.1. Importance

“Tospovirus” is an acronym derived from the name of the group’s most prolific, and first recognized member the TSWV. This genus belongs to the large family Bunyaviridae (Francki *et al.*, 1991; Murphy *et al.*, 1995). TSWV was first described as spotted wilt in Australia (Brittlebank, 1919) and reported to be transmitted by thrips several years later (Pittman, 1927). Samuel *et al.* (1930) showed it to have a viral

etiology. Subsequently the disease has been recorded from Europe, South America, Africa and Asia (Best, 1935). In India, Todd *et al.* (1975) observed this disease first time in tomato cv. Marglobe in Nilgiri hills during 1964. Later on it has been observed in serious form in tomato from southern states (Rao *et al.*, 1980; Sastry 1982; Sabitha *et al.*, 1984 and Harikrishnan *et al.*, 1984), chillies (Bidari and Reddy 1984), urdbean and mungbean (Ghanekar *et al.*, 1979), peas (Rao *et al.*, 1985) and groundnut (Chohan, 1974 and Ghanekar *et al.*, 1979). It ranks one among the ten economically most important plant viruses causing crop losses worldwide of more than 1 billion dollars (Goldbach and Peters, 1994). It has been found to infecting over 100 plant species throughout the world (Chatzivassiliou *et al.*, 2001). Several weeds and perennial ornamentals served as a reservoir of this virus and its insect vector (Watterson, 1986). In India, the most common weeds like *Ageratum conyzoides*, *Cassia tora*. (Reddy *et al.*, 1983), *Portulaca oleracea* (Kalloo, 1986) etc. were found to serve as the same.

Until the late 1980s, TSWV was considered to be the sole member of the group. Increased interest in tospoviruses during last 15 years has led to characterization of several new tospoviruses from Brazil, Japan, India, Taiwan and USA. Apart from TSWV the species recognized and named included Tomato Chlorotic Spot Virus (TCSV), Groundnut Ring Spot Virus (GRSV), Impatiens Necrotic Spot Virus (INSV), Watermelon Silver Mottle Virus (WSMV), Peanut Bud Necrosis Virus (PBNV), Melon Spotted Wilt Virus (MSWV), Peanut Yellow Spot Virus (PYSV) (Pappu, 1997) and Iris Yellow Spot Virus (IYSV) (Cortes *et al.*, 1998). In India, a report on PBNV infecting tomato cultivated in Kerala was available (Jain *et al.*, 2002) which was confirmed by serological as well as nucleo-capsid protein sequence analysis. Sialer *et al.*, (2002) reported 53 – 55 per cent primary nucleotide sequence similarity of TSWV-Southern Italy strain with PBNV and WSMV. In India, apart from TSWV and PBNV, PYSV (Pappu, 1997) and WSMV (Singh and Reddy, 1995) were observed but on different crops.

Currently eight species of thrips have been reported to transmit TSWV (Maris *et al.*, 2003). The prominent species involved in the transmission of TSWV are *Thrips tabaci* Lindl. (Pittman, 1927), *Frankliniella schultzei* Trpb. (Samuel *et al.*, 1930), *F. occidentalis* Pergande (Gardner *et al.*, 1935), *F. fusca* Hind. and *Thrips setosus* Moulton (Kobatake *et al.*, 1984), *Thrips palmi* Karney (Yeh *et al.*, 1992) and *F. Intosa* (Wijkamp *et al.*, 1995). The adults persistently transmit the virus while feeding though only the nymph can acquire the virus from the host (Watterson, 1986). Vector control with insecticides didn't prevent successful transmission due to short time of feeding has been found to be necessary for infection to occur (Nagata, 1999). Moreover broad host range of the vector and increased resistance to insecticides (Maris *et al.*, 2003) impede the control of TSWV by insecticide application.

2.2.2. Symptomatology

Many workers have explained the symptoms caused by TSWV on tomato (Best, 1968; Black, 1972; Dixon, 1981; Sabitha *et al.*, 1984; Cho *et al.*, 1989; Raychaudhari and Nariani, 1989 and Chowfla *et al.*, 1999).

Best (1968) classified different TSWV strains into three main groups based on the systematic symptoms developed by them on plants *viz.* those producing severe necrosis along with the formation of brown (or purple) pigment (Strains A, B, D); those producing only very mild surface necrosis unaccompanied by pigmentation often in the form of ring spot or parallel line pattern (Strains C₁ & C₂) and those in which neither visible necrosis nor pigmentation (Strain E). Strain A was distinguished from all by the fact that it was the only one which produced primary pigmented necrotic disc lesions on inoculated tomato leaves and was the only one which produced apical necrosis (so called tip blight). All strains caused a stunting of tomato plants. Strains A and B also caused leaves and leaflets to curl downwards, whereas strains C₁, C₂ and E caused a flattening of leaves and

leaflets and a marked shortening of internodes. Strain D was characterized by the formation of purple pigment along with petioles and veins.

Dixon (1981) also reported that different strains of this virus were known to cause distinctive symptoms on tomato *viz.* tip blight, necrosis, ring spot, mild mosaic and severe mottle. In Tamil Nadu, Sabitha *et al.* (1984) noted that the pathogen attacked all stages of the crop. Infected plants showed dwarf growth and produced only a few fruits of small size. The initial symptoms were distinct chlorotic ring spots on the leaf immediately below the terminal bud on the terminal shoots followed by flaccidity of leaves. Later necrotic concentric rings appeared on the leaves. The severely infected plant produced brown necrotic streaks on the petioles, stems and on terminal bud. They noted bronzing of leaves as one of the typical characteristic symptoms of TSWV infection in tomato.

Watterson (1986) reported that pattern of spread of TSWV was distinctive in that infection radiates from a central point rather occurring down the row. According to Chowfla *et al.* (1999), development of bronze coloured marking on the upper side of the leaflets was accompanied by slight downward curling of leaves, followed by upward marginal rolling, stiffening of leaflets and formation of small circular spots on leaves. Necrosis extended to stem causing the latter to wilt. Pale areas of various shapes ranging from irregular mottling, blotchiness to distinct concentric rings was observed by them on fruits formed after the infection.

2.2.3. Epidemiology

It is believed that TSWV is not seed transmitted (Tomaru *et al.*, 1982 and Iwaki *et al.*, 1984) and climatic factors including multiplication and dispersion of vectors significantly contribute to the spread of the virus.

Temperature ranging from 20 - 35° C favoured thrips migration (Harding, 1961; Cho *et al.*, 1987) while temperature above and below that range contributed to the

reduction in thrips movement and TSWV incidence (Harding, 1961). During the years of high rainfall, TSWV incidence was considerably reduced (Cho *et al.*, 1987 and Kobatake *et al.*, 1984) due to reduced thrips infection (Harding, 1961). Wind velocity also plays an important role in the migration of thrips. Mass flights of *Frankliniella schultzei* occurred when wind velocity at 3 m above the crop canopy was greater than 10 km per hour. Relative humidity appeared to be less important than temperature as a stimulus for inducing the migration of the thrips (Sakimura, 1963)

Kumar (1988) noticed a higher incidence of TSWV in January - March transplanting and that of lesser incidence in November transplanting under Coimbatore conditions. Jasmine (1991) observed a lesser incidence of the same during October planting. Singh and Tripathi (1991) reported highest incidence of TSWV in January – February.

2.2.4. Interaction within the strains

A number of strains of TSWV occurred naturally and each of which had genetic continuity and could be characterized and identified by using diagnostic symptoms on the indicator plants as genetic markers (Best, 1968). Reports on both synergism and cross protection are available (Best 1954 and Finlay, 1952). According to Best (1954) presence of strain E in the plants had partially protected them against severe damage by strain B when it was inoculated 19 days later. In contrast Finlay (1952) reported a synergistic effect between two strains in *L. pimpinellifolium*.

Apart from this, Best (1968) also proposed the existence of genetic recombination of different strains of TSWV. In his study, he applied an inoculum containing two strains of TSWV to either tomato or *Nicotiana glutinosa* plants and found the formation of new strains which were stable and bred true over many years and satisfied the requirements of recombinants.

2.2.5. Screening for resistance

Screening for resistance to TSWV under field conditions along with susceptible controls was followed by many workers (Hutton and Peak, 1949; Kumar, 1988; Jasmine, 1991 and Aramburu and Rodriquez, 1999).

Hutton and Peak (1952) have shown that the efficiency of TSW virus inactivating system in some resistant potato species varied considerably with the changes in temperature. They suggested that inoculation of hybrid progenies with ring-spot strain of TSW at a constant temperature of 90°F might facilitate the selection of resistant and susceptible phenotypes. Since atleast five genes control the resistance to a group of strains of TSWV Finlay (1953) further added that atleast one strain from each group might be used in inoculation of clones of hybrid progenies to facilitate selection of resistant and susceptible phenotypes under standardized environmental conditions.

A method of mechanical transmission of TSWV was suggested by Rao *et al.*(1980). The sap from infected leaves of tomato plants collected and ground in 0.1 M PO₄ buffer (pH 7.0) containing mercaptoethanol (1g per 3 ml) was rubbed on the upper surface of the two or three pairs of leaves just below the growing point after abrasion with corborundum powder. Reddy *et al.* (1983) suggested side-grafting of tender diseased scion in to the test plants for screening against TSWV.

A rapid efficient mechanical inoculation procedure was developed to screen a large number of tomato plants for TSWV resistance. Inoculation preparation by triturating young systemically infected tomato leaves in phosphate buffer was applied with a pressurized air brush to the test leaves after applying carborundum powder. With this method 600 – 1000 plants could be efficiently inoculated in a single day with an infection rate of 85 – 95 per cent (Cho *et al.*, 1987). Mechanical method of inoculation was followed by many workers with or without modifications as and when required (Paterson *et al.*, 1989; Stevens *et al.*, 1994; Roca *et al.*, 1997; Latham and Jones, 1998; Rosello *et al.*, 1999 and Aramburu and Rodriquez, 1999). But Kumar *et al.* (1993) opined

that sole use of mechanical inoculation might result in the loss of valuable germplasm because species with vector resistance mightn't be identified.

There are also reports on successful screening of varieties for resistance to TSWV based on transmission by thrips (Kumar *et al.*, 1993 and Rosello *et al.*, 1999). Rosello *et al.* (1999) reported that inoculation by populations of thrips proved to be more efficient than mechanical transmission independent of isolates. Virus-free nymph of *Frankliniella occidentalis* were allowed to feed on the plants infected with different isolates of TSWV and the resultant adults were kept in cages (115 x 70 x 60 cm) and covered with anti-thrips mesh (150 µm). The plants to be tested along with control were raised in trays (34.5 x 23 x 6.5 cm) and kept inside the cage and a periodical change in location and orientation of trays was done in order to assure homogenous plant exposure to thrips. More than 500 thrips were estimated to be in each transmission cage.

2.2.6. Assessment of disease

Visual symptom pertaining to TSWV infection was used as initial criterion for evaluation by many workers (Paterson *et al.*, 1989; Stevens *et al.*, 1994 and Kumar *et al.*, 1993). Holmes (1948) classified the reaction of tomato plants for TSWV under natural conditions into dead, severely diseased, moderately affected and apparently healthy. Last two categories had appreciable number of fruits.

Since all kinds of symptom appear in plants once infected with TSWV, employing a graded scale is difficult. Hence Kumar (1988) rated plants as susceptible if exhibited any of the characteristic symptom of TSWV infection; field resistant if didn't show any of the symptom up to harvest despite severe natural infection and resistant if they didn't exhibit symptom even after graft-inoculation at any stage of crop growth

In contrast Juliatti and Maluf (1996) made observation based on the scale from 1 (apparently healthy plants) to 5 (dead plants). Boiteux *et al.* (1999) developed a disease scoring method to each individual plant qualitatively viz. 1 - no disease, 2 - weak apical

leaf curl, 3 – top distortion plus chlorotic or purple coloration of apical leaves, 4 – severe top distortion and necrosis of apical leaves, 5- severe stunting and general necrosis. Readings were converted to a Disease Severity Index (DSI) using following procedure i.e. the number of plants in each symptom category was multiplied by the corresponding numerical grade and the products were added and then divided by the total number of plants. They also calculated Disease Incidence (DI) as the proportion of the plants exhibiting the symptom and included as an additional evaluation criteria.

2.2.7. Sources and inheritance of resistance

Reports about inheritance TSWV resistance are quite confusing. Unstable nature of virus and presence of numerous strains add the confusion as well. It was shown that resistance appeared to be strain specific. Initial observation of apparently single gene resistance are now found to be confounded by modifying gene action (Watterson *et al.*, 1989)

Smith (1944) reported that though resistance to TSWV was fully recessive, F₁ heterozygotes were found to be more difficult to infect systematically than were the susceptible parents. Kikuta and Frazier (1946) after obtaining highly significant ratios of 3 (resistant) to 1 (susceptible) from the F₂ progeny of crosses between resistant and susceptible lines reported that resistance to TSWV in Pearl Harbor variety was apparently due to single dominant gene.. Holmes (1948) concluded that resistance in an Argentinian variety Rey de los Tempranos appeared to be controlled by a single recessive gene.

In an attempt to breed TSWV resistant hybrids using Porter's strain of *L. pimpinellifolium* Hutton and Peak (1949) reported that the inheritance of resistance as being obscure but likely to be controlled by a polygenic system. Reporting the 12 years of testing resistance of tomato hybrids to natural infection of TSWV in field plots, Smith and Gardner (1951) noted that the level of resistance found in the Porter's strain of *L. pimpinellifolium* hadn't been recovered in any of its progenies following a cross with

susceptible variety. No evidence of simple mendelian inheritance of resistance was observed by him.

Finlay (1951) noted that the F_1 heterozygotes of Pearl Harbor and Rey de los Tempranos had a very high resistance to TSW complexes in the field though two parent varieties were susceptible. The resistance possessed by each of the parents to some of the individual strains was transferred to the F_1 hybrids additively producing resistance to a wide range of strains as may be found in the field complexes.

In all the above breeding programmes, resistance or susceptibility of the phenotypes was assessed by their reaction to either infection by the disease in the field or to a natural complex of the virus strain mechanically inoculated (Finlay, 1953) hence led to a meager and confused understanding of inheritance mechanism of TSWV resistance in tomatoes.

Finlay (1952, 1953) gave the first glimmerings of understanding of the mechanism of inheritance of resistance to TSWV. He mapped the resistance or susceptibility of various reputedly resistant and susceptible varieties of tomato to 10 strains of TSWV under controlled condition. He found that 3 independently inherited recessive genes (sw_3 , sw_4 and sw_5) and two dominant alleles (SW_1^a and SW_1^b) were responsible for TSWV resistance in the parents studied. He used four sources of TSWV resistant genes *L. pimpinellifolium*, Pearl Harbor, Rey de los Tempranos and Manzana of *L. esculentum*. He reported that only those plants having the two dominant alleles SW_1^a and SW_1^b could only be completely resistant to all strains studied by him. He further found that variety Rey de los Tempranos was resistant to 7 out of 10 strains of TSWV listed and resistance was controlled by four genes viz. sw_3 , sw_4 , sw_5 and SW_1^b . Resistance to other 3 strains was controlled by the allele SW_1^a which couldn't be present along with SW_1^b in any homozygous cultivar. He suggested that since two of these five genes namely SW_1^a and SW_1^b proved to be allelic, complete resistance could be achievable only in F_1 hybrids. Hence he concluded that it wouldn't possible to breed a homozygous variety of

tomato that was completely resistant to all the known strains of TSWV by using the four resistant varieties used by him.

Kumar (1988) after studying 15 crosses involving 5 susceptible parents and 3 wild species viz. *L. peruvianum* var. *humifusum*, *L. hirsutum* f. *glabratum* and *L. hirsutum* found that resistance was controlled by a few resistant genes, in certain cases apparently more than four. He suggested that simple selection mightn't be possible since resistance involving additive dominance and duplicate epistasis. Watterson *et al.* (1989) after developing TSWV resistant lines from *L. peruvianum* concluded that inheritance of resistance to TSWV appeared to be due to major dominant gene with undetermined modifying genes.

Stevens *et al.*, (1992) reported that resistance in cv. Stevens derived from *L. esculentum* and *L. peruvianum* to the tested isolate of TSWV was controlled by a single dominant gene. From F₁, back crosses and F₂ populations of the resistant parent CNPHTX 405 and susceptible parent IPA – 5. Boiteux and de Giordano (1993) indicated that inheritance of resistance to TSWV is controlled by a single dominant gene *Sw – 5*. They supported the hypothesis framed by Robinson in 1976 that the dominant gene is neither isolate specific nor species specific. Due to its almost complete prevention of infection, *Sw – 5* gene seemed to display a mode of action similar to that of any vertical resistant gene.

From the data of F₁, back crosses and F₂ populations of crosses between two *L. esculentum* varieties with two TSWV resistant lines having common ancestor Rey de los Tempranos, Juliatti and Maluf (1995) indicated that dominance variance was of greater magnitude than additive variance in both the crosses. They estimated 1 – 3 genes with partial dominance for resistance was responsible for resistance in breeding lines and suggested the selection of resistant plants in the segregating populations. Juliatti *et al.* (1996) tested two populations F₁ and F₂ of the cross Santa Clara x F₁ (Santa Clara x

Stevens) for an isolate of TSWV in Brazil. They found that resistance was controlled by single dominant gene *Sw* – 5.

Rosello *et al.* (1998) reported that in breeding line UPV – 32 obtained from resistance introgression from *L. peruvianum*, apart from *Sw* – 5, UPV – 1 resistance genes, another UPV – 32 gene (proposed by them as *Sw* – 6) segregating independently of the former two was also responsible for TSWV resistance they further indicated that resistance conferred by it is of a lesser extent than that of them. Rosello *et al.* (2001) pointed out that in a breeding line UPV – 1 derived from PE – 18 accession of *L. peruvianum*, resistance was controlled by a dominant gene. The interesting observation made was that penetrance of that resistance gene was complete in mechanical inoculation and incomplete when thrips transmission using *Frankliniella occidentalis*. Linkage tests between resistance gene of the lines of UPV and RDD (carrying *Sw* -5) series indicated allelism. In heterozygotes the level of resistance expressed by UPV was higher than that expected from RDD (*Sw* -5) explaining that resistance to TSWV from UPV – 1 may be of higher value for development of commercial hybrids.

2.3. Breeding for Multiple Virus Resistance (MVR)

Once the information about the inheritance of individual diseases is acquired, then we can make a programme to combine the appropriate resistance to several viruses along with desirable attributes. In plants MVR can be obtained from two means *viz.* harnessing resistance imparted by single or two or more genes showing linkage controlling different viral resistance and

another by integration of resistance controlled by different genes from individual parents in F₁ hybrids or subsequent progenies.

From breeder's perspective linkage among resistant loci are beneficial when they derive from an individual parent at line (*cis* configuration). When crossed with susceptible parents, these resistances will be inherited as a single unit and likely to remain intact in the subsequent progeny. When linked resistant genes are donated by different parents (*trans* configuration) recombination between the loci is necessary to obtain both the resistance into a single line.

Such resistance to multiple viruses was frequently shown to be possible to those viruses which are taxonomically related. The works of Boiteux and de Giordano (1993) and Brommomschenkel and Tanskley (1997) and Scott (2001) in tomato and many examples from cucurbits for potyvirus resistance (Gilbert-Albertini *et al.*, 1993 and Grumet *et al.*, 2001) could sufficiently prove the same. At the same time when there is an unwanted linkage between gene(s) resistant to one virus and gene susceptible to another virus may be a bottleneck to the breeders for the development of MVR genotypes.

In tomato the breeding line CNPHTX 405 was found to be resistant to two tospoviruses namely TSWV and Tomato Chlorotic Spot Virus (TCSV) both controlled by a single dominant gene *Sw* – 5 (Boiteux and de Giordano, 1993) whereas in IPA – 5 line, the same gene imparted resistance to 3 tospoviruses *viz.* TSWV, TCSV and Groundnut Ring Spot Virus (GRSV) (Brommomschenkel and Tanskley, 1997). In addition, recently resistance has also been observed against the Groundnut Bud Necrosis Virus (GBNV) in tomato carrying the *Sw*-5 gene (Rolf *et al.*, 1998)

2.3.1. Screening for MVR

Williams (1977) and Kalloo (1988) advocated simultaneous or sequential screening for more than one disease. Alternatively one can use a line already resistant to one or two diseases and incorporate resistance from other diseases in the same line from other sources. Anon. (1998) suggested the mechanical inoculation for screening *Capsicum* spp. Resistant to PVY and TMV and developed routine screening methods for all major pepper viruses.

2.3.2. Breeding methods for MVR

Breeding for resistance to one virus is relatively straightforward. The difficulty arises in breeding for MVR (Provvidenti, 1985). Flexibility in choice and application of breeding methods is needed to pyramid resistance genes into breeding lines and varieties quickly and easily (Nelson, 1973).

Various selection procedures for MVR were given by different authors which that includes tandem selection (Halleur and Miranda, 1981) entailing sequential selection on a series of traits, independent culling (Hazel and Lush, 1942) involving sequential or concurrent selection for desirable traits over every generations and index selection (Smith, 1936) based on an aggregate scale as a means of differentiating genotypes possessing superior trait combinations. Tandem selection is recommended for mono-/oligogenic traits whereas independent culling for oligo-/polygenic and index selection for quantitative traits.

Barnes (1961) reported that plants having promising complimentary traits in F_3 to F_5 generations might be crossed for developing multiple resistant cultivars. Khush (1977) suggested that single cross F_1 hybrid could be crossed with other resistant donors to make double or top crosses to combine resistance to given disease. Bosch *et al.* (1990) also advocated going for three way or four way crosses and then selection of resistant plants in segregating generations for more than one disease and they recommended pedigree method for handling segregating populations.

According to Kyle and Provvidenti (1993), each disease problem is handled separately in breeding programmes because of the commonly accepted generalization that monogenic resistance, which is the most simple to transfer, is narrow in effect i.e., isolate or strain specific. When advanced lines are developed, resistance to each pathogen must be combined and selected in a commercially acceptable type through back cross or intercrossing. For transfer of recessive genes and oligogenic or polygenic characters, this final phase may require a number of

generations. Clearly simply inherited MVR would expedite the development of varieties with adequate levels of resistance to several viral diseases.

Agarwal *et al.* (2000) suggested controlled mating among the resistant progenies in the backcross or F₂ or succeeding generations in case of polygenic system. For oligogenic resistance they suggested backcross method. Sharma (1997) suggested Reciprocal Recurrent Selection method in simultaneous improvement of horticultural characters while incorporating disease resistance.

2.3.3. MVR Works in tomato

Walter (1956a) developed tomato lines carrying three recessive genes for resistance to TMV and combined this resistance to single recessive gene resistant to TEV (Walter 1956 b).

Thomas and Mink (1998) obtained two lines *viz.* Pr 18-4, Pr 8-5 each F₅ progeny derived from a separate interspecific cross between *L. esculentum* Bonny Best and Acc. 143 obtained from *L. peruvianum* PI.128655. According to them these lines were unique because they contained a resistance mechanism that non-specifically confers immunity or extreme resistance to phloem inhibiting pathogens of potato and tomato. These pathogens include Potato Leaf Roll Virus, Turnip Yellow Top Virus, and Beet Curly Top Virus. They suggested these lines for further improvement of tomato for MVR and also to investigate the nature and inheritance of pathogen resistance

2.4. Biochemical basis of resistance

A wide range of phytochemicals impart protective action against plants. According to van Loon (1982) once local lesions start to develop due to virus infection, changes in activities of enzymes involved in respiration, aromatic biosynthesis and enzymes *viz.* glucose-6- phosphate dehydrogenase, 6-phosphogluconate, phenylalanine ammonia-lyase, cinnamic acid-4-hydroxylase. Caffeic acid o-methyl transferase, polyphenol oxidase, peroxidase, ribonuclease, acid phosphatase and protease activities become apparent. He further quoted that an

understanding of mechanism of disease induction can be derived only from comparison of cytological, physiological and biochemical differences between healthy and infected plants thereby measures for controlling viral pathogen might be eventually devised (van Loon, 1987). Literature pertaining to some important parameters contributing to biochemical basis of resistance is reviewed.

2.4.1. Phenols

Accumulation of phenolic compounds in host parasite reaction is the general phenomenon of resistance and breakdown of these compounds determined the degree of resistance (Farkas and Kirlyay 1962 and Sindhan and Parashar 1984). Post infectious increase of phenols might be due to the tendency of phenols to accumulate at the site of infection which is involved in the defense mechanisms of plants through the interference in the metabolic activities of pathogens (Farkas and Kirlyay, 1962). Phenolic substances and their oxidation products, quinones have been reported to play an important role in imparting resistance against pathogens (Kosuge, 1969 and Goodman *et al.* 1967).

Gard and Maaloe (1959) proposed that any irreversible change in nucleic acid is sufficient to cause a complete loss of infectivity. Tannins are general protein precipitants. Hence they also can precipitate viruses which are also proteins (Mahadevan, 1991). Schonbeck and Schlossev (1976) reported that high phenol content can inactivate the virus. Non-diffusible chemicals like tomatine, phenols etc. have a key role in plant defense mechanism as reported by Tapliyali and Nene (1967). Many reports suggested an increased synthesis of phenols in plants in very large quantities due to plant pathogen interactions (Vidhyasekharan, 1990)

2.4.1.1. Total phenols

Sasikumaran *et al.* (1979) observed decrease in content of total phenol in TLCV infected leaves of tomato. *L. hirsutum* f. sp. *glabratum* B 6013, a resistant wild species had a high total

phenol content (134.22 µg/g) when compared to the susceptible commercial variety Red Cherry (24.42 µg/g) and the difference in total phenol content was highly significant (Kalloo, 1986).

Singh and Abidi (1988) also reported high phenol (514 mg/100 g to 933 mg/100 g) content in cultivars of tomato which showed high disease resistance. Sudha (1991) observed that increased permeability of the cell membrane followed by increased intensity of metabolism of the invaded cell and neighboring tissues the time plant got infected with TSWV. She indicated that in resistant tissues such reaction preceded more rapidly and thereby accumulating phenolics. She also found lines resistant at field level, had remarkably higher amounts of phenols than the commercial varieties.

Jasmine (1991) reported that phenol content was highest in ARTH 4 (376.56 mg/100g) among indeterminate hybrids and ARTH 3 (335 mg/100g) among the determinate hybrids in the attempt to screen the hybrids for TSWV under natural conditions. Sankari (2000) reported that TLCV resistant hybrids and moderately resistant hybrids had higher total phenolics than susceptible ones. Jamou (2002) reported that total phenol content was increased in both healthy and tospovirus inoculated plants of tomato with increase in age of the plants and increase was more in infected rather than healthy plants. Muthulakshumi (2003) found negative association between total phenol content and TLCV incidence. She observed high total phenol content in H 24 among parents and MLCR 2 x CLN 2123A and CLN 2123A x MLCR 2 in the crosses studied.

2.4.1.2. Orthodihydroxy phenols

Orthodihydroxy phenols are known to be highly toxic and play a major role in disease resistance (Mahadevan, 1966). They get easily oxidised by polyphenol oxidase and peroxidase to highly reactive quinones which are effective inhibitors of sulph-hydryl enzymes thereby preventing the metabolic activity of host and parasitic cells (Mahadevan, 1970). Orthodihydroxy phenolic compounds such as caffeic acid, chlorogenic acid, orthoquinones and tannins were shown to strongly inhibit the activities of cellular enzymes produced by microorganisms in addition to growth inhibition (Hunter, 1978).

Sasikumaran *et al.* (1979) observed decrease in content of OD phenol in TLCV infected leaves of tomato. Sankari (2000) also reported higher amount of total and orthodihydroxy phenols in the leaves of hybrids and its parents in tomato resistant to leaf curl virus.

2.4.2. Host enzymes

Host enzyme like polyphenol oxidase and peroxidase play an important role in disease resistance. These enzymes are responsible for degradation and synthesis of phenolics and quinones respectively. Peroxidases have been implicated in the regulation of cell wall elongation, phenol oxidation, polysaccharide cross-linking, IAA oxidation, cross linking of extension monomers, oxidation of hydroxyl-cinnamyl alcohols onto free radical intermediates and wound healing (Vidhyasekharan, 1997).

According to van Loon (1987) combined action of PO and PPO leads to the polymerization of phenols thereby yielding pigments responsible for the color of the developing necrotic spots. Bradley *et al.* (1992) reported that the increased PO action has been correlated with resistance in many plants and these are involved in the polymerization of proteins and lignin or suberin precursor into plant cell walls or movement through vessels. Phenylalanine ammonia-lyase (PAL), one of the key enzymes in the phenyl propanoid and flavanoid pathway was increased in both incompatible and compatible interactions between plants and pathogen (O'Neil and Sounders, 1994). PAL induces synthesis of salicylic acid (SA) which induced systemic resistance in many plants.

Schwarze (1954) reported that the disturbance in chlorophyll synthesis was accompanied by higher PO activity possibly because of more precursors common to both PO and chlorophyll. Vidhyasekharan (1988) reported that higher level of Polyphenol in the infected tissue could be one reason for the increased PPO activity in virus-infected plants. According to him, due to virus infection the redox potential at the host is altered as a result of abrupt rise in the enzyme activity.

Lodh *et al.* (1973), Sasikumaran *et al.* (1979) and Ragupathi (1995) reported an increased activity of PO and PPO in TLCV infected leaves. Markose (1996) reported that polyphenol oxidase activity was more in bacterial wilt resistant variety of chilli. Paul (1998) also confirmed

similar type of behaviour for polyphenol oxidase activity with respect to wilt resistance in chilli, brinjal and tomato. Ragupathi (1995), Kandan (2000) and Vasanthi (2001) reported a significant increase in activity of PAL in TLCV infected leaves. Jamou (2002) reported an increase in PO and PPO activities in tomato plants inoculated with tospovirus. Healthy plants also exhibited an increase in activity of these enzymes with increase in age.

2.4.3. Proteins

In many plant species, response to infection by pathogen or due to various abiotic stresses is accompanied by the synthesis of a variety of host proteins, several of which are termed as pathogenesis related proteins. They show high resistance against degradation by proteolytic enzymes and are present in the intercellular fluids of the leaves. In tomato plants infected with TLCV, Ragupathi (1995) reported the presence of one additional protein with the molecular weight of 18 KDa in addition to seven proteins present in healthy plants. Sudha (1996) reported that two additional proteins with molecular weights of 52 and 34 KDa in the TSWV infected plants in addition to similar proteins present in the healthy plants.

2.4.4. Isozyme analysis

Isozymes are different variants of the same enzymes, having identical or similar functions and present in the same individual (Market and Molter, 1959). These enzymes were known to increase in number as a result of disease or injury (Kanazawa *et al.*, 1965).

Studies conducted by Bashan *et al.* (1987) on the relation of enzymes and resistance against *Pseudomonas syringae* pv. *tomato* revealed the presence of four dibased PO isozymes in extracts from diseased plants, while only one was present in healthy plants. Wang *et al.* (1994) carried out a genetic analysis of a complex hypersensitive reaction to bacterial spot in tomato using eighteen isozymes and a morphological marker. They reported significant heterosis score as linkage between the marker locus and a hypersensitive reaction factor in cv. Hawaii 7988.

Lindhout (1995) extensively reviewed the use of markers in tomato for identification of cultivars against various pathogens and pest. About 25 genes were reported so far. Gupta *et al.* (1996) studied the level of total phenol, polyphenol oxidase and peroxidase in leaves of *Alternaria* leaf blight resistant and susceptible cultivars of *Brassica* spp. They reported an increased level of total phenol and more number of bands for polyphenol oxidase in resistant cultivars.

Peroxidase induction and its isozyme patterns in leaves of *Alternaria solani* resistant tomato cultivar (NCEBR – 1) and susceptible (HC – 3880) were studied by Fernandez *et al.* (1996). They reported an increase in number of bands and enzyme activity in resistant cultivars and suggested that the possibility of PO being involved in defense mechanism against the pathogen.

Solarzano *et al.* (1996) reported an increase in number of bands, peroxidase and polyphenol oxidase activity in cultivars of tomato resistant to *Alternaria solani*. Gazaryan *et al.* (1996) suggested a specific interaction between plant peroxidase and IAA oxidation because IAA plays a crucial role in Shikimate pathway resulting in production of secondary metabolites like phenols.

Bose (1999) screened 20 tomato cultivars/accessions against bacterial wilt using peroxidase and polyphenol oxidase isozymes. He also reported that resistant varieties had more number of bands compared to susceptible varieties. Sankari (2000) reported isozyme pattern of peroxidase and polyphenol oxidase in tomato hybrid and parents against TLCV incidence. She reported that though the banding patterns were similar, the induction of enzyme activity was earlier in case of resistant hybrid and its parents when compared to their susceptible counterparts.

2.5. Genetic studies

For an efficient breeding programme, through understanding of genetic system of crop plants is must. For breaking yield barriers and evolving varieties having built – in high yield potential, hybridization is the most potent technique. The selection of suitable parents on the basis of genetic value for hybridization is one of the most important steps in a breeding

programme. Diallel analysis helps to evaluate the parents in terms of genetic make up and brings out two components of variation *viz.* additive genetic variance and dominance variance.

The results on the study of *per se* performance, heterosis, combining ability, correlation, path coefficient analysis and gene action on tomato have been reviewed hereunder.

2.5.1. *Per se* performance

Per se performance is the true realized mean of the crosses and this being a direct estimate based on no assumptions, selection of crosses on the basis of *per se* performance assumes more reliable (Govindarasu *et al.*, 1981)

2.5.1.1. Plant height

Dod *et al.* (1992) noted a mean plant height of 119.71 cm in the cross Marglobe x AC 238. Jasmine and Ramadass (1993) evaluated the indeterminate hybrids of tomato and observed the maximum plant height in the hybrid ARTH-1. Among the 21 types evaluated, Kumar *et al.* (1995) recorded a plant height of 109.60 cm from the cross Sel 30 x Flora - dade, whereas in the parents, it was 88.00 and 66.33 cm respectively. Mageswari (1996) recorded the higher plant height from the hybrid LE 625 x CO 2. Sankari (2000) reported a maximum plant height of 133.05 cm in the combination FLCR 1 x MLCR 3 among the 36 hybrids studied. Indu Rani (2002) recorded the same in SL 120 x PT 4716 A (124.69 cm) out of 80 crosses studied.

2.5.1.2. Number of branches per plant

Dod *et al.* (1992) noted the highest number of branches in the cross Pusa Early Dwarf x Marglobe. According to Singh and Singh (1993) best crosses with respect to number of branches per plant were Punjab Chhuhara x 84 – 8 and Pusa Ruby x 84 – 8. Kumar *et al.* (1995) recorded 14.67 branches per plant in the cross involving Hisar Arun and Sel – 30. Mageswari (1996) recorded the highest no. of branches in the hybrid LE 625 x CO 2 whereas Sankari (2000) reported a value of 12.5 in the combination FLCR 2 x MLCR 9 among the 36 hybrids studied

2.5.1.3. Number of fruits per plant

Singh and Singh (1993) reported the highest number of fruits per plant in the cross Punjab Chhuhara x 84-8. Pujari and Kale (1994) indicated the cross EC 11927 x Punjab Chhuhara to be the best with 126.6 fruits per plant. Kumar *et al.* (1995) obtained the highest fruits per plant in the cross Hisar Arun x Anteny out of several cross combinations evaluated. Mageswari (1996) reported 101.88 fruits per plant in the cross between CHT 267 and KS7. Joshi *et al.* (1998) reported the highest number of fruits per plant in the hybrids Meenakshi and Sutton Prolific. Sankari (2000) reported maximum number of fruits 59.40 in the combination FLCR 2 x MLCR 6 among the 36 hybrids studied. Premalakshmi (2001) reported the highest number of fruits per

plant in the hybrid LE 625 x LE 990. Indu Rani (2002) recorded the same in PT 4716 A x SL 120 (69.48) out of 80 crosses studied.

2.5.1.4. Individual fruit weight

Jasmine and Ramadass (1993) opined fruit weight to be the most important character determining the yield and FM-2 (an indeterminate hybrid) which recorded the highest fruit weight registered maximum fruit yield per plant. Singh and Singh (1993) obtained a higher mean fruit weight in the hybrid Pusa Ruby x 84-8. Similar results of the highest mean fruit weight was also recorded from the cross Ace x Flora-dade (Kumar *et al.*, 1995) and LE 812 x Arka Ahuti (Mageswari, 1996). Joshi *et al.* (1998) reported the highest mean fruit weight in the hybrids Sutton Prolific and BSS 3. Among 36 hybrids, the maximum fruit weight of 110.10 g was reported in FLCR 5 x MLCR 9 (Sankari, 2000). Premalakshmi *et al.* (2002) reported the highest fruit weight in LE 812 x LE 68 (63.34g) and LE 812 x PKM-1(62.10g). Indu Rani (2002) observed the same in CLN 2026 C x SL 120 (66.09) out of 80 crosses studied.

2.5.1.5. Yield per plant

Increase in yield of tomato is due to the manifestation of hybrid vigour. This was observed by several workers (Jasmine and Ramadass, 1993; Singh and Singh 1993, Pujari and Kale, 1994 and Jawaharlal, 1994)

Mageswari (1996) reported the highest yield per plant in the cross IHR 709 x LE 812. Sadashiva *et al.* (1997) found that the processing hybrids Arka Shreshta and Arka Abhijit recorded highest yield per plant. Saglam *et al.* (2000) reported that among the seven hybrids, the hybrid 89-8 F₁ and 91-6 F₁ recorded the highest yield per plant. Sankari (2000) recorded highest yield of 4.217, 4.050, 3.850, 3.795 and 3.775 kg per plant in the hybrids FLCR 5 x MLCR 9, FLCR1 x MLCR 6, FLCR 1 x MLCR 7, FLCR 5 x MLCR 3 and FLCR 1 x MLCR 8 respectively. Indu Rani (2002) observed highest yield in LE 812 x SL 120 (2.38 Kg) out of 80 crosses studied.

Premalakshmi *et al.* (2002) reported that two hybrids PKM 1 x LE 68 and LE 812 x PKM 1 recorded highest yield of 5.07 Kg per plant.

2.5.1.6. Total Soluble Solids (TSS)

According to Villareal (1980) tomato meant for processing purposes should have a minimum of 4.5° Brix. Higher TSS in hybrid were reported by Bajaj *et al.* (1990), Kasrawi and Amr (1990) and Supe and Kale (1991). Dod and Kale (1992) found the cross Pusa Ruby x AC 238 and Reddy and Reddy (1994) observed the cross No. 6 - 1286 x Slava - VF for the highest TSS. Mageswari (1996) recorded the highest TSS of 6.20° Brix in the cross PKM 1 x LE 113. Saglam *et al.* (2000) found the hybrid CLX 3704 F₁ recorded the highest TSS of 6.17° Brix. Sankari (2000) recorded the highest TSS of 5.00° Brix in the cross between FLCR 5 and MLCR 9. Premalakshmi (2001) reported that the hybrid Arka Ahuti x LE 812 and its reciprocal cross recorded the highest TSS of 5.97° and 5.62° Brix respectively. Indu Rani (2002) recorded highest TSS in PT – 4716A x Hissar N₂ (5.89° Brix) out of 80 crosses studied. Makesh *et al.* (2003) reported that PKM 1 x CO 3 and PKM 1 x Acc.No. 378642 and Acc.No. 378642 x Acc.No. 378893 recoded highest TSS of 4.50°Brix out of 30 hybrids studied.

2.5.1.7. Acidity

Keeping quality of processed tomatoes and tomato products is influenced by acidity through the inhibition of spore germination of the thermophilic organisms. Moreover along with sugars it forms an important parameter for determining flavour. Gould and Berry (1972) suggested an acidity value of 0.35 to 0.55 per cent to be ideal for processing. Higher value of titrable acidity in hybrids were observed by Bajaj *et al.* (1990) and Kasrawi and Amr (1990). Reddy and Reddy (1994) for EC 128767 x EC 130042 and Mageswari (1996) for IHR 709 x LE 812 recorded the highest value of titrable acidity. Sankari (2000) recorded the highest value of acidity in the hybrid FLCR 1 x MLCR 9. Premalakshmi (2001) recorded the highest value of acidity in the hybrid LE 68 x

PKM 1. Indu Rani (2002) recorded highest acidity in terms of citric acid in CLN 2026 C x SL - 120 (0.68 %) out of 80 crosses studied.

2.5.1.8. Ascorbic acid

The highest ascorbic acid content in hybrids was recorded by Jamwal *et al.* (1984) and Bajaj *et al.* (1990). Dod and Kale (1992) recorded the highest value of ascorbic acid in the cross Punjab Chhuhara x S 12. Mageswari (1996) reported that the highest ascorbic acid content in the cross Processor 40 x Arka Saurab with a mean of 18.97 mg/100g. Sankari (2000) recorded the highest ascorbic acid content in the cross FLCR 3 x MLCR 9. Premalakshmi (2001) observed the highest ascorbic acid content in the hybrids LE 625 x PKM1, LE 625 x LE 812 and LE 812 x PKM 1. Indu Rani (2002) recorded highest ascorbic acid content in Hisar N₁ x CLN 1466J (29.5 mg/100g).

2.5.1.9. Lycopene

Lycopene pigment in tomato fruit decides the optimum stage of ripening and also an important criterion for processing. Recently it has been identified as a nutritional factor because of its antioxidant property. Hence breeding for high lycopene tomato would also help in developing tomato varieties or hybrids which would improve the general health of tomato consumers. Sankari (2000) recorded the highest lycopene content of 5.11mg/100g in the cross FLCR 3 x MLCR 9. Indu Rani (2002) reported highest lycopene content in CO 3 x Patriot (6.65 mg/100g)

2.5.2. Heterosis

Heterosis would be is at its maximum in the F₁ hybrids. It is the property of heterozygosity and due to the superior gene combination in a hybrid contributed by both the parents (Mather, 1955). Heterosis for yield reflects through the heterosis in its individual components. It is essential to consider the *per se* performance and the heterosis percentage together while judging the hybrid combination for exploitation of heterosis. Extensive studies which have been conducted on heterosis for both quantitative and qualitative characters in tomato are reviewed hereunder.

2.3.1. Plant height

Positive heterosis for plant height has been reported by Jawarhalal (1994), Kumar *et al.* (1995) and Sidhu and Singh (1995). Among the 21 hybrids studied by Kumar *et al.* (1995), only five exhibited significant positive heterosis over superior parent which ranged from 11.82 to 24.54 per cent while the cross Sel 30 x Flora-dade (13.30 per cent) exhibited positive heterosis over the best check Naveen. Sankari (2000) recorded the maximum heterosis of 115.57 per cent in the first season and 84.66 per cent in the second season over the best parent in the cross FLCR 1 x MLCR 3. Premalakshmi (2001) observed positive heterosis of 15.21 per cent in the hybrid Arka Ahuti x LE 990. Indu Rani (2002) recorded the maximum heterosis of 7.47 per cent in PT

4716A x SL 120. Profound positive heterosis over mid parent for this trait was also reported by Sekar (2001a).

2.5.2.2. Branches per plant

According to Singh and Singh (1993) the hybrid Pusa Ruby x 84 – 8 exhibited the highest heterobeltiosis (12.26 per cent). Jawaharlal (1994) obtained positive heterosis of 3.38 over the best parent in the cross CH 7 x Punjab Chhuhara. Studies by Kumar *et al.* (1995) revealed the cross Hisar Arun x Sel 30 exhibited positive significant heterosis. Sankari (2000) recorded the highest significant positive relative heterosis of 127.03 per cent in the cross FLCR 2 x MLCR 9.

2.5.2.3. Days to flowering

Negative heterotic effects are of immense value especially in tomato. Since early fruits tend to bring the highest price in the fresh market. Jamwal *et al.* (1984) recorded highest negative heterosis over better parent in EC 119292 x Solan Surkha. Singh and Singh (1993) reported that the hybrid Punjab Chhuhara x 84 – 3, HS 102 x 84 – 8 and Pusa Ruby x 84 – 8 exhibited significant negative heterosis for days to flowering over best parent indicating their usefulness in breeding for early tomato varieties. Pujari and Kale (1994) revealed that the cross EC 119275 x Punjab Chhuhara recorded significant negative heterosis over both better and best parent. Jawaharlal (1994) reported that the hybrid CHT. 267 x KS. 7 recorded a favourable minimum mean performance with negative heterosis for days to flowering among the thirty F₁ hybrids studied. Mageswari (1996) reported that the di value for days to flowering was negative and significant and was highest in the hybrid Processor – 40 x Arka Saurabh and Arka Ashish x LE 812 irrespective of seasons.

2.5.2.4. Number of fruits per plant

Pujari and Kale (1994) have obtained a higher level of 91.90 per cent heterosis over the superior parent in the cross LA 126 x Punjab Chhuhara and 58.00 per cent over the best parent in EC 119275 x Punjab Chhuhara. Kumar *et al.* (1995) obtained a very high level of 193.55 per cent

heterosis over the superior parent in the cross Sel.30 x Flora-dade. Mageswari and Natarajan (1999) reported that the cross CHT. 267 x KS 7 registered the highest relative heterosis of 80.97 per cent and heterobeltiosis 45.20 per cent over mid and better parent. Lakshmanan (1996) estimated 59.15 per cent heterosis over best parent for LE 368 x Pusa Ruby. Aruna and Veeraragavathatham (1996) reported that the hybrid PKM 1 x LE 113 showed positive heterosis estimate over the better parent. They concluded that the hybrids of parents with high fruit number didn't show heterosis and that of low fruit number did show. Sankari (2000) obtained the highest heterosis over the best parent in the cross FLCR 5 x MLCR 3. Premalakshmi (2001) recorded positive heterosis over the best parent in the hybrid LE 625 x LE 990. Positive and significant heterosis over better parent was recorded by Indu Rani (2002) in four hybrids out of 80 hybrids studied with maximum heterobeltiosis value of 25.96 per cent in CLN 2026C x Hisar N₁.

2.5.2.5. Fruit weight

Out of 66 F₁ hybrids evaluated by Dod *et.al.* (1992), only four crosses showed significance for fruit weight and the heterosis percentage ranged from 8.08 to 24.91 over the better parent. Singh and Singh (1993) observed a high heterotic effect of 27.72 per cent over better parent in the cross Punjab Chhuhara x 84 – 8. Similar results of high heterosis (23.84 per cent) over the best parent has been reported in the cross LA 126 x Punjab Chhuhara by Pujari and Kale (1994). Reddy and Reddy (1994) also observed a significant and positive relative heterosis in the cross Miliana x Sioux (39.98 per cent). Mageswari (1996) reported that the individual fruit weight in LE 812 x Arka Ahuti cross recorded higher relative heterosis during both the seasons. Premalakshmi *et al.* (2002) observed that the hybrids LE 812 x LE 68 and LE 812 x PKM 1 recorded positive heterosis of 40.31 and 39.08 per cent over the best parent for individual fruit weight. Patgaonkar *et al.* (2003a) observed higher diii value of 32.94 and 39.61 per cent in hybrids L 3960 x CL 32–6–19–0–0 and LE 79 x Sel. 120

2.5.2.6. Yield per plant

Yield per plant is the key factor by which hybrids are usually evaluated. Heterosis in terms of increased yield has been reported by several workers (Naranderakumar *et al.*, 1988; Kanthaswamy and Balakrishnan, 1989; Prema, 1989; Kurian, 1990 and Aruna, 1992).

Mandal *et al.*(1992) observed significant positive heterosis for yield per plant in the hybrids Pusa Early Dwarf x KS-1, J 10-2-2 x CO 3, KS-1 x La Bonita, J 10-2-2 x La Bonita and J 10-2-2 x KS-1. Pujari and Kale (1994) reported a high heterobeltiosis (123.40 per cent) in the cross Manalucie x Roma. High heterotic effect of 87.06 per cent over best parent was exhibited by the cross Hisar Arun x Sel 30 (Kumar *et al.*, 1995). Sankari (2000) recorded 88.30 per cent heterosis over the best parent for the cross FLCR 5 x MLCR 3. Similarly Indu Rani (2002) reported the highest heterosis over the best parent in CLN 2026C x SL – 120.

2.5.2.7. Total Soluble Solids

Heterotic effects have been reported for total soluble solids by Kurian (1990), Aruna (1992) and Dod and Kale (1992).

Reddy and Reddy (1994) observed the cross EC 128767 x EC 130042 performed better when compared with other crosses studied and recorded 41.77 and 36.46 per cent heterosis with respect to mid and better parents. Mageswari and Natarajan (1999) recorded significant positive heterobeltiosis values in the cross LE 812 x Arka Saurabh (11.18 per cent). Dev and Rattan (1996) observed that heterosis over best parent was 80.81 per cent for TSS in the cross combination of EC 35383 x Gola. Sankari (2000) reported that the hybrid FLCR 5 x MLCR 9 recorded the positive heterosis for total soluble solids content. Premalakshmi (2001) obtained positive heterosis in the hybrid Arka Ahuti x LE 812 (25.65 per cent). Positive and significant heterosis (15.37 per cent) over better parent was recorded by Indu Rani (2002) in LE 812 x SL 120. Patgaonkar *et al.* (2003b) observed higher dii and diii value of 15.93 per cent in hybrid L 3960 x L 4139 out of seven hybrids studied.

2.5.2.8. Acidity

Heterosis for acidity was reported Kanthaswamy and Balakrishnan (1989) and Aruna (1992). Reddy and Reddy (1994) observed significant positive herotic value of 41.77 per cent over mid parental value in the cross EC 128767 x EC 130042. Jawaharlal (1994) pointed out a heterosis of 2.92 per cent over better parent. The heterosis over better parent for acidity ranged from -3.23 (HS 101 x Pusa Ruby) to 31.10 per cent (Punjab Chuhara x Hisar Arun) was reported by Rai *et al.* (1996). Sankari (2000) obtained positive heterosis over better parent during both the seasons in the hybrid FLCR 1 x MLCR 9. Positive and significant heterosis over best parent was recorded by Indu Rani (2002) in only two out of eighty hybrids. Out of eight crosses studied, Singh *et al.* (2002) recorded significant positive heterobeltiosis of 136.11 per cent in DVRT-2 x H 36 out of eight crosses studied. Significant and positive heterosis of 21.31 per cent over the best parent was reported by Patgaonkar *et al.* (2003b) in the cross L 3960 x CL 32-D-0-1-19-0-0.

2.5.2.9. pH of the fruit juice

Organic acids, the important components of processing tomatoes determine the pH of the juice. Conti *et al.* (1988) reported that F₁ hybrids had lower pH values than their parents. Kurian (1990) and Aruna (1992) reported that the heterosis over mid and better parents were all negative. Premalakshmi (2001) obtained highest significant negative heterobeltiosis in LE 68 x LE 625 (-25.11 per cent) followed by PKM 1 x LE 812 (-24.54 per cent).

2.5.2.10. Ascorbic acid

Heterosis for ascorbic acid content of fruits was reported by several workers (Kurian, 1990; Aruna, 1992; Dod and Kale, 1992 and Jawaharlal, 1994). Dev and Rattan (1996) estimated diiii value of 57.20 per cent for ascorbic acid in the cross EC 133711 x Marutham. Sankari (2000) reported that the hybrid FLCR 5 x MLCR 1 recorded the highest heterosis over the better parent (34.64 per cent). Premalakshmi (2001) reported that the hybrid LE 625 x PKM 1 recorded the

highest positive heterosis over the best parent (22.03 per cent) whereas Indu Rani (2002) reported the same in LE 812 x SL 120. Singh *et al.* (2002) recorded a range of 24.14 (Agata x H 36) TO 59.67 (DVRT- 2 x H 24) for heterobeltiosis.

2.5.2.11. Lycopene

Dorairaj (1981), Kurian (1990) and Shoba and Arumugam (1991) estimated a heterosis over the mid and better parent in hybrids. Aruna (1992) reported that all the hybrids exhibited significant positive heterosis over the mid parental values and the hybrid PKM 1 x FFS 120 recorded the maximum of 11.76 and 15.44 per cent heterosis over higher parent in first and second season respectively. Jawaharlal (1994) estimated heterosis for lycopene was higher in the hybrid CHT 267 x Processor - 40. Lakshmanan (1996) recorded positive and significant heterosis for 28 out of 36 hybrids studied. Wang Lei *et al.* (1998) reported low heterosis for lycopene content. Sankari (2000) reported that the cross combination FLCR 3 x MLCR 9 recorded the highest heterosis over the better and best parent for lycopene content. Singh *et al.* (2002) reported three out of eight crosses studied namely DVRT- 2 x H 24, DVRT- 2 x H 36 and Sel. 18 x H 24 showed significant positive heterosis over mid and better parent.

2.5.2.12. Disease resistance

Shoba (1990) reported that all the six crosses studied by her exhibited negative heterosis for TLCV incidence.

2.5.3. Combining ability

Combing ability refers to the capacity or ability of a genotype to transmit superior performance to its crosses. General combining ability (*gca*) refers to the average performance of a line in a series of crosses whereas specific combining ability (*sca*) is a deviation from the performance predicted on the basis of *gca*. Generally *gca* is a consequence of dominance and epistasis (Handerson, 1950).

Study on the combining ability helps to understand the genetic background of each character. It helps in the evaluation of inbreds in terms of their genetic value and in the selection of suitable parents for hybridization. Superior cross combinations can also be identified by this technique. Singh and Narayana (1993) expressed their view that combining ability analysis could

be more reliable since it is not restricted to one gene model and it operated with feasible assumptions.

2.5.3.1. Fruit weight

Anbu *et al.* (1980) found that *gca* effects of line 113 and tester LE 68 were highly significant and positive which resulted in high *sca* effect for fruit weight. A significant and positive *sca* was noticed for fruit size for the crosses, *viz.* Rossol x Punjab Chhuhara (Virdelwala *et al.*, 1981) and LE 758 x LE 113 (Govindarasu *et al.*, 1982).

Jawaharlal (1994) found that the parent Punjab Chhuhara was identified as the best general combiner for individual fruit weight and yield per plant. Wang Lei *et al.* (1998) found that highly significant *gca* for parents and *sca* for hybrids was observed for fruit weight in five processing tomato cultivars. Sankari (2000) reported that the lines FLCR 1 and FLCR 2 as best combiners. Significant positive *sca* for maximum fruit weight (12.8) in CLN 1464A x SL 120 by Indu Rani (2002)

2.5.3.2. Yield and yield contributing characters

Sidhu *et al.* (1981) reported that the crosses Sel 152 x Roma, Gamed x Sel 152, Roma x Punjab Chhuhara, La Bonita x Punjab Chhuhara and Gamed x La Bonita showed high *sca* for mean fruit weight, number of fruits, fruit yield, plant height and number of branches per plant. Virdelwala *et al.* (1981) observed that F₁ which expressed significant and positive *sca* effects had atleast one good general combining parent indicating that additive x dominance epistatic effects contributed to *sca* effects.

Govindarasu *et al.* (1982) reported that LE 758 among the lines and LE 68 among the testers were identified as the best general combiners for yield and its components. The hybrids LE 736 x LE 413, LE 740 x LE 570, LE 758 x LE 113 and LE 782 x LE 413 were found to be good specific combiners for yield, plant height, fruit size and number respectively. Kurian (1990) found that the parents Sakthi, TK 318, Fresh Market 9 and HW 208 F were good general

combiners and the hybrid Sakthi x TH 318, Sakthi x Fresh Market 9, LE 206 x St 64, LE 206 x Ohio 8129 showed significant positive *sca* effects for yield.

Jawaharlal (1994) found that the parent Punjab Chuhara as the best general combiner for individual fruit weight and yield per plant. Kumar *et al.* (1997) found that the parents Pusa 120, WIR 3900, Pusa Gaurav and Chiku had significant positive *gca* effect for fruit yield per plant. Rai and Syamal (1998) observed that the magnitude of specific combining ability variances were higher in plant height and number of primary branches and lower in days to flowering. Wang Lei *et al.* (1998) indicated that *gca* and *sca* were highly significant for fruit equatorial diameter, fruit polar diameter, fruit index, fruit flesh thickness, fruit weight, number of fruits per plant and fruit yield.

Premalakshmi (2001) reported that the parent LE 635 was identified as the best general combiner for number of fruits per plant, LE 990 for earliness, plant height and laterals per plant, LE 68 for yield per plant and LE 812 for fruit weight. Indu Rani (2002) reported positive and significant *gca* for yield per plant in CLN 2026C, CLN 1464A, CO 3 and LE 812 and recorded maximum *sca* of 0.5 in CLN 1464A x SL 120.

2.5.3.3. Fruit quality characteristics

Jamwal *et al.* (1984) observed the combination EC 12175 x Lalmani showed high *sca* estimates for ascorbic acid content. Khalaf - Allah *et al.* (1985) reported that the variety 'Yellow plum' showed high *gca* effects for TSS and acidity. Kurian (1990) reported high *sca* effects in LE 20 x St 64 and Sakthi x TH 318 for TSS and in LE 206 x Ohio 8129 for lycopene and LE 214 x Fresh Market 9 for ascorbic acid.

Sathyanarayana and Anand (1992) estimated *gca* and *sca* effects from Line x Tester analysis and reported that the hybrid BWR 5 x 674 (5.13 B) and BWR 15 x 858 (5.53 B) showed significant positive *sca* effects for TSS, even though parents involved showed high x low *gca* for lycopene. The hybrid BWR 14-1 x 1032-2 exhibited high *sca* effects for lycopene content with a *per se* of 10.64 mg/100g involving parents of low x high *gca* effects, while for acidity

BWR 15 x 1614 recorded significant *sca* effects. Aruna (1992) found that *gca* estimates were higher than *sca* estimates in the case of ascorbic acid and lycopene. Jawaharlal (1994) reported that the parents Processor 40 and KS 7 were regarded as good general combiners for TSS and lycopene and parent CHT 267 for acidity.

Lakshmanan (1996) reported that the parent LE 8 was identified as the best general combiner for acidity and ascorbic acid. Kumar *et al.* (1997) identified the parents Pant Bahar and Arka Vikas showed high positive *gca* effect and the cross Pusa 120 x Pant Bahar exhibited highest *sca* effect for ascorbic acid and Chiku exhibited highly significant positive *gca* effects and the cross Pusa Sheetal x Chiku exhibited significant *sca* effect for lycopene. Premalakshmi (2001) observed the parents LE 68 as the best general combiner for lycopene and PKM 1 for acidity. Indu Rani (2002) suggested Arka Ahuti as best combiner for TSS.

2.5.3.4. Disease resistance

Shoba (1990) observed negative *gca* effects for the lines CO 3 and LE 1125 whereas positive *gca* for PKM 1 for TLCV. The *sca* effect of the hybrids ranged from -0.47 in the cross LE 1125 x LE 376 to 2.14 in the cross CO 3 x LE 376. Sankari (2000) found that the SCA variance was higher than GCA variance in a line x tester analysis comprising of four lines and nine testers indicating the preponderance of non-additive type of gene action for TLCV incidence at 75 DAT. She reported that *sca* effect of hybrids varied from -25.52 in FLCR 3 x MLCR 6 to 30.54 in FLCR 1 x MLCR 5 in the 36 hybrids studied.

2.5.4. Association of characters

2.5.4.1. Correlation studies

Yield is a complex character with polygenic inheritance, hence direct selection for yield could not be possible. Correlation studies provide information that selection for one character results in progress for all positively correlated characters. Importance of correlation studies in

selection programmes is appreciable when highly heritable characters are associated with the important character like yield. The reported information on correlation between yield and other component characters and disease resistance in tomato is reviewed here under.

Character correlated with yield	Reported by	
	Positive correlation	Negative correlation
Plant height	Padda <i>et al.</i> (1968) Paranjothi (1974) Singh and Mital (1976) Singh and Singh (1980) Bangaru (1981) Parthasarathy <i>et al.</i> (1982) Manivannan and Irulappan (1986) Mishra and Mishra (1989) Patil and Bojappa (1993) Jawarhalal (1994) Indu Nair (1995) Mageswari <i>et al.</i> (1997) Elakkuvan (1997) Aruna and Veeragavathatham (1997) Mala (1999) Sankari (2000)	Nandpuri <i>et al.</i> (1973) Verma <i>et al.</i> (1976) Kumar (1978) Das and Chakrabarthy (1984)
Branches per plant	Kumar <i>et al.</i> (1979) Parthasarathy <i>et al.</i> (1982) Manivannan and Irulappan (1986) Patil and Bojappa (1993) Singh and Tripathy (1995) Mageswari <i>et al.</i> (1997)	
Number of fruits per plant	Tayel <i>et al.</i> (1960) Goldenberg (1967) Padda <i>et al.</i> (1968) Srivastava and Sachan (1973) Singh <i>et al.</i> (1974) Nandpuri <i>et al.</i> (1976) Caviechi and Silvetti (1976) Singh and Mittal (1976)	Kumar <i>et al.</i> (1979) Aruna and Veeragavathatham (1997) Indu Rani (2002)

	Prasad and Prasad (1977) Bangaru (1981) Dudi and Kalloo (1982) Das and Chakrabarthy (1984) Manivannan (1985) Raijadhav <i>et al.</i> (1986) Khattra <i>et al.</i> (1988) Susheela <i>et al.</i> (1990) Jawaharlal (1994) Indu Nair (1995) Mageswari <i>et al.</i> (1997) Das <i>et al.</i> (1998) Premalakshmi (2001)	
Fruit weight	Srivastava and Sachan (1973) Nandpuri <i>et al.</i> (1976) Bangaru (1981) Dudi and Kalloo (1982) Manivannan (1985) Jawaharlal (1994) Ghosh and Syamal (1994) Mageswari (1996) Aruna and Veeragavathatham (1997) Das <i>et al.</i> (1998) Yadav and Singh (1998) Premalakshmi (2001) Sankari (2000) Indu Rani (2002)	Nandpuri <i>et al.</i> (1973) Kumar (1978) Raijadhav <i>et al.</i> (1986) Susheela <i>et al.</i> (1990)
Total soluble solids	Singh and Singh (1982) Manivannan (1985) Khattra <i>et al.</i> (1988) Padmini (1995)	Bangaru (1981) Aruna (1992) Patil and Bojappa (1993) Verma <i>et al.</i> (1997)

Acidity	Patil and Bojappa (1993) Aruna and Veeraragavathatham (1997) Sankari (2000) Indu Rani (2002)	Sarala Devi (1984)
Ascorbic acid	Sarala Devi(1984) Jawaharlal (1994) Aruna and Veeragavathatham (1997) Indu Rani (2002)	Patil and Bojappa (1993) Indu Nair (1995)
Lycopene	Medhi and Parthasarathy (1981) Indu Nair (1995) Aruna and Veeragavathatham (1997) Indu Rani (2002)	
TLCV incidence		Shoba (1990) Mala and Vadivel (1999) Sankari (2000) Kulkarni and Madalageri (2002) Muthulakshumi (2003)
TSWV incidence		Kumar (1988) Sudha (1996)

2.5.4.2. Path coefficient analysis

A path coefficient is simply a standardized partial regression coefficient and as such measures the direct influence of one variable upon another and permits the separation of the correlation coefficient into components of direct and indirect effects. The use of this method requires a cause and effect situation among the variables (Dewey and Lu, 1959)

Based on path coefficient analysis, Srivastava and Sachan (1973) reported that fruit weight had negative direct effect on yield due to its significant negative correlation with number of fruits per plant whereas number of fruits per plant had the highest positive direct effect on yield, followed by fruit diameter. Singh and Mittal (1976) reported that number of primary branches and fruit weight in early pickings had high positive direct effect on total yield. Kumar *et al.* (1979) reported that the number of fruits per plant exerted the highest direct effect on yield. The indirect effect contributed through this trait was also positive especially for number of lateral per plant. Singh and Singh (1982) indicated that TSS had positive effects on yield per plant

directly and also indirectly through plant height, fruit size, number of fruits per plant and days to flowering.

Khattra *et al.* (1988) reported that the highest contribution for yield per plant was by number of fruits per plant through direct effect, followed by the indirect contribution of average mean fruit weight. Most characters influenced fruit yield through number of fruits per plant. Sharma (1990) reported that plant height had the direct effect on yield of the fruits. Supe and Kale (1992) conducted path analysis studies and indicated that the importance of the character, number of branches per plant, which had strong positive direct effect on yield. It was followed by number of fruits per plant for high positive indirect effect. The direct effect of plant height was negative.

Indu Nair (1995) reported that the number fruits per plant, number of locules per fruit and TSS exhibited positive, direct effect on yield. Mageswari *et al.* (1999) reported that positive direct effects on yield were exerted by the traits fruits per plant, fruit weight and laterals per plant. Fruit exerted the highest positive direct effect and also it influenced the yield through flesh thickness, days to last harvest and laterals per plant.

Sankari (2000) pointed out that fruit weight exerted a direct positive effect on yield while number of fruits, TSS, acidity, ascorbic acid, lycopene exerted direct positive indirect effect on yield. She also pointed out that TLCV incidence at 75 DAT had negative direct effect on yield. Negative indirect effects were observed through plant height, number of branches per plant, TSS, total phenol, OD phenol and TLCV incidence at 15 and 45 DAT. Direct positive effect of fruit weight on yield was also observed by Indu Rani (2002).

2.5.5. Gene action

Gene actions have been studied for several economic attributes in tomato. The results may be used in choosing the effective breeding procedure and identifying characteristics for making selection effective in segregating generations. The gene effects studied for different characters are presented herewith.

Character	Gene action	Author(s)
Plant height	Additive	Singh and Mital (1978) Sidhu <i>et al.</i> (1981) Kurian (1990) Lakshmanan (1996)
	Non-additive	Kaloo <i>et al.</i> (1974) Anbu <i>et al.</i> (1980) Govindarasu <i>et al.</i> (1981) Aruna (1992) Jawaharlal (1994) Sankari (2000)
	Additive and non-additive	Baroncelli <i>et al.</i> (1972) Peter and Rai (1980) Sekar (2001a)
Branches per plant	Non additive	Kaloo <i>et al.</i> (1974) Aruna (1992) Jawaharlal (1994) Lakshmanan (1996) Sankari (2000)
	Additive and non-additive	Sekar (2001a)
Earliness	Additive	Ponnuswamy and Muthukrishnan (1979) Banerjee and Kaloo (1989)
	Non additive and additive	Brandolini <i>et al.</i> (1974) Sekar (2001a)
Individual fruit weight	Additive	Kolhe (1967) Khalaf-Allah (1970) Kaul and Nandpuri (1972) Singh and Nandpuri (1974) Mital <i>et al.</i> (1974) Anand (1977) Mital and Singh (1978) Dixit <i>et al.</i> (1980) Govindarasu <i>et al.</i> (1981)
	Additive and non-additive	Swamy and Mathai (1982) Singh <i>et al.</i> (1988) Aruna (1992) Kumar <i>et al.</i> (1997) Wang Lei <i>et al.</i> (1998) Singh and Nandpuri (1974) Raijadhav and Kale (1985) Kurian (1990) Sekar (2001a)

	Non-additive	Mital <i>et al.</i> (1974) Anbu <i>et al.</i> (1980) Gibrel <i>et al.</i> (1982) Sankari (2000)
	Additive and additive- epistasis	Virdelwala <i>et al.</i> (1981)
Number of fruits per plant	Additive	Kolhe (1967) Kaul and Nandpuri (1972) Conti (1974) Kalloo <i>et al.</i> (1974) Dixit <i>et al.</i> (1980) Swamy and Mathai (1982) Sundaram (1986) Aruna (1992)
	Non-additive	Mital <i>et al.</i> (1974) Singh and Nandpuri (1974) Rattan and Saini (1976) Mital and Singh (1978) Anbu <i>et al.</i> (1980) Peter and Rai (1980) Sidhu <i>et al.</i> (1981) Bhutani and Kalloo (1988) Kurian (1990) Lakshmanan (1996) Sankari (2000) Indu Rani (2002)
	Additive and non-additive	Baroncelli <i>et al.</i> (1972) Singh and Nandpuri (1974) Raijadhav and Kale (1985) Sekar (2001a)
Yield per plant	Additive	Kaul and Nandpuri (1972) Kalloo <i>et al.</i> (1974) Singh and Nandpuri (1974) Maggiore <i>et al.</i> (1976) Dixit <i>et al.</i> (1980) Singh <i>et al.</i> (1988) Jawaharlal (1994)

	Non-additive	Chaudhary (1970) Avarado and Cortazar (1972) Conti (1974) Mittal <i>et al.</i> (1974) Anbu <i>et al.</i> (1980) Bhutani (1981) Govindarasu <i>et al.</i> (1981) Gibrel <i>et al.</i> (1982) Bhutani and Kalloo (1988) Kurian (1990) Wang Lei <i>et al.</i> (1998) Sankari (2000)
	Additive and non-additive	Raijadhav and Kale (1985) Jamwal <i>et al.</i> (1984) Aruna (1992) Rai <i>et al.</i> (1997)
	Additive and dominance epistasis	Virdelwala <i>et al.</i> (1981)
	Additive and dominance	Baroncelli <i>et al.</i> (1972) Raijadhav and Kale (1985)
Acidity	Additive	Mittal <i>et al.</i> (1974) Singh and Nandpuri (1975) Gibrel (1983) Sundaram (1986) Kurian (1990) Jawaharlal (1994)
	Non-additive	Chaudhary (1970) Kalloo <i>et al.</i> (1974) Aruna (1992) Kumar <i>et al.</i> (1997) Sankari (2000) Indu Rani (2002)
	Dominance	Arora <i>et al.</i> (1982)
	Partial dominance	Bhutani (1981) Monma and Kamimora (1982) Kurian (1990) Premalakshmi (2001)
	Additive and epistasis	Lukyanenko and Lukyanenko (1986)
Ascorbic acid	Additive	Kurian (1990) Aruna (1992) Lakshmanan (1996)
	Non additive	Chaudhary (1970) Kalloo <i>et al.</i> (1974)

TSS	Additive	Sundaram (1986) Sekar (2001b)
	Additive and non-additive	Singh and Nandpuri (1975) Indu Rani (2002)
	Non additive	Chaudhary (1970) Kalloo <i>et al.</i> (1974)
Reducing sugar	Non additive	Sundaram (1986)
pH of the juice	Additive and non-additive	Peter and Rai (1980)

CHAPTER III

MATERIALS AND METHODS

The present investigation on tomato (*Lycopersicon esculentum* Mill.) was carried out during 2000- 2003 at the experimental fields of Department of Vegetable Crops, Horticultural College & Research Institute, Tamil Nadu Agricultural University, Coimbatore. It is located at an altitude of 426.6m above mean sea level, at 11° N latitude and 77° E longitude.

3.1. Screening for individual virus resistance

3.1.1. Screening for TLCV resistance

Six lines and one released variety known to be tolerant to TLCV based on the previous investigation from TNAU, Coimbatore (Sankari, 2000) were subjected to screening studies for confirmity. This germplasm includes the following

SI. No.	Line/ Variety	Origin	Source
1	LCR 1	R ₁ R ₁ F ₃ – P ₁ F ₆ – 6; a seedling progeny derivative of somoclonal variant from CO 3 isolated for leaf curl virus tolerance and big sized fruit	Dept. of Vegetable Crops, TNAU, Coimbatore
2	LCR 2	R ₁ R ₃ F ₃ 1-F ₆ -1; a seedling progeny derivative of somoclonal variants from leaf disc calli of CO 3	Dept. of Vegetable Crops, TNAU, Coimbatore
3	LCR 3	366-1; product of single plant selection from AVRDC line free from TLCV	Dept. of Vegetable Crops, TNAU, Coimbatore

- | | | | |
|---|--------------------------|---|--|
| 4 | LCR 4 | R ₁ R ₃ F ₃ 2-F ₆ -2-2; a seedling progeny derivative of somoclonal variants from leaf disc calli of CO 3 | Dept. of Vegetable Crops, TNAU, Coimbatore |
| 5 | LCR 9 | R ₁ R ₄ F ₃ 2-F ₆ -5; a seedling progeny derivative of somoclonal variants from leaf disc calli of CO 3 | Dept. of Vegetable Crops, TNAU, Coimbatore |
| 6 | CLN
2123A | Derivative of a multiple cross | AVRDC, Taiwan |
| 7 | H 24
(Hisar
Anmol) | Variety derived from the cross <i>L. esculentum</i> Cv. Sel 7 x <i>L. hirsutum</i> f. <i>glabratum</i> B 6013 through modified backcross pedigree selection | HAU, Hissar |

Screening was conducted both in insect proof glasshouse as well as field conditions during January – April, 2001.

3.1.1.1. Screening under field conditions

Twenty five plants were maintained in each genotype. All test plants were encircled with highly susceptible variety CO 3. Weeds around the experimental plots were not removed to facilitate more probable infection. Utmost care was taken not to spray any pesticide so that the vector activity was not hindered.

3.1.1.1.1. Symptomology of TLCV

Infected tomato plants exhibited clearing of veins, stunting and marked reduction in leaf size. The plants expressed typical mosaic symptoms, which varied from mild to severe degree. The reduction in size of the successive leaves was accompanied by shortening of the internodes with the result the leaves were crowded together. The leaflets were deformed and their margin curled inwards or outwards. The younger leaves were pale in colour with light green and dark

green areas. Puckering of the leaflet was a common symptom of the disease. The plant had the greater tendency to produce stunted lateral branches which ultimately resulted in a bushy appearance. The plants infected while young, remain stunted and seldom attained a size of more than 25 to 40 cm against 60 to 80 cm in uninfected plants. The disease induced partial or complete sterility of the infected plant depending on the stage at which infection had taken place. In case of late infection, the plant had borne no fruits but in earlier infection they had borne reduced floral structures. The flowers that developed after infection were invariably sterile.

3.1.1.2. Screening under insect-proof glasshouse conditions

Twenty five plants were maintained in each genotype. After 20 days of transplanting they were grafted with scions of plants previously infected with TLCV and tied airtight with polythene film, later on covered with polythene bags to maintain favorable environment for the transmission of the disease. The entire test plants are encircled with highly susceptible variety CO 3 and no plant protection measures was taken up.

3.1.2. Screening for Tospovirus resistance

Since almost all reports of studies conducted so far revealed that mechanical or graft-inoculated plants are susceptible, only field resistance to Tospovirus is taken as a tool for working out Percent Disease Infection (PDI). The source materials used were as follows

SI. No	Line/variety	Origin	Source
1	Stevens	Variety derived from the cross <i>L. esculentum</i> x <i>L. peruvianum</i> ; reported to carry a dominant gene <i>Sw-5</i> for TSWV	Dr. C.M. Rick, University of California, USA

resistance

- 2 LE 415 A germplasm reported to be tolerant under KAU, Thrissur field conditions

The test plants were tested along with those of TLCV under the same experiment with same experimental techniques under 3.1.1.1.

3.1.2.1. Symptomology of Tospovirus

The pathogen attacked all stages of crop growth. The pattern of spread was quite random. There were two sets of symptoms noticed.

1. In this case, the plants showed only bronzing symptom. Bronze colored markings developed first on nerves and later on whole ventral side of comparatively young leaflets accompanied by slight downward curling of leaves. Later upward marginal rolling and stiffening of leaflets were found to occur. Plants infected early were stunted and hardly produced any flower. But those infected late produced a few very small sized fruits with no blotched areas or concentric rings. But in any case plants were not killed. Premature fruit ripening was often noticed.
2. Small distinct thin crescent shaped black mark with pale margin intervened by green islands usually on the leaf immediately below the terminal bud depicted the initial symptom. Later dark more or less circular necrotic spots along with yellow speckling appeared on the leaves. As the disease advanced, those spots coalesce and extended very rapidly towards petioles, then to stem and occasionally to growing tips. At that stage severe deformation of leaves occurred. In some infected plants bronzing was also noticed but with much lesser extent than too at a later stage. Plants when affected at early stage became flaccid and killed. Those infected at later stage produced a few small fruits with pale red, yellow or blotched areas and occasionally with yellow concentric rings. Fruits formed prior to infection may show no symptom.

Criteria used for the selection of parents to target virus were

1. Plants free from particular virus incidence under high inoculum pressure even though they possess lesser fruit weight
2. Plants with better fruit weight and fairly higher level of resistance to particular virus
3. To ensure combined resistance in the F₁ hybrids, both the parents with fairly high level of resistance alone were considered.

Based on the above criteria five genotypes *viz.* LCR 1, LCR 3, LCR 9, H 24, CLN 2123A and one line LE 415 were chosen as a preferred material for TLCV and Tospovirus resistance respectively. Stevens which is highly acclaimed to be resistant to Tospovirus in several countries was found to be highly susceptible to Tospovirus which is particular in causing bronzing of stem and ventral surface of the leaf lamina without any necrotic symptom. It was observed that H 24 and CLN 2123A were also found to be free from Tospovirus under natural epiphytotic condition. In order to harness this added advantage of these lines in bringing out both disease resistances into possibly more number of hybrids, all the selected parents were crossed with each other, both direct and reciprocal to get maximum number of cross combinations. Crossing was done twice during August 2001 and February 2002.

3.2. Crossing the selected parents

3.2.1. Selfing technique

This technique is adopted to maintain the parents as inbreds for subsequent evaluation along with the crosses. Flower buds about to open in the following day were covered individually with butter-paper covers in all the parents.

3.2.2. Crossing technique

Flower buds of selected female parents were emasculated with finger nail in the afternoon between 3 and 5 p.m. one day prior to anthesis and were immediately covered with butter-paper cover. Simultaneously the selected unopened flower buds of male parents were also covered to avoid any contamination of foreign pollen. In the next day between 7.30 to 9.30 a.m. pollen grains from one day previously bagged flowers of male parent were dusted on the emasculated flowers of the female parent with the help of camel hair brush. After the transfer of pollens, the flowers were labeled and again covered with butter-paper covers. After ensuring proper fruit set in the crossed flowers, the covers were removed. Seeds were extracted from red ripe fruits, cleaned, washed, dried to a moisture content of eight per cent and stored for future use.

3.3. Evaluation of F₁ hybrids

During crop period, meteorological observations namely day and night temperature, relative humidity, rainfall in rainy days, wind velocity etc. were recorded.

3.3.1. Assessment of diseases

For both the diseases, observation was taken at fortnightly interval starting from 15 days after transplanting.

3.3.1.1. TLCV

A disease reaction scale suggested by Banerjee and Kalloo (1987a) with little modification in the range of coefficient of infection was adopted. Symptom severity grades designated with numerical values of 0 – 4 were given on the basis of visual observation. To quantify the disease severity, calculations were made as shown below

Sl.No.	Symptoms	Symptom severity grade	Response value	Coefficient of infection	Reaction
1	Symptoms absent	0	0	0.0 – 4.0	Highly Resistant (HR)

2	Very mild curling (up to 25 % leaves)	1	0.25	4.1 – 9.0	Resistant (R)
3	Curling and puckering of 26 – 50 % leaves	2	0.50	9.1 – 19.0	Moderately Resistant (MR)
4	Curling and puckering of 51 – 75 % leaves	3	0.75	19.1 – 39.0	Moderately Susceptible (MS)
5	Severe curling and puckering of > 75 % leaves	4	1.00	39.1 – 69.0	Susceptible (S)
				69.1 - 100.0	Highly Susceptible (HS)

$$\text{Calculated Symptom Severity (CSS)} = \frac{\text{Total symptom severity value}}{\text{Total no. of plants infected}}$$

$$\text{Calculated Response Value (CRV)} = \frac{\text{CSS} \times \text{Response value}}{2}$$

$$\text{Per cent Disease Incidence (PDI)} = \frac{\text{No. of diseased plants}}{\text{Total no. of plants observed}} \times 100$$

$$\text{Coefficient of Infection (CI)} = \text{PDI} \times \text{CRV}$$

3.3.1.2. Tospovirus

Since a plant once infected with Tospovirus, it ultimately leads to death of the plants or bronzing of whole plant leading to no further growth and development, mere PDI and the stage at which infection occurred were taken as criteria for the assessment of disease.

Total PDI was calculated after adding the PDI values of respective and combined virus infected plants.

3.3.2. Evaluation of F₁ hybrids and parents for combined resistance to diseases, yield and quality attributes during January – April 2002

Seeds of Thirty hybrids and six parents were sown in raised nursery bed. Seeds were treated with *Azospirillum* 2 g per Kg one day before sowing. Beds were treated with copper-oxy-chloride (2.5g/lit) for controlling damping off and were not covered with nylon net so that the vector activity was not hindered. Nursery seedlings were not given any granular application or spray of systemic insecticides. Thirty day old seedlings were raised in the field during January – April 2002 in RBD with two replications. The size of the plot was 3.0 x 2.4 m so as to accommodate 25 plants for each replication. Seedlings were planted at a spacing of 45 x 60 cm. All the thirty six entities were bombarded by planting susceptible variety CO 3 in rows over four

sides of the test plants. Weeds around the experimental plots were not removed. Normal fertilizer dose recommended for hybrids (250:250:250 Kg NPK/ha.) was applied. Half the nitrogen and full dose of phosphorus and potassium were applied as basal dose and incorporated well at the time of planting. The remaining dose of nitrogen was applied at two splits viz. 30 and 60 days after transplanting. Irrigation was given at 5 to 7 days interval. All recommended package of practices were followed but without spraying any pesticides.

Following observations were recorded on five equally competent representative plants selected at random per replication in each cross and parent.

3.3.2.1. Growth parameters

3.3.2.1.1. Plant height

The height of the plant from cotyledonary node to the tip of the plant was measured at the time of final harvest and expressed in centimeters.

3.3.2.1.2. Branches per plant

The number of branches borne on the main stem was expressed as number of branches per plant.

3.3.2.1.3. Days to 50 per cent flowering

Number of days taken for flowering of 50 per cent of the population from transplanting was counted.

3.3.2.2. Fruit characters

3.3.2.2.1. Number of fruits per plant

The number of fruits in each harvest was counted and total number of fruits from all harvests was expressed as number of fruits per plant.

3.3.2.2.2. Fruit weight

Average weight of fruits in each harvest was arrived at by dividing yield per plant obtained in that harvest with number of fruits harvested and the mean value of those pertaining to all harvests was taken as fruit weight. It was expressed in grams.

3.3.2.2.3. Yield per plant

The red ripe fruits picked from each plant were weighed immediately after harvest and the cumulative weight of fruits of all the harvests was taken as yield per plant. It was expressed in kilograms.

3.3.2.3. Quality traits

For this purpose unblemished, red ripe fruits were chosen at random from each of the five randomly selected plants at peak harvest stage in replication for all entities. The fruits were pulped by using pestle and mortar and the pulp was utilized for following analysis. Characters viz. p^H , sugars, sugar to acid ratio and pectin were recorded only for selected F_1 hybrids raised during July – Oct. 2002

3.3.2.3.1. Total Soluble Solids (TSS)

The Total Soluble Solids (TSS) content of the fruit pulp was determined by using ERMA Hand Refractometer. The reading was recorded as degree brix after deducting the correction factor.

3.3.2.3.2. Acidity

Titration acidity as per cent citric acid was determined by the method suggested by AOAC (1975).

3.3.2.3.3. Ascorbic acid

Ascorbic acid content was estimated by following the method of AOAC (1975) and expressed as mg 100 g⁻¹ fruit pulp.

3.3.2.3.4. Lycopene

Lycopene was estimated as per the method given by Ranganna (1977) and expressed as mg 100 g⁻¹ fruit pulp.

3.3.2.4. Biochemical basis of combined virus resistance

3.3.2.4.1. Total phenol

The total phenol content of the leaf sample was estimated by the method employing folin ciocalteu reagent (Bray and Thorpe, 1954) and expressed as mg per 100 g material.

3.3.2.4.2. Ortho-dihydroxy phenol

Ortho-dihydroxy phenol content of the leaf sample was estimated by employing Arnow's reagent as suggested by Johnson and Schaal (1957).

3.3.3. Evaluation of F₁ hybrids and parents for combined resistance to both the diseases during March – June 2002

Experimental technique followed in this season was similar to that described in 3.3.2. Since the crop is evaluated under severe summer, fruit set was adversely affected. Moreover fruit size was also reduced much. So yield, other quantitative and qualitative characteristics were not taken up with the view that these yields are not representative of the true hybrid yield.

3.3.4. Statistical analysis

The mean data of thirty F₁ hybrids and their parents obtained for each character from both the seasons was tabulated and subjected to analysis of variance (Panse and Sukhatme, 1957).

3.3.4.1. Estimation of heterosis

3.3.4.1.1. Relative heterosis

Heterosis expressed as per cent deviation of the cross from its mid parental value was estimated adopting the following formula

$$\text{Relative heterosis (d}_i\text{)} = \frac{\overline{F_1 - MP}}{\overline{MP}} \times 100$$

Where,
F₁ - mean of the cross or hybrid

$$\overline{MP} = \frac{P_1 + P_2}{2}$$

P₁ and P₂ are the parents involved in the cross.

3.3.4.1.2. Heterobeltiosis

Heterosis expressed as per cent deviation of the cross from the better parent involved in that particular cross combination was estimated by adopting the following formula

$$\text{Heterobeltiosis (dii)} = \frac{\overline{F_1 - BP}}{\overline{BP}} \times 100$$

Where, \overline{BP} - mean of the better parent

3.3.4.1.4. Best parent heterosis

Heterosis expressed as per cent deviation of the cross from the best parent in the experimental material in respect of a particular character was calculated by adopting the following formula

$$\text{Best parent heterosis (diii)} = \frac{F_1 - \overline{\text{BtP}}}{\overline{\text{BtP}}} \times 100$$

Where, $\overline{\text{BtP}}$ - mean of the best parent

3.3.4.1.5. Standard heterosis

Heterosis expressed as per cent deviation of the cross from the standard commercial hybrid in the experimental material in respect of a particular character was calculated by adopting the following formula (Singh and Narayana, 1993). This was worked out for season III alone using two commercial hybrids *viz.*, COTH 1 and Ankush as standard hybrids.

$$\text{Standard heterosis (SH)} = \frac{F_1 - \overline{\text{CH}}}{\overline{\text{CH}}} \times 100$$

Where, $\overline{\text{CH}}$ – mean value of local commercial hybrid

3.3.4.1.4. Test of significance

The standard errors for testing significance of heterosis were calculated as suggested by Wynne *et al.* (1970)

3.3.4.2. Diallel analysis

Using the data of selected parents and hybrids, diallel analysis was carried out.

‘Null hypothesis’ was tested to show that there were no genotypic differences among the progenies. This was worked out using randomized block design.

Source	df	Expectation
Blocks (b)	(b-1)	
Genotypes (g)	(g-1)	$\sigma^2e + \sigma^2g$
Error	(b-1)(g-1)	σ^2e

σ^2e = error variance

σ^2g = genotype variance

3.3.4.2.1. Combining ability analysis

Estimation of general and specific combining ability was done as per the procedures outlined by Griffing (1956) for method I of diallel analysis which included parents, F_1 's and reciprocals, assuming the following general formula for the model.

ANOVA with expected mean squares is furnished below

Source	df	Sum of squares	Mean sum of squares	Expected mean squares
gca	p-1	Sg	Mg	$\sigma^2e + \frac{1}{(P-1)} \sum i g_i^2$
sca	$\frac{P(P-1)}{2}$	Ss	Ms	$\sigma^2e + \frac{2}{(P-1)} \sum i \sum g S_{ij}^2$
Reciprocal	$\frac{P(P-1)}{2}$	Sr	Mr	$\sigma^2e + \frac{2}{(P-1)} \sum i \sum j r_{ij}^2$
Error	M	Se	Me ¹	σ^2e

Where,

$$S_g = \frac{1}{2p} \sum i (X_{i.} + X_{.i})^2 - \frac{2}{p^2} X_{..}^2$$

$$S_s = \frac{1}{2} \sum_i \sum_j X_{ij} (X_{ij} + X_{ji})^2 - \frac{1}{2p} \sum_i (X_{i.} + X_{.i})^2 + \frac{1}{p^2} X_{..}^2$$

$$S_r = \frac{1}{2} \sum_i \sum_j (X_{ij} - X_{ji})^2$$

P = number of parents involved

Expected mean sum of squares,

$$Me^1 = \frac{Me}{B}$$

Me = experimental error mean square for randomized block design

B = number of blocks

3.3.4.2.1.1. Calculation of combining ability effects

3.3.4.2.1.1.1. General combining ability effects (*gca*)

$$g_i = \frac{1}{2p} (X_{i.} + X_{.i}) - \frac{1}{p^2} X_{..}$$

Where,

P = Number of parents

X_{i.} = Row total of parents in the array

X_{.i} = Column total of parents in the array

X_{..} = Grand total of diallel table

3.3.4.2.1.1.2 Specific combining ability effects (*sca*)

$$S_{ij} = \frac{1}{2} (X_{ij} + X_{ji}) - \frac{1}{2p} (X_{i.} + X_{.i} + X_{j.} + X_{.j}) + \frac{1}{p^2} X_{..}$$

Where, P = Number of parents

X_{ij} = Array means of F₁

- X_{ji} = Array means of reciprocal F₁
- X_{i.} = Row total of first parent
- X_{.i} = Column total of first parent
- X_{j.} = Row total of second parent
- X_{.j} = Column total of second parent
- X_{..} = Grand total of diallel table

Reciprocal effect

$$r_{ij} = \frac{1}{2} (X_{ij} + X_{ji})$$

The variances of these effects were estimated as follows

$$\text{Var}(g_i) = \frac{P-1}{2P^2} \sigma^2_e$$

$$\text{Var}(s_{ij}) = \frac{1}{2} (P^2 - 2P + 2) \sigma^2_e$$

$$\text{Var}(r_{ij}) = \frac{1}{2} \sigma^2_e$$

The square root of the variance provide the corresponding standard error for calculating critical differences

3.3.4.2.1.2. Test of hypothesis

The validity of hypothesis was tested by t^2 test as suggested by Hayman (1954 a,b)

$$t^2 = \frac{(n-2) \sum (Var V_r - Var W_r)}{(\sum (Var V_r - Var W_r) - Cov^2(V_r W_r))}$$

where, V_r = Variance of the progeny of r^{th} parental array

W_r = Covariance of offspring of r^{th} array with respect to non-recurrent parent

t^2 value was compared with F value with 4 and (n-2) degrees of freedom. A significant t^2 indicated failure of atleast one of the assumptions of diallel analysis.

3.3.4.2.1.3. Deviation of regression coefficient (b) from zero and unity

The regression of covariance on variance and its standard error were calculated as follows

$$b = \frac{\text{Cov}(W_r, V_r)}{\text{Var}(V_r)}$$

$$\text{SE}(b) = \frac{\text{Var. } W_r - b \text{ Cov}(W_r, V_r)^{1/2}}{\text{Var } V_r (n-2)}$$

The significance of 'b' from zero and unity was tested as follows

$$H_0 = \frac{b-0}{\text{SE}(b)}$$

$$H_0 = \frac{1-b}{\text{SE}(b)}$$

The calculated values were tested against the table values of 't' for (n-2) degrees of freedom.

Hayman's diallel analysis

The following two procedures were adopted in the present investigation to gather information about gene action from diallel data,

1. Graphical analysis
2. Genetic analysis

V_r = The variance of r^{th} array

W_r = The covariance between the non-recurring parent and the offspring of the r^{th} array

W_r' = the covariance between array mean and the offspring of the r^{th} array

V_0L_0 = Variance of all parental means

V_0L_1 = Variance of the means of the arrays

V_1L_1 = Mean variance of arrays

W_0L_{01} = Mean covariance between the parents and the arrays

ML_1-ML_0 = Difference between the mean of the parents and the mean of their n^2 progeny

E = Expected environmental component of variation.

$W_r.V_r$ graph was drawn for all the characters studied and the limiting parabola for $W_r.V_r$ graph was constructed using the formula

$$W_r^2 = V_r \times V_0L_0$$

3.3.4.2.2. Genetic analysis

The following genetic components were estimated by Hayman (1954b) using second degree statistics and error mean squares

$$D = V_0 L_0 - E$$

$$F = 2 V_0 L_0 - 4 W_0 L_{01} - 2(P-2) E/P$$

$$H_1 = V_0 L_0 - 4 W_0 L_{01} + 4 V_1 L_1 - (3n-2) E/P$$

$$H_2 = 4 V_1 L_1 - 4 V_0 L_1 - 2E$$

$$h^2 = 4 (ML_1 - ML_0)^2 - 4(P-1) E/P^2$$

where,

D = Component of variation due to additive effects of genes

F = Covariance of additive and dominance effects

H₁ = Component of variation due to dominance effects of genes

H₂ = Proportion of dominance variation due to positive and negative effects of genes, H₁ [1-(U-V)²]. Where U and V are the proportions of positive and negative genes respectively

h² = Net dominance effect over all the loci

E = Environmental variation obtained for Me¹ from the analysis of variance table

P = Number of parents

The significance of above-mentioned parameters was tested by 't' test at (n-2) degrees of freedom using the standard errors of respective genetic parameters

$$T = \frac{\text{Parameter}}{\text{SE of parameter}}$$

The standard errors were calculated using the equation $S_2 = \text{Var} (W_r - V_r)/2$ and in terms of the main diagonal of covariance matrix given by Hayman (1954 a,b) as corresponding multipliers.

3.3.4.2.3. Ratios of genetic components

From the components estimated, the following genetic ratios were computed

$(H_1/D)^{1/2}$ = Mean degree of dominance over all loci

$H_2/4H_1$ = proportion of genes with positive and negative effects in the parents

$\frac{4(DH_1)^{0.5} + F}{4(DH_1)^{0.5} - F}$ = Proportion of dominant & recessive genes in parent

h^2/H_2 = Number of groups of factors controlling the character and exhibiting dominance

$$\text{Heritability (narrow sense)} = \frac{1/2D + 1/2H_1 - 1/2H_2 - 1/2F}{1/2D + 1/2H_1 - 1/4H_2 - 1/2F + E}$$

In addition to the above ratios parameter 'r' was also computed

r = Correlation coefficient between the parental measurement Yr and the parental order of dominance (Wr + Vr)

3.3.4.3. Association of characters

Correlation coefficient was estimated among the different yield components as per the method suggested by Panse and Sukhatme (1957).

$$\text{Correlation coefficient 'r'} = \frac{SP_{xy}}{SS_x \cdot SS_y}$$

SP_{xy} = Sum of products of two variables x and y

SS_x = Sum of squares of variable x

SS_y = Sum of squares of variable yield

Genotypic correlation coefficient

Genotypic correlation coefficients were estimated according to the following formulae given by Johnson *et al.* (1955).

$$r_g X.Y = \frac{\text{Genotypic covariance between x and y}}{(\text{Genotypic variance of X} \times \text{Phenotypic variance of Y})^{1/2}}$$

where, r_g = Genotypic correlation coefficient

x and y = Variables or characters under consideration

Significance of the phenotypic and genotypic correlation coefficients was tested by referring to the standard table given by Snedecor and Cockhran (1967).

3.3.4.4. Path coefficient analysis

Path coefficient analysis was carried out according to Dewey and Lu (1959), by partitioning the genotypic correlation coefficients into direct and indirect effects.

3.3.5. Evaluation of selected F₁ hybrids and parents for combined resistance to diseases, yield and quality attributes during July-October. 2002

After considering number of critical parameters viz. PDI (for TLCV, Tospovirus and their Total), CI for TLCV at 75DAT (obtained from I & II crop), number of fruits per plant, average fruit weight, yield per plant, acidity (from data on I crop), number of crossed seeds per fruit obtained in the female parent during crossing programme, heat tolerance, field establishment etc., the eight hybrids were short listed for further evaluation.

Selected F₁ hybrids were raised along with respective parents and two hybrids released for commercial cultivation namely Ankush (from Namdhari Seeds) and COTH 1 (from TNAU) during July- October 2002 in RBD with three replications. Experimental technique followed in this season was similar to that described in 3.3.1. One additional operation i.e. training with bamboo poles was done.

Apart from the observations as mentioned in 3.3.2, following observations were additionally done

3.3.5.1. Quality traits

For this purpose unblemished, red ripe fruits were chosen at random from each of the five randomly selected plants at peak harvest stage in replication for all entities. The fruits were pulped by using pestle and mortar and the pulp was utilized for following analysis.

3.3.5.1.1. p^H

One hundred grams of the pulp was taken in a 250 ml beaker and p^H meter (Digital p^H M/V meter DPH 14) was dipped on it (Dorairaj, 1981). The reading appeared was taken as p^H of the fruit pulp.

3.3.5.1.2. Sugars

The total, reducing and non reducing sugars were estimated as per the method suggested by Ranganna (1977) and expressed in per cent.

3.3.5.1.3. Sugar to acid ratio

Sugar to acid ratio was calculated by dividing total sugar in per cent with titrable acidity in per cent.

3.3.5.1.4. Pectin

Pectin as percent calcium pectate was determined as per the method suggested by Ranganna (1977)

The data was tabulated and subjected to analysis of variance, heterosis, genotypic correlation, path coefficient analysis and multiple regression as detailed in 3.3.4.

3.3.6. Evaluation of F₁ hybrids with overall best performance identified during December 2002 – March 2003

Based on the performance in the previous three seasons especially for disease resistance, further short listing of hybrids was done and they were evaluated during peak thrips activity (on 18-12 –2002) and Tospovirus incidence period based on previous work in this location (Kumar, 1988). They were grown without replication and were given special package of practices as follows in order to obtain their potential maximum yield under a system of cultivation without the use of inorganic pesticides.

A. Nursery management

- ◆ Neem cake @ 400g/m² while preparation
- ◆ Azospirillum treatment to seeds (4g/Kg of seed)
- ◆ Fytolon 2.5g/lit drenching whenever required

B. Main field preparation and transplanting

- ◆ Two tractor ploughing to a fine tilth
- ◆ FYM 25t/ha during last ploughing
- ◆ Basalin @ 4ml/lit before transplanting
- ◆ 50 – 300 – 50 Kg NPK/ha as basal; P applied as enriched FYM
- ◆ Just before transplanting, ZnSO₄ @2.5 Kg/ha +Borax @ 5 Kg/ha + 4 Kg/ha of each of Azospirillum, phosphobacteria and *Pseudomonas fluorescens*
- ◆ Transplanting was done during evening hours.

C. Aftercare

- ◆ First weeding at 25DAT followed by topdressing 50 Kg each of N and K/ha and earthed up
- ◆ Staking with bamboo sticks (40 DAT)
- ◆ Fertilizer application at 45 & 60 DAT @ 50 Kg each of N and K/ha
- ◆ Weeding 4 times from 25 DAT at fortnightly interval
- ◆ Irrigation once in 5-7 days
- ◆ CaCl₂ 0.1 per cent spray during fruit set and maturity to arrest fruit cracking if any
- ◆ Leaf extracts of *Adhatoda vasica* at 10 % at 10DAT and later at fortnightly interval and Panchagavya spray at 30 DAT.

3.3.7. Biochemical basis of combined virus resistance

For the assay of enzymes and electrophoretic studies, best hybrids and their parents along with susceptible check CO 3 were used for the study. Recently matured physiologically active leaf of five randomly selected representative plants of 60 day old was used. For studies on PO

and PPO enzymes activity, the samples were collected after 0, 24, 48, 72, 96 and 120 hr after grafting both the viruses simultaneously but in different portions of the stem, whereas for isozyme analysis of PO and PPO, samples collected after 0, 48 and 96 hr were used. In case of studies on PAL enzyme activity and crude protein using SDS-PAGE, samples after 48 hr alone were used.

3.3.7.1. Assay of enzymes

Leaf samples (200 mg) obtained from different time interval were homogenized in chilled pestle and mortar with 1 ml of cold 0.1 M phosphate buffer (p^H 6.5). The extract was centrifuged at 6000 rpm for 10 minutes at 4° C in a refrigerated centrifuge and the supernatant was used as enzyme extract.

3.3.7.1.1. Peroxidase

Peroxidase activity was assayed, following the method described by Srivastava (1987). Reaction mixture consists of 1.5 ml of guaiacol solution, 100 µl of enzyme preparation and 100 µl of 1% H₂O₂. At the start of the enzyme reaction, the absorbance of the mixture was set to zero at 420 nm and change in the absorbance were recorded at 30 seconds intervals. Boiled enzyme preparation was kept as control. Peroxidase activity was expressed as changes in absorbance per minute per g fresh weight.

3.3.7.1.2. Polyphenol oxidase

Polyphenoloxidase activity was assayed using the method described by Srivastava (1987). Standard reaction mixture contained 1.5 ml of 0.1 M phosphate buffer (pH 6.5), 0.5 ml of enzyme preparation and 0.5 ml of 0.01 M catechol. At the start of the enzyme reaction the absorbance was set to zero at 495 nm. The changes in the absorbance were recorded at 30 seconds intervals and the polyphenol oxidase activity was expressed as changes in the OD of the reaction mixture per minute per 200 mg of fresh weight of tissue.

3.3.7.1.3. Phenylalanine ammonia-lyase (PAL)

The PAL activity was determined as per the procedure given by Dickerson *et al.* (1984). The reaction mixture contained 0.4 ml of the sample extract, 0.5 ml of 0.1 M borate buffer (pH 8.8) and 0.5 ml of 12 mM L- phenylalanine in the same buffer. The reference cell contained 0.4 ml of the sample extract and 1 ml of borate buffer. PAL activity was determined at 30° C by direct spectrophotometer measurement of L- phenylalanine to trans – cinnamic acid at 290 nm. The amount of trans – cinnamic acid synthesized per minute was calculated using the extinction coefficient of $9630 \text{ m}^{-1} \text{ cm}^{-1}$ for trans – cinnamic acid in 0.1M of borate buffer. The absorbance 9630 is equal to 1 mol/ml/min. or the absorbance is 0.963, the product formed is 100 n mol/ml/min. The enzyme activity was expressed as $\mu\text{mol min}^{-1} \text{ g}^{-1}$ fresh weight.

3.3.7.2. Sodium Dodecyl Sulphate Polyacrylamide Gel Electrophoresis (SDS-PAGE) (Laemmli, 1970)

1. Acrylamide solution

Thirty gram of acrylamide and 0.8g of bisacrylamide (Sigma, USA) were dissolved in 60 ml of distilled water and the final volume was made upto 100 ml and stored at 4°C in a brown bottle.

2. Separating gel buffer

A quantity of 18.15g of Trizma base was dissolved in 60 ml deionized water, the pH was adjusted to 8.8 with HCl and the volume was made up to 100 ml.

3. Stacking gel buffer

Six gram of Trisma base was dissolved in 60 ml of deionized water and the pH adjusted to 6.8 with HCl. Then, the volume was made upto 100 ml.

4. Sodium dodecyl sulphate (SDS, 10%)

Ten gram of sodium dodecyl sulphate dissolved in 100 ml of distilled water.

5. Ammonium persulphate (10%, APS)

100 of ammonium persulphate was dissolved in one ml of distilled water.

N, N, N¹, N¹ –Tetramethyl ethylene diamine (TEMED).

Separating gel composition (volume 15 ml)

Stock solutions	Final stock solution (ml)			
	Final acrylamide concentration			
	8%	10%	12%	15%
Acrylamide mixture	4.00	5.000	6.000	7.500
1.5M Tris-HCl (pH 8.8)	3.75	3.750	3.750	3.750
10% SDS	0.10	0.100	0.100	0.100
Distilled water	7.25	6.250	5.250	3.750
10% APS	0.05	0.050	0.050	0.050
TEMED	0.01	0.010	0.010	0.010

Stacking gel composition (4%, 5 ml)

Acrylamide mixture	0.650 ml
0.5 M Tris-HCl; pH 6.8	1.250 ml
10% SDS	0.050 ml
Distilled water	3.050 ml
10% APS	0.025 ml
TEMED	0.005 ml

Preparation of separating and stacking gel

Acrylamide stock, corresponding Tris buffer, 10% SDS and water were mixed and degassed in vacuum for 5 min. Then 10% APS and TEMED were added and mixed well. The stock solution was used immediately.

Sample buffer

Glycerol	20% (v/v)
Tris-HCl, pH 6.8	0.125 M
Na ₂ EDTA	5 mM
SDS	20%
Bromophenol blue	0.1% (w/v)
2-mercaptoethanol	1% (v/v)

Electrode buffer (5%)

Glycine : 72.0 g

SDS : 5.0 g

Trizma base : 15.0 g

Contents were dissolved in 350 ml of distilled water and the volume was made upto 1000 ml with distilled water.

Staining solution

Coomassie Brilliant Blue R 250 : 100 mg

Acetic acid : 10 ml

Methanol : 40 ml

Water : 50 ml

Destaining solution

Acetic acid : 10 ml

Methanol : 40 ml

Water : 50 ml

100 μ of protein different treatments was taken and mixed with 10 μ l of sample buffer in microfuge tube, boiled for 4 min and incubated for at 4°C for 30 minutes. Then the samples containing equal amount of proteins were loaded into the wells of polyacrylamide gels. The medium range molecular weight markers (Bangalore Genei, India) were used. Electrophoresis was carried out at constant voltage of 75 volts. The gels were stained with 0.2% comassie brilliant blue (R 250) solution. Based on the Rf value of each protein band, the molecular weight was calculated.

3.3.7.3. Gel electrophoresis

I. Extraction buffer

The extraction buffer used is given below:

Phosphate buffer (pH 7.0)

0.1 M NaH₂PO₄ . 2H₂O

0.1 M Na₂HPO₄ . 2H₂O

II. Enzyme extraction

One gram of tomato leaves at different time interval after grafting diseased tissue on healthy plant and the samples were extracted with 2 ml of 0.1M phosphate buffer (pH 7.0). The homogenate was centrifuged at 6000 rpm for 15 minutes at 4°C in a refrigerated centrifuge and the supernatant was used for isozyme analysis.

A non-denaturing get (without SDS) was run to separate the isozymes. Acrylamide was polymerized with bisacrylamide as a cross linking reagent in the presence of ammonium per sulphate as catalyst and a chain initiator TEMED.

III. Reagents and gel preparation for isozyme

Separating gel (8%) (pH 8.8)	Stacking gel (4%) (pH 6.8)
------------------------------	----------------------------

		1 gel	2 gel
Acrylamide	2.00 ml	0.65 ml	1.30 ml
Distilled water	3.625 ml	8.25 ml	6.5 ml
Tris (8.8)	1.875 ml	1.25 ml	2.5 ml
APS	25 μ l	25 μ l	50 μ l
Temed	5 μ l	5 μ l	10 μ l

APS - Ammonium per sulphate

TEMED - (N, N, N', N' - Tetra methyl ethylenediamine)

IV. Sample buffer

Deionised water	-	2.6 ml
0.5 M Tris HCl pH 6.8	-	1.0 ml
2-mercaptoethanol	-	0.8 ml
Glycerol	-	1.6 ml
10% (W/V) SDS	-	1.6 ml
0.5% Bromophenol blue	-	0.4 ml

V. Electrode buffer

Glycine	-	4.320 g
Tris-base	-	900 mg
Distilled water	-	300 ml

VI. Procedure

1. The glass plates, comb, spacers and the electrophoresis apparatus were cleaned thoroughly and wiped. The spacers were placed on the edges in between the plates and clamped with clips tightly.

2. 1.0% molten agar was poured along the sides of the plate and allowed to solidify and it seals the edges.
3. The separating gel mixture was gently mixed without forming bubbles and carefully poured in between the glass plates. A layer of distilled water was added above the gel and allowed to polymerize.
4. The stacking gel mixture were mixed gently and poured in between the glass plates above the separating gel. The comb was placed between the plates and polymerization occurred in about 30 min.
5. After polymerization, the comb and clips were removed carefully and the glass plates with the polymerized gel were placed in the electrophoresis apparatus.
6. The electrode buffer was added to the tank and the air bubbles were removed.

VII. Loading the sample

The enzyme extract was loaded with the help of a micro syringe into the wells of the acrylamide gel. The power pack was put on and the gel was run at 4°C in a cold room. An initial current of 15mA was given till the loaded samples transverse the stacking gel and after reaching the separating gel the current supply was increased to 30 mA and the gel was run till the samples reached the bottom of the gel plate. The electrophoresis was stopped and the gel plate was removed from the unit and the spacers were removed. The two plates were separated and the separating gel was gently removed from the plate without any damage and stained.

VIII. Activity gel electrophoresis

1. Polyphenol oxidase (PPO)

After native electrophoresis, the gel was equilibrated for 30 min in 0.1 M phosphate buffer (pH 7.0) containing 0.1% p-phenylenediamine followed by 10 mM catechol in the same buffer. The

addition of catechol was followed by a gentle shaking which resulted in the appearance of dark brown color discrete brown bands (Jayaraman *et al.*, 1987).

2. Peroxidase (PO)

To study the expression pattern of different isoforms of peroxidases in the different treatments, activity gel electrophoresis was carried out. For native anionic polyacrylamide gel electrophoresis, resolving gel of 8 per cent acrylamide concentration and stacking gel of 4 per cent acrylamide concentration were prepared. After electrophoresis, the gels were incubated in the solution containing 1 per cent benzidine in Acetate buffer. Then drops of 30 per cent H₂O₂, were added with constant shaking till the bands appear. After shaking, the gel was washed with distilled water and photographed (Sindhu *et al.*, 1984).

3.3.8. Physical manipulation of source - sink relationship in the hybrid H 24 X CLN 2123A (December 2002 – March 2003)

An experiment was laid out during December 2002 – March 2003 to observe the effect of different physical treatments on source and sink relationship in the hybrid H 24 X CLN 2123A. The treatment details were as follows

- T₁ : Nipping (40 DAT, afterwards once in 20 days up to first harvest, specification is top 1 cm)
- T₂ : Flower thinning (two/truss)
- T₃ : Truss thinning (leaving six basal trusses i.e, earliest appearing ones)
- T₄ : Alternate truss thinning (leaving six earliest appearing ones after removing alternate inflorescence).
- T₅ : Control

Following observation was made in addition to plant height, branches per plant, number of fruits per plant, average fruit weight, yield per plant, TSS and acidity

3.3.8.1. Polar diameter (cm)

Length of fruit at ripening was measured from the place of fruit attachment to the tip of fruit using vernier calipers and was considered as polar diameter.

3.3.8.2. Equatorial diameter (cm)

Length of fruit placed as such i.e. opposite to polar ends at the region of maximum girth at ripening was considered as equatorial diameter.

3.3.8.3. Outer pericarp wall thickness (cm)

Fruit was transversely cut and thickness of outer wall of the pericarp was measured using screw gauge.

3.3.8.4. Septa thickness (cm)

Thickness of the radial wall, which separates the adjacent locules was measured using screw gauge and was recorded as septa thickness.

CHAPTER IV

RESULTS

The analysis of variance for the parents and hybrids in randomized block design showed highly significant differences among the genotypes for all the characters in all the three seasons studied (Table 1a, 1b and 2).

4.1. *Per se* performance

4.1.1. Diallel experiment for the season I (January – April 2002)

4.1.1.1. Plant height (Table 3)

The plant height ranged from 49.2 (LE 415) to 87.10 cm (CLN 2123A) in parents and 46.10 (LCR 3 x LE 415) to 104.00 cm (H 24 x CLN 2123A) in hybrids. The mean for the hybrids was 66.64 cm whereas for the parents it was 63.13 cm.

4.1.1.2. Number of branches per plant (Table 4)

The number of branches per plant was highest in the parent LCR 9 (7.50) and the lowest in LCR 3 (5.90). Among the hybrids, H 24 x LCR 9 recorded the highest number of branches per plant (9.50) followed by CLN 2123A x LCR 9 (8.60) and LCR 9 x H 24 (8.00). The hybrid LCR 3 x LE 415 recorded the lowest number of branches per plant (5.20). Only narrow difference was exhibited in the mean of parents and hybrids.

4.1.1.3. Days to 50 per cent flowering (Table 5)

Among the parents, LCR 3 recorded 50 per cent flowering in 26.00 DAT, whereas, LCR 9 took a maximum of 38.00 days for the same. Among the hybrids, LCR 3 x H 24 and LE 415 x LCR 3 exhibited earliest flowering (26.00 days) followed by LCR 3 x LE 415 (26.50) and LCR 1 x LCR 3 (26.50), while LCR 9 x CLN 2123A arrived at 50 per cent flowering only after 38.50 days. The mean for hybrids was 32.37 days whereas for the parents it was 31.92 days.

4.1.1.4. Number of fruits per plant (Table 6)

The number of fruits per plant was highest in the parent LCR 3 (80.70) and the lowest in LCR 9 (20.40). Among the hybrids, H 24 x CLN 2123A recorded the highest number of fruits (79.70) followed by H 24 x LE 415 (77.5) and LE 415 x LCR 3 (74.20). The hybrid LCR 1 x LCR 9 recorded the lowest number of fruits per plant (23.30). The mean for hybrids was 43.83 and for parents it was 39.21.

4.1.1.5. Fruit weight (Table 7)

Maximum average fruit weight of 111.22 g was recorded by the parent LCR 1 and the minimum of 28.05 g in LE 415. Among the hybrids, this trait ranged from 26.60 to 81.25 g. Crosses involving LCR 3 as one of the parents recorded lower fruit weight. Maximum fruit weight was registered by LCR 1 x LCR 9 (81.25 g) followed by CLN 2123A x LCR 9 (73.00 g). Minimum fruit weight of 26.60g was recorded in both LCR 3 x CLN 2123A and LCR 3 x LE 415.

4.1.1.6. Yield per plant (Table 8)

Among the parents, LCR 3 and LE 415 registered highest and lowest yield of 2.66 and 0.81 kg per plant respectively. Among the hybrids, the yield ranged from 1.09 to 3.25 kg per plant. However crosses involving LCR 3 as one of the parents recorded comparatively lower yield per plant. Among the hybrids, highest yield was recorded in H 24 x LE 415 (3.25 kg) followed by H 24 x CLN 2123A (3.12 kg) and H 24 x LCR 9 (2.85 kg), while the hybrid LCR 1 x LCR 3 recorded least yield of 1.09 kg per plant. Mean fruit yield was higher in the hybrids than the parents.

4.1.1.7. Total Soluble Solids (TSS) (Table 9)

TSS of fruits varied from 3.52 (LCR 9) to 4.54 (LE 415)°Brix in parents and 3.29 (CLN 2123A x LE 415) to 4.90 (LCR1 x LCR 9) °Brix in hybrids. There was only little difference noticed in mean TSS value for both parents and hybrids.

4.1.1.8. Acidity (Table 10)

The acidity ranged from 0.38 (LCR 9) to 0.61 (LCR 9) per cent in parents and 0.42 (H 24 x LCR 9) to 0.62 (LCR 3 x CLN 2123A) per cent in the hybrids.

4.1.1.9. Ascorbic acid (Table 11)

The parents H 24 and LCR 9 recorded the lowest value for ascorbic acid (26.75 mg/100g) whereas LCR 3 recorded the highest value (33.72). In hybrids, it ranged from LCR 1 x LCR 3 (23.20) to 37.21 (LCR 3 x LCR 9 and CLN 2123A x LCR 9) mg/100g.

4.1.1.10. Lycopene (Table 12)

The highest lycopene content of 5.45 mg/100g was observed in the parent H 24 whereas the lowest of 2.19 mg/100g in LCR 3. Among the hybrids, LCR 3 x CLN 2123A registered highest lycopene content (4.86) followed by H 24 x LCR 1 (4.85) and CLN 2123A x LCR 3 (4.65), while the hybrid LCR 3 x LCR 1 recorded the lowest value of 2.18 mg /100g for this trait.

4.1.1.11. Total phenol (Table 13)

Among the parents LE 415 recorded the highest value of 305.90 mg/100g and whereas LCR 9 recorded the lowest value of 237.05 mg/100g. In hybrids, this trait ranged from 192.55 (LCR 1 x LCR 3) to 307.60 (H 24 x CLN 2123A) mg/100g. The mean total phenol content of parents and hybrids were 274.14 and 255.46 mg/100g respectively. Generally crosses exhibiting H 24 as one of parents showed higher value for this trait.

4.1.1.12. OD phenol (Table 14)

OD phenol content in the leaves varied from 96.95 (LCR 9) to 271.45 (LE 415) mg/100g in parents and 56.40 (LCR1 x CLN 2123A) to 247.35 (H 24 x LCR 1) mg/100g in the hybrids. Parental mean was significantly higher (173.90) than that of hybrids (142.85).

4.1.1.13. Disease incidence at 15 DAT (Table 15)

Since no TLCV disease incidence was noticed at this stage, both Tv and total PDI remained the same. The Tv incidence at 15 DAT ranged from 0.00 (LE 415) to 10.71 (LCR 1) per cent in the parents. In hybrids, no incidence of Tv was noticed in LCR 3 x LCR 1 and LE 415 x LCR 3 at this stage. CLN 2123A x LE 415 recorded the maximum incidence of 22.00 per cent followed by H 24 x LE 415 (17.85 per cent), while the susceptible check variety recorded 14.1 per cent disease incidence in total including PDI due to combined infection (1.27).

4.1.1.14. Disease incidence at 30 DAT (Table 16a, 16b and 16c)

Except the parent LCR 1, all other parents exhibited no incidence of TLCV whereas all were prone to Tv attack. Total PDI ranged from 7.14 (LCR 9 and LCR 3) to 17.86 (CLN 2123A) in parents (Table 16c). Among the hybrids least incidence of Tosspovirus was exhibited in LE 415 x LCR 3 (5.50 per cent) followed by LE 415 x LCR 9 (6.14) and LCR 9 x LCR 1 (6.55) (Table 16b). Total PDI ranged from 9.79 (LCR 9 x H 24) to 38.78 (CLN 2123A x H 24) per cent. The susceptible check variety recorded 28.50 per cent disease incidence in total (Table 16c).

4.1.1.15. Disease incidence at 45 DAT (Table 17a, 17b and 17c)

In this stage also, all the parents exhibited no incidence TLCV except LCR 1 (Table 17a). Maximum incidence of TLCV was recorded in LCR 3 x LCR 9 (16.88 per cent) whereas 14.14 per cent of susceptible check variety plants were infected (Table 17a). Tv disease incidence in hybrids ranged from 5.50 (LE 415 x LCR 3) to 35.45 (LCR 9 x CLN 2123A) per cent whereas the susceptible check variety recorded 23.32 per cent incidence (Table 17b). With respect to total PDI, LCR 3 recorded the minimum incidence of 9.65 per cent among the parents and H 24 x LCR 3 and H 24 x LCR 1 (14.58 and 15.15 per cent respectively) among the hybrids. The mean percentage disease incidence of hybrids was higher than that of parents.

4.1.1.16. Disease incidence at 60 DAT (Table 18a, 18b and 18c)

Except LCR 9 and LCR 1, all other parents recorded no TLCV incidence (Table 18a). Crosses involving CLN 2123A as one of the parents recorded either lower value or no incidence of TLCV (Table 18a). LE 415 registered maximum Tv disease incidence (25.00 per cent) as a parent. The hybrids LE 415 x LCR 3 (5.50 per cent) followed by LCR 9 x H 24 (8.00 per cent) and CLN 2123A x LCR 3 (10.00 per cent) recorded lowest PDI values for Tv incidence whereas the susceptible check variety registered 38.25 per cent incidence (Table 18b). Total PDI ranged from 19.65 (H 24) to 35.72 (LCR 9) in parents and 15.15 (H 24 x LCR 1) to 67.14 (CLN 2123A x H24) in hybrids. The mean total PDI values for both hybrids and parents were 34.23 and 25.16 per cent respectively.

4.1.1.17. Disease incidence at 75 DAT (Table 19a, 19b and 19c)

Parents LCR 3, CLN 2123A, H 24, LE 415 and hybrids LCR 9 x CLN 2123A, LCR 1 x CLN 2123A, H 24 x CLN 2123A and H 24 x LCR 1 recorded no TLCV incidence at this stage (Table 19a). Generally crosses involving CLN 2123A as one of the parents registered lower or no incidence of TLCV. Tv incidence ranged from 14.26 (LCR 9) to 28.57 (LE 415) per cent in parents and 5.50 (LE 415 x LCR 3) to 63.57 (CLN 2123A x H 24) per cent in hybrids (Table 19b). LCR 3 (21.79) among the parents and H 24 x LCR 1 (15.15) and H 24 x LCR 9 (21.76) among the hybrids, recorded lowest total PDI whereas the susceptible check variety registered 83.91 per cent (Table 19c).

4.1.1.18. Disease incidence at 90 DAT (Table 20a, 20b, 20c)

Only CLN 2123A and LE 415 among the parents and LCR 9 x CLN 2123A, LCR 1 x CLN 2123A and H 24 x CLN 2123A among the hybrids recorded no TLCV incidence (Table 20a). Maximum incidence of TLCV was noticed in parent LCR 9 (28.57) and hybrid LE 415 x LCR 9 (34.57) where as the susceptible check variety value was only 19.63 per cent (Table 20a). Incase of Tv incidence the least incidence was noticed in LCR 1 (14.28) among the parents whereas LE 415 x LCR 3 (5.50) and LCR 9 x H 24 (11.14) among the hybrids, while the

susceptible check variety recorded a maximum Tv incidence of 52.09 per cent (Table 20b). Total PDI ranged from 23.81 (CLN 2123A) to 42.86 (LCR 9) per cent in parents and 16.93 (H 24 x LCR 1) to 76.07 (CLN 2123A x H 24) where the corresponding figure for the susceptible check variety was 87.98 per cent.

4.1.1.19. Coefficient of Infection (CI) for TLCV incidence at 75 DAT (Table 21)

CI for TLCV ranged from 0.00 (LCR 3, CLN 2123A, H 24 and LE 415) to 12.51 (LCR 9) among the parents and 0.00 (LCR 9 x CLN 2123A, LCR 1 x CLN 2123A, H 24 x CLN 2123A and H 24 x LCR 1) to 22.89 (LCR 3 x LCR 9) among the hybrids. The mean CI for parents and hybrids was 2.68 and 4.51 respectively. Generally crosses with CLN 2123A as any one of the parents registered lower values of CI.

4.1.2. Diallel experiment for the season II (March – June 2002)

4.1.2.1. Disease incidence at 15 DAT (Tables 22a, 22b and 22c)

There existed no significant differences among the treatments involving parents and hybrids for TLCV incidence at 15 DAT (Table 22a). Tv incidence ranged from 0.00 (LE 415) to 4.44 (LCR 1) per cent among the parents and 0.00 (LCR 9 x LCR 3, LCR 9 x H 24, LCR 1 x LCR 9, LCR 1 x H 24, LCR 1 x CLN 2123A, LCR 3 x LCR 9, LCR 3 x LCR 1, LCR 3 x CLN 2123A, CLN 2123A x H 24, CLN 2123A x LE 415, H 24 x CLN 2123A and H 24 x LE 415) to 14.28 (LCR 3 x LE 415) per cent in the hybrids. The corresponding susceptible check variety value was 12.00 per cent (Table 22b). Regarding total PDI, LE 415 among the parents and the same hybrids as mentioned for Tv incidence except LCR 1 x H 24, CLN 2123A x H 24 and H 24 x LE 415 recorded no disease incidence (Table 22c).

4.1.2.2. Disease incidence at 30 DAT (Table 23a, 23b and 23c)

Generally hybrids involving CLN 2123A registered lowest TLCV values at this stage. Tv incidence ranged from 1.67 (LE 415) to 11.61 (LCR 3) per cent among parents and 0.00 (LCR 1 x LCR 9, LCR 1 x CLN 2123 A, LCR 3 x LCR 1 and H 24 x CLN 2123A) to 25.06 (LE 415 x CLN 2123A) per cent. While the corresponding susceptible check variety recorded 24.35 per cent infection (Table 23b). The hybrids recorded zero values for Tv incidence also recorded zero per cent disease incidence in total whereas as the susceptible check variety yielded 28.28 per cent infection (Table 23c).

4.1.2.3. Disease incidence at 45 DAT (Table 24a, 24b and 24c)

The TLCV disease incidence was zero in parent CLN 2123A which was also reflected in its crosses (Table 24a). The highest value was recorded in the hybrid LCR 1 x LCR 9 (25.00 per cent) (Table 24a). Tv incidence ranged from 6.67 (LE 415) to 16.54 (CLN 2123A) among the parents and 5.00 (LCR1 x CLN 2123A) to 38.10 (LCR 3 x LE 415) among the hybrids while the corresponding value in the susceptible check variety was 30.32 per cent (Table 24b). Regarding total PDI, hybrids H 24 x CLN 2123A (7.32), LCR 1 x CLN 2123A (7.78) and H 24 x LCR 1 (9.13 per cent) recorded lowest value whereas in susceptible variety the value was 49.39 per cent (Table 24c).

4.1.2.4. Disease incidence at 60 DAT (Table 25a, 25b and 25 c)

No incidence of TLCV was noticed in the parent CLN 2123A and in the hybrids LCR 9 x H 24, LCR 9 x LE 415, LCR 1 x CLN 2123A, CLN 2123A x LCR 9 and H 24 x LCR 1 whereas the susceptible check variety recorded 28.11 per cent incidence (Table 25a). Tv incidence ranged from 6.66 (LE 415) to 21.21 (CLN 2123A) per cent among the parents and 9.10 (H 24 x LE 415) to 51.43 (LCR 9 x CLN 2123A) per cent in the hybrids (Table 25b). With respect to total PDI, it ranged from 11.66 (LE 415) to 28.87 (LCR 3) per cent among the parents and 12.59

(CLN 2123A x LCR 1) to 59.52 (LCR 3 x LE 415) per cent among hybrids. The susceptible check variety at this stage experienced 60.03 per cent incidence in total (Table 25c).

4.1.2.5. Disease incidence at 75 DAT (Table 26a, 26b and 26c)

TLCV incidence at this stage ranged from 5.00 (CLN 2123A) to 19.17 (LE 415) among parents and 0.00 (CLN 2123A x LCR 9, LCR 9 x H 24, LCR 9 x LE 415) to 26.79 (LCR 9 x LCR 3) per cent among the hybrids (Table 26a). Least incidence of Tv was noticed in the parent LE 415 (8.30 per cent) and in the hybrids H 24 x LCR 9 (12.08), H 24 x LCR 3 (13.07) and H 24 x CLN 2123A (14.65 per cent) (Table 26b), while least total disease incidence was noticed in LE 415 (27.47 per cent) among the parents and H 24 x CLN 2123A (19.19) and LCR 1 x CLN 2123A (22.14) among the hybrids. The corresponding susceptible check variety value was 68.87 per cent (Table 26c).

4.1.2.6. Disease incidence at 90 DAT (Table 27a, 27b and 27c)

At this stage the least PDI for TLCV was recorded in CLN 2123A (5.00 per cent) among the parents while among hybrids, the combinations LCR 9 x H 24 and LCR 9 x LE 415 recorded the least incidence of 0.00 per cent. The susceptible check variety recorded 32.64 per cent TLCV incidence (Table 27a). Tv incidence ranged from 8.33 (LE 415) to 28.89 (CLN 2123A) per cent among the parents and 13.07 (H 24 x LCR 3) to 53.57 (LCR 3 x LE 415) per cent among the hybrids (Table 27b). Least total PDI was noticed in the parent LE 415 (27.50 per cent) and in the hybrids H 24 x CLN 2123A and CLN 2123A x LCR 1 (21.97 and 22.96 per cent respectively). At this stage, the corresponding susceptible check variety recorded 71.34 per cent total PDI (Table 27c).

4.1.2.7. Coefficient of Infection (CI) for TLCV incidence at 75 DAT (Table 28)

Least CI for TLCV at 75 DAT was noticed in the parent CLN 2123A (0.96). In the hybrids, generally those crosses involving CLN 2123A as one of the parents recorded the least

CI value. The hybrids LCR 9 x H 24, LCR 9 x LE 415 and CLN 2123A x LCR 9 recorded zero value.

4.1.3. *Per se* performance for the season III (July –October 2002)

Eight hybrids were selected based on the *per se* performance of those hybrids in the previous two seasons and evaluated during the III season. The results obtained were given below.

4.1.3.1. Plant height (Table 29a)

The plant height ranged from 64.50 (H 24 x LCR 1) to 86.60 cm (H 24 x LCR 9) in hybrids. The commercial hybrid COTH 1 recorded the value of 69.00 cm.

4.1.3.2. Number of branches per plant (Table 29a)

The number of branches per plant was highest in the cross H 24 x LCR 9 (8.6) and the lowest in LCR 1 x LE 415 (6.13).

4.1.3.3. Days to 50 per cent flowering (Table 29a)

Earlier flowering was noticed in the hybrid H 24 x CLN 2123A and the commercial hybrid Ankush (28.00 days). Among parents, earlier flowering was noticed in CLN 2123A and H 24 (each 28.67 days).

4.1.3.4. Number of fruits per plant (Table 29a)

The number of fruits per plant ranged from 24.40 in LCR 1 to 35.35 in H 24 in the parents. Among the hybrids, H 24 x CLN 2123A recorded highest number of fruits of 73.33 followed by H 24 x LCR 9 (45.07).

4.1.3.5. Fruit weight (Table 29a)

Among the parents, the individual fruit weight varied between 35.03 g in LE 415 and 117.35g in LCR 1. Among the hybrids, maximum fruit weight was registered in LCR 1 x CLN 2123A (75.74g) whereas the commercial hybrid Ankush recorded an average fruit weight of 66.03g.

4.1.3.6. Yield per plant (Table 29a)

Among the parents, LCR 1 recorded highest yield of 2.86kg whereas among the hybrids, H 24 x CLN 2123A recorded highest yield of 3.20 kg followed by LCR 1 x LE 415 (2.73kg). The commercial hybrids COTH 1 and Ankush recorded the yield of 2.34 and 2.73 kg per plant respectively.

4.1.3.7. pH of the fruit juice (Table 29b)

The pH of the fruit juice ranged from 3.57 to 4.06 in the parents CLN 2123A and H 24 respectively. The corresponding value for the hybrids varied from 3.57 (H 24 x LCR 9) to 4.06 (LCR 1 x LE 415).

4.1.3.8. Acidity (Table 29b)

Among the parents, CLN 2123A registered highest value of 0.60 per cent whereas among the hybrids, H 24 x CLN 2123A recorded the same of 0.58 per cent. The commercial hybrids COTH 1 and Ankush recorded acidity of 0.49 and 0.58 per cent respectively.

4.1.3.9. Total Soluble Solids (TSS) (Table 29b)

The TSS ranged between 4.64 (LE 415) and 6.04° Brix (CLN 2123A) among the parents. Among the hybrids, CLN 2123A x LCR 9 registered highest TSS of 5.62° Brix followed by LCR 1 x CLN 2123A (5.57 per cent).

4.1.3.10. Ascorbic acid (Table 29b)

Among the parents LE 415 recorded highest ascorbic acid content of 27.37 mg per 100g and CLN 2123A recorded the lowest of 22.88 mg per 100g. Among the hybrids, ascorbic acid content ranged from 17.36 (LCR 1 x CLN 2123A) to 29.62mg per 100g (CLN 2123A x LCR 9). COTH 1 and Ankush recorded comparatively lower values of 22.71 and 22.22 mg per 100g respectively.

4.1.3.11. Lycopene (Table 29b)

Among the parents the highest lycopene content of 4.62 mg/100g was observed in H 24 whereas the lowest (3.17 mg/100g) in LCR 1. Among the hybrids, H 24 x LCR 1 registered high lycopene content (4.75) followed by LCR 1 x LE 415 (4.24) and LCR 1 x CLN (4.20).

4.1.3.12. Total sugar (Table 29b)

The parents H 24 and LCR 1 recorded highest and lowest value total sugar of 2.87 and 3.57 per cent among parents. The hybrid H 24 x LCR 9 recorded highest total sugar (3.87 per cent) followed by LCR 1 x LE 415 (3.57 per cent). The commercial hybrid COTH 1 recorded the highest value (4.39 per cent) of all the fifteen treatments studied.

4.1.3.13. Reducing sugars (Table 29b)

Among the parents, reducing sugar content ranged from 2.41 (LCR 1) to 2.92 per cent (CLN 2123A), while, the hybrids ranged between 2.36 (LCR 1 x CLN 2123A) and 3.41 (H 24 x LCR 9). The commercial hybrid COTH 1 registered the highest value of 3.91 per cent of all the fifteen treatments studied.

4.1.3.14. Sugar to acid ratio (Table 29b)

Among the parents highest value of sugar to acid ratio was noticed in LCR 1 (7.67) while in hybrids, LCR 1 x LE 415 (8.02) recorded the same. The commercial hybrid COTH 1 recorded the highest value of 8.99 among all the fifteen treatment studied.

4.1.3.15. Pectin (Table 29b)

A wide range of 0.14 (LE 415) to 0.45 per cent (CLN 2123A) pectin content was noticed among the parents. Similarly hybrids also exhibited wide range of 0.09 (H 24 x CLN 2123A) to 0.33 per cent (H 24 x LCR 9).

4.1.3.16. Total phenol (Table 29b)

The highest value of 301.77, 309.07 and 217.03 mg per 100g was exhibited by LE 415 among the parents, H 24 x LCR 1 among the hybrids and COTH 1 respectively.

4.1.3.17. OD phenol (Table 29b)

Among the parents, OD phenol content ranged from 100.62 (LCR 9) to 236.10 mg per 100g (LE 415). Among the hybrids, LCR 1 x CLN 2123A recorded lowest value of 103.10 mg per 100 g and H 24 x CLN 2123A recorded the highest value of 214.93 mg/100g.

4.1.3.18. Disease incidence at 15 DAT (Table 30)

No incidence of TLCV was noticed in five out of eight hybrids, three out of five parents and both the commercial hybrids, while Tv incidence ranged from 0.00 per cent in LE 415 and LCR 1 to 4.16 per cent in LCR 9 among the parents. Least incidence of Tv was noticed in LE 415 x LCR 1 (1.85 per cent) whereas maximum incidence was recorded in LCR 1 x H 24 (6.65 per cent). The commercial hybrids COTH 1 and Ankush exhibited 6.25 and 4.34 per cent Tv infection at 15 DAT. The susceptible check variety CO 3 recorded 12.01 per cent total disease incidence.

4.1.3.19. Disease incidence at 30 DAT (Table 30)

Among the parents LCR 1 and LCR 9 recorded the least incidence of TLCV, Tv (4.16 per cent each) and total PDI (8.32). Among the hybrids, LCR 1 x H 24, H 24 x LCR 1, H 24 x CLN 2123A recorded no TLCV incidence whereas least incidence of tospovirus was noticed in LE 415 x LCR 1 (3.87 per cent) followed by H 24 x CLN 2123A (4.95 per cent). The total PDI ranged between 4.95 in H 24 x CLN 2123A and 22.50 in H 24 x LCR 9 among the crosses. The susceptible check variety CO 3 recorded 30.76 per cent total disease incidence at this stage.

4.1.3.20. Disease incidence at 45 DAT (Table 31)

Lowest incidence of TLCV was recorded in H 24 (5.85 per cent) among the parents and H 24 x CLN 2123A (0.00 per cent) among the hybrids. Among the parents Tv incidence ranged from 8.33 (LCR 1) to 16.03 per cent (CLN 2123A) whereas among the hybrids, it ranged from 6.34 (H 24 x LCR 1) to 16.78 per cent (CLN 2123A x LCR 9). The same parents and hybrids exhibited similar trend for total PDI. The susceptible check variety CO 3 recorded 40.93 per cent total disease incidence.

4.1.3.21. Disease incidence at 60 DAT (Table 31)

The parents H 24 recorded lowest incidence of incidence of both TLCV (5.85 per cent) and Tv (7.89 per cent) hence expressed lowest total disease incidence of 13.74 per cent. Among the hybrids, H 24 x CLN 2123A recorded no TLCV incidence whereas the least incidence of Tv was noticed in H 24 x LCR 1 (8.42 per cent). The total PDI ranged between 10.60 (H 24 x LCR 1) and 25.24 per cent in the hybrid CLN 2123A x LCR 9 among the hybrids studied. The commercial hybrids COTH 1, Ankush and susceptible check variety CO 3 recorded the total PDI values of 25.00, 42.48 and 52.39 per cent respectively.

4.1.3.25. Disease incidence at 75 DAT (Table 32)

Among the parents, the lowest incidence of TLCV was exhibited in CLN 2123A (6.46 per cent) whereas LCR 1 exhibited the lowest incidence of Tv and total disease (14.58 and 22.91 respectively). Among the hybrids, TLCV incidence varied from 3.71 (LE 415 x LCR 1) to 9.12 per cent (H 24 x LCR 9) whereas Tv incidence ranged from 10.60 (H 24 x LCR 1) to 18.54 per cent (LCR 1 x LE 415). Lowest total PDI was noticed in the hybrid H 24 x LCR 1(14.87) among the hybrids studied. The susceptible check variety CO 3 recorded 56.52 per cent total disease incidence.

4.1.3.26. Disease incidence at 90 DAT (Table 32)

The parent LCR 1 recorded least incidence of both the diseases TLCV (8.33 per cent) and Tv (16.66 per cent) hence recorded lowest total PDI of 25.00 per cent among the parents.

Lowest incidence of TLCV and Tv was recorded in LE 415 x LCR 1 (3.71 per cent) and LCR 1 x H 24 (10.80 per cent) respectively. Total PDI ranged between 15.15 (LCR 1 x H 24) to 27.50 per cent (LCR 1 x CLN 2123A) among the hybrids. The susceptible check variety recorded 58.01 per cent incidence of both the diseases.

4.1.3.27. Coefficient of infection (CI) for TLCV at 75 DAT (Table 32)

Among the parents, CI ranged from 0.79 (CLN 2123A) to 4.76 (LE 415) while among the hybrids, the same ranged between 0.52 (H 24 x LCR 9) and 1.40 (LE 415 x LCR 1). The commercial hybrids COTH 1 and Ankush registered CI of 3.64 and 3.17 respectively.

4.1.4. *Per se* performance of field-grown best performed hybrids (December 2002 - March 2003)

Two identified hybrids H 24 x LCR 1 and H 24 x CLN 2123A were evaluated along with their parents and susceptible check variety under organic system of cultivation. The results are as follows (Table 33).

4.1.4.1. Disease incidence at 15 DAT

None of the entries registered TLCV incidence. Whereas minimum incidence of Tv was noticed in H 24 x LCR 1 (4.29 per cent). The susceptible check variety recorded 33.33 per cent Tv incidence at 15 DAT.

4.1.4.2. Disease incidence at 30 DAT

H 24 x CLN 2123A, LCR 1, H 24 and CO 3 recorded no incidence of TLCV. Tv incidence ranged from 9.86 (H 24 x LCR 1) to 50.00 (CO 3) per cent. Total disease incidence ranged from 11.20 (H 24 x LCR 1) to 50.00 (CO 3) per cent at this stage.

4.1.4.3. Disease incidence at 45 DAT

Only H 24 x CLN 2123A and CLN 2123A recorded no incidence of TLCV. Tv incidence ranged from 15.71 (H 24 x LCR 1) to 50.00 (CO 3) per cent. Total disease incidence was lowest in H 24 x LCR 1 (17.10 per cent) and maximum in CO 3 (70.37 per cent).

4.1.4.4. Disease incidence at 60 DAT

TLCV incidence ranged from 0.00 (CLN 2123A) to 33.33 (CO 3) per cent. Least incidence of Tv was noticed in H 24 x LCR 1 (18.57) while the susceptible check variety recorded 57.41 per cent incidence. Total PDI was minimum in the hybrids H 24 x LCR 1 (25.57 per cent) followed by H 24 x CLN 2123A (32.50 per cent) and maximum in susceptible check variety CO 3 (90.74 per cent).

4.1.4.5. Disease incidence at 75 DAT

No TLCV incidence was noticed in CLN 2123A whereas the susceptible check variety recorded 40.71 per cent incidence. Least incidence of Tv was noticed in H 24 x LCR 1 (20.00 per cent) while the susceptible check variety registered 57.41 per cent incidence. Total disease incidence ranged from 37.50 (H 24 x CLN 2123A) to 98.15 (CO 3) per cent.

4.1.4.6. Coefficient of infection for TLCV at 75 DAT

CI for TLCV ranged from 0.00 (CLN 2123A) to 14.48 (LCR 1).

4.1.4.7. Yield parameters

Number of fruits per plant ranged from 13.20 (LCR 1) to 70.70 (H 24 x CLN 2123A). Average fruit weight was maximum LCR 1 (101.27g) whereas it was lowest in H 24 x CLN 2123A (43.40g). Yield per plant was highest in H 24 x CLN 2123A with 3.07 kg under unprotected condition.

4.1.5. Physical manipulation of source - sink relationship in the hybrid H 24 X CLN 2123A (Table 34)

4.1.5.1. Plant height

The treatment T₃ recorded highest plant height (93.73cm) followed by T₅ (90.67cm) and T₄ (90.04cm). Notably T₁ recorded lowest of all (65.47 cm).

4.1.5.2. Number of branches

Treatment T₃ recorded maximum number of primary branches of 9.18. T₁ recorded the lowest number (7.67).

4.1.5.3. Number of fruits per plant

T₅ recorded the maximum of 76.58 fruits whereas treatment T₃ recorded lowest number of fruits (33.78).

4.1.5.4. Average fruit weight

Highest fruit weight of 57.23g was exhibited in T₃ whereas lowest fruit weight (40.67g) was noticed in T₅.

4.1.5.5. Polar diameter

Similar to average fruit weight, here also T₁ recorded highest polar diameter (4.57cm) whereas T₅ recorded lowest polar diameter (3.85cm).

4.1.5.6. Equatorial diameter

Here also T₁ and T₃ recorded maximum equatorial diameter of 5.01 and 4.92cm respected. As like above, treatment T₅ recorded lowest equatorial diameter (4.19cm).

4.1.5.7. Pericarp thickness

4.1.5.7.1. Outer wall thickness

Outer wall thickness of the Pericarp was found to be highest in treatment T₄ (0.58cm). Treatments T₅ and T₃ recorded lowest value of 0.42 and 0.44cm respectively.

4.1.5.7.2. Inner wall (septa) thickness

Treatment T₅ recorded highest septa thickness of 0.62cm respectively. The lowest thickness was observed in treatment T₅ (0.41cm) and T₂ (0.45cm).

4.1.5.8. TSS

In contrast above observations, TSS was found to be more in T₅ (5.97° Brix) whereas treatments T₄, T₃ recorded lowest value of 5.25 and 5.32° Brix respectively.

4.1.5.9. Total Acidity

Treatments T₁ and T₅ recorded higher values of total acidity (0.69 per cent). Lowest value was noticed by T₂ (0.50 per cent).

4.1.5.11. Yield per plant

Highest yield was noted in T₅ (3.11 kg). Lowest of all was recorded in treatments T₃ and T₄ namely 1.93, 1.92 kg respectively.

4.2. Heterosis

The magnitude of heterosis for different characters in all the thirty hybrids in case of diallel experiments were estimated as the performance of F₁ in comparison with mid parent (di), better parent (dii) and the best parent (diii) The lowest value was considered as the better and best parental value to work out heterosis for disease incidence, days to 50% flowering and pH. Negative heterosis was considered as better index for these traits.

4.2.1.1. Diallel experiment for the season I (January – April 2002)

4.2.1.1. Plant height (Table 35)

The heterosis over mid parental value for plant height ranged from -21.50 (CLN 2123A x LE 415) to 64.42 (H 24 x LE 415) per cent. Relative heterosis was positive and significant for thirteen hybrids out of thirty studied. Positive heterobeltiosis ranged from 0.46 (LE 415 x LCR 9) to 58.38 (H 24 x LE 415) per cent. Among these seven were significant. Only 3 hybrids H 24

x CLN 2123A (19.40 per cent), CLN 2123A x LCR 9 (15.27 per cent) and H 24 x LCR 9 (6.54 per cent) exhibited significant positive heterosis over best parent.

4.2.1.2. Number of branches per plant (Table 35)

For the number of branches per plant, thirteen out of thirty hybrids exhibited significant positive heterosis over mid parental value, which varied from 7.48 (LE 415 x CLN 2123A) to 32.87 (H 24 x LCR 9) per cent. The range of heterobeltiosis was between -40.00 (LCR 3 x LCR 9) to 26.67 (H 24 x LCR 9) per cent. Only 3 hybrids H 24 x LCR 9, CLN 2123A x LCR 9 and LCR 9 x H 24 recorded significant positive heterosis over best parent with the values of 26.67, 14.67 and 6.67 per cent respectively.

4.2.1.3. Days to 50 per cent flowering (Table 36)

The di estimates in respect of days to 50 per cent flowering ranged from -16.78 (LCR 1 x LCR 9) to 31.53 (CLN 2123A x H 24) per cent. The highest negative and significant heterobeltiosis was exhibited in LCR 3 x LCR 1 and LCR 3 x LCR 9 (-17.31 per cent each) followed by LCR 1 x LCR 9 (-15.07 per cent).

When the hybrids were compared with the best parent, the heterotic effect varied from -17.31 (LCR 3 x LCR 1 and LCR 3 x LCR 9) to 48.08 (LCR 9 x CLN 2123A) per cent.

4.2.1.4. Number of fruits per plant (Table 36)

The di estimate ranged from -43.50 (CLN 2123A x LCR 3) to 122.22 (H 24 x LE 415) per cent. The positive and significant heterosis over mid parent was recorded in 16 hybrids. The dii ranged from -60.59 (LCR 1 x LCR 3) to 89.26 (H 24 x LE 415) per cent. Out of thirty hybrids, ten registered positive significant dii value. None of the hybrids recorded positive heterosis over the best parent.

4.2.1.5. Fruit weight (Table 37)

For fruit weight, twelve out of 30 hybrids exhibited significant positive heterosis over mid parental value which varied from 4.96 per cent in H 24 x LCR 3 to 46.45 per cent in LE 415 x H 24. The range of heterobeltiosis was between – 69.16 per cent in LCR 1 x LCR 3 to 28.00 per cent in LE 415 x H 24. All hybrids exhibited negative diii, which ranged from -76.08 (LCR 3 x CLN 2123A) to -26.94 (LCR 1 x LCR 9) per cent.

4.2.1.6. Yield per plant (Table 37)

Among the 30 hybrids, 18 exhibited significant and positive heterosis over mid parent which ranged from 10.48 (H 24 x LCR 3) to 176.10 (H 24 x LE 415) per cent. The dii estimate ranged from -59.02 in LCR 1 x LCR 3 to 111.04 per cent in H 24 x LE 415. Only two hybrids H 24 x LE 415 and H 24 x CLN 2123A exhibited significant heterosis in the positive direction over the best with values 22.18 and 17.11 per cent respectively.

4.2.1.7. Total soluble solids (TSS) (Table 38)

Positive and significant heterosis over mid parent was recorded in 10 hybrids with highest di value in the hybrid LCR1 x LCR 9 (31.02 per cent). The heterobeltiosis for TSS ranged from -27.53 per cent in CLN 2123A x LE 415 to 23.74 (LCR 1 x LCR 9). Significant positive heterosis over best parent was registered in only three hybrids namely LCR 1 x LCR 9 (7.93 per cent), CLN 2123A x LCR 1 (4.19 per cent) and LCR 1 x LCR 3 (3.96 per cent).

4.2.1.8. Acidity (Table 38)

The di estimate ranged from -9.43 per cent in LE 415 x CLN 2123A to 17.65 per cent in LCR 1 x H 24. Out of 30 hybrids, thirteen showed positive significant value. Significant positive heterobeltiosis ranged from 2.48 (LCR 3 x CLN 2123A) to 10.00 (LCR 1 x H 24) per cent. Only one hybrid LCR 3 x CLN 2123A showed significant positive best parent heterosis (2.48 per cent).

4.2.1.9. Ascorbic acid (Table 39)

The di value ranged from -24.71 per cent in LCR 1 x LCR 3 to 36.16 per cent in CLN 2123A x LCR 9. Out of 30 hybrids, 16 noticed significant positive di value. The dii value ranged from -31.20 (LCR 1 x LCR 3) to 33.32 (CLN 2123A x LCR 9) per cent, of which 14 exhibited significant positive value. Heterosis over best parent ranged from -31.20 per cent in LCR 1 x LCR 3 to 10.35 per cent in LCR 3 x LCR 9.

4.2.1.10. Lycopene (Table 39)

For lycopene content, 15 out of thirty hybrids showed significant positive heterosis over mid parental value which varied from 7.89 per cent in LCR 9 x LCR 1 to 78.82 per cent in LCR 3 x CLN 2123A. The range of heterobeltiosis was between -50.14 per cent in H 24 x LCR 3 and 49.85 per cent in LCR 3 x CLN 2123A. None of the hybrids exhibited positive heterosis over best parent.

4.2.1.11. Total phenol (Table 40)

The range of di estimate was from -28.57 (LCR 1 x LCR 3) to 9.60 (LCR 3 x H 24) per cent. The highest dii estimate was noticed in H 24 x CLN 2123A (7.35 per cent) and the lowest was observed in LCR 1 x LCR 3 (-30.79 per cent). None of the hybrids showed positive heterosis over best parent.

4.2.1.12. OD phenol (Table 40)

The heterosis for OD phenol ranged from -67.19 in LCR 1 x CLN 2123A to 58.28 per cent in LCR 1 x LCR 9 over their corresponding mid parental values. Six hybrids exhibited significant and positive di estimate. The heterosis over the better parent ranged from -71.81 per cent in LCR 1 x CLN 2123A to 32.53 per cent in LCR 1 x LCR 9. None of the hybrids expressed positive heterosis over best parent.

4.2.1.13. Disease incidence at 75 DAT (Table 41)

4.2.1.13.1. TLCV incidence

Negative di estimate ranged between -96.21 per cent in LCR 9 x CLN 2123A and -0.66 per cent in LCR 1 x H 24. The dii and diii value ranged from 18.46 per cent (LCR 9 x LCR 1) in case of former and 1301.72 per cent (CLN 2123A x LE 415) in case of latter to 5663.79 per cent in LCR 1 x LCR 3 in both the cases.

4.2.1.13.2. Tv incidence

Four out of 30 hybrids showed significant negative relative heterosis for this trait, while significant negative dii and diii values was exhibited by only one hybrid LE 415 x LCR 3 with values -51.26 and -38.95 per cent respectively. Significant positive heterosis over mid, better and best parent was exhibited by 7, 11 and 15 hybrids respectively out of 30 studied.

4.2.1.13.3. Total disease incidence

Relative heterosis value ranged between -27.82 per cent in H 24 x LCR 1 to 87.35 per cent in CLN 2123A x H 24. H 24 x LCR 1 was the only hybrid showing significantly negative heterosis over better parent. None of the hybrids exhibited the same over best parent.

4.2.2. Diallel experiment for the season II (March – June 2002)

4.2.2.1. Disease incidence at 75 DAT (Table 42)

4.2.2.1.1. TLCV incidence

The highest significant negative heterosis over mid parent was exhibited in LCR 9 x LE 415 (-97.35 per cent) followed by LCR 9 x H 24 (-96.90 per cent) and CLN 2123A x LCR 9 (-96.22 per cent). The dii estimate ranged between -96.74 (LCR 9 x H 24) and 105.65 (CLN 2123A x LCR 3) per cent. Only three hybrids showed significantly negative heterosis over best parent.

4.2.2.1.2. Tv incidence

Three out of 30 hybrids exhibited significant and negative heterosis over mid parent with values being -22.94 (H 24 x CLN 2123A), -22.87 (H 24 x LCR 3) and -20.40 (CLN 2123A x LCR 1) per cent respectively. The di and diii estimate ranged from -19.86 and 21.51 per cent (both in H 24 x LCR 9) respectively to 181.48 (LCR 3 x LE 415) per cent. None of the hybrids showed significantly negative heterosis over better and best parent.

4.2.2.1.3. Total disease incidence

The di value ranged between -24.61 per cent in H 24 x CLN 2123A and 66.34 per cent (LCR 3 x LE 415). Among them, five were significantly negative and 16 were significantly positive. With respect to dii and diii values, H 24 x CLN 2123A recorded highest negative heterosis of -22.22 and -17.81 per cent respectively while LCR 3 x LE 415 recorded highest positive heterosis of 75.70 per cent for both the estimates.

4.2.3. Season III

4.2.3.1. Plant height (Table 43)

Out of eight hybrids, only one hybrid LCR 1 x LE 415 showed significantly negative heterosis of -3.47 per cent. Relative heterosis was positive and significant for four hybrids which ranged from 11.60 (LCR 1 x H 24) to 26.33 per cent (H 24 x LCR 9). The dii value ranged from -10.91 (H 24 x LCR 1) to 10.22 (LE 415 x LCR 1) per cent. Highest significant and positive heterosis over best parent and the commercial hybrids namely Ankush and COTH 1 was noticed in H 24 x LCR 9 (9.62, 25.51 and 23.54 per cent respectively).

4.2.3.2. Number of branches per plant (Table 44)

For this character, none of the hybrids differed significantly over mid or better or best parent or commercial hybrid COTH 1. The diii value ranged from -18.70 (LCR 1 x LE 415) to 14.06 (H 24 x LCR 9) whereas the same hybrids recorded similar trend for standard heterosis.

4.2.3.3. Days to 50 per cent flowering (Table 45)

The relative heterosis was negative and significant in two crosses out of eight studied. None of them showed significant positive di value whereas five hybrids showed significant positive dii values, CLN 2123A x LCR 9 registered highest significantly positive diii and SH of 17.44 and 14.80 per cent respectively. All hybrids showed positive heterosis over best parent and commercial hybrids for this character except H 24 x CLN 2123A.

4.2.3.4. Number of fruits per plant (Table 46)

Seven out of eight hybrids showed significant positive heterosis over mid parental value with highest being 115.42 per cent in H 24 x CLN 2123A. Only one hybrid LCR 1 x CLN 2123A registered negative heterosis over better and best parent. The SH value for COTH 1 ranged from -37.00 (LCR 1 x CLN 2123A) to 58.03 (H 24 x CLN 2123A) per cent. Four hybrids out of eight, showed significant positive SH over Ankush.

4.2.3.5. Average fruit weight (Table 47)

Except CLN 2123A x LCR 9 (8.36 per cent) and H 24 x CLN 2123A (5.24 per cent), all hybrids showed negative relative heterosis for this trait. The dii and diii values were negative in all the eight hybrids with highest value of -51.43 (LE 415 x LCR 1) and -62.80 (H 24 x CLN 2123A) per cent respectively. The heterosis over commercial hybrid COTH 1 ranged between -13.34 (H 24 x CLN 2123A) and 50.37 (LCR 1 x CLN 2123A) per cent and over Ankush ranged from -33.89 (H 24 x CLN 2123A) to 14.71 (LCR 1x CLN 2123A) per cent.

4.2.3.6. Yield per plant (Table 48)

The di estimates of all eight hybrids were positive except one (LE 415 x LCR 1). The dii estimate ranged from -25.52 (CLN 2123A x LCR 9) to 122.22 (H 24 x CLN 2123A) per cent. The best parent heterosis was positive and significant in only one hybrid (H 24 x CLN 2123A) with value being 11.88 per cent. Except LCR 1 x CLN 2123A, all other hybrids exhibited positive heterosis over COTH 1 whereas one hybrid exhibited (H 24 x CLN 2123A) significant positive heterosis over Ankush.

4.2.3.7. pH of the fruit juice (Table 49)

Only two hybrids showed significant di estimates, of which one is positive (10.18 per cent) and another is negative (-8.69 per cent). The heterobeltiosis ranged from -5.05 (H 24 x LCR 9) to 13.09 (LCR 1 x LE 415) per cent. None of the hybrids recorded negative best parent heterosis for this trait. The standard heterosis over COTH 1 ranged from -7.75 (H 24 x LCR 9) to 4.91 (LCR 1 x LE 415). Only one hybrid (H 24 x LCR 9) showed significant negative heterosis over Ankush for this character.

4.2.3.8. Acidity (Table 50)

The heterosis over mid parental value ranged from -10.00 per cent in the hybrid LCR 1 x LE 415 to 10.00 per cent in LCR 1 x H 24. Four out of eight hybrids recorded significant and negative heterosis over better parent whereas seven showed significant and negative heterosis over best parent. Regarding standard heterosis over COTH 1, except one hybrid (LCR 1 x LE 415), all others showed positive values while seven out of eight hybrids exhibited significant negative heterosis over Ankush.

4.2.3.9. Total Soluble Solids (TSS) (Table 51)

Out of eight hybrids three registered significantly negative relative heterosis while four showed significantly positive relative heterosis. The heterobeltiosis ranged from -13.61 (LCR 1 x H 24) to 6.18 (LCR 1 x LE 415) per cent. All hybrids showed significant negative heterosis over best parent with highest being -30.36 per cent in LCR 1 x H 24. The SH value over COTH

1 ranged between -13.96 (LCR 1 x H 24) and 15.40 (CLN 2123A x LCR 9) per cent. Four out of eight hybrids exhibited significant positive heterosis over the commercial hybrid Ankush.

4.2.3.10. Ascorbic acid (Table 52)

Three out of eight hybrids showed significant heterosis over mid parent of which one was negative (LCR 1 x CLN 2123A) and other two were positive (CLN 2123A x LCR 9 and LCR 1 x LE 415). The highest positive heterosis of 13.57, 8.22 and 30.43 per cent over better and best parent and commercial hybrid COTH 1 was exhibited in the cross CLN 2123A x LCR 9 while the highest negative heterosis of -33.82, -36.57 and -23.56 per cent respectively was recorded in LCR 1 x CLN 2123A. Only three hybrids out of eight exhibited positive SH over Ankush.

4.2.3.11. Lycopene (Table 53)

The di estimate ranged from -35.30 (LCR 1 x H 24) to 27.27 (LCR 1 x CLN 2123A). The heterosis over better parent ranged between -45.45 (LCR 1 x H 24) and 22.45 (LCR 1 x CLN 2123A) per cent. While seven out of eight hybrids recorded significantly negative diii estimates. The standard heterosis over COTH 1 ranged between -37.31 per cent in LCR 1 x H 24 and 18.10 per cent in H 24 x LCR 1. Only one hybrid (H24 x LCR 1) showed significant positive heterosis over the commercial hybrid Ankush.

4.2.3.12. Total sugar (Table 54)

Only one hybrid (H 24 x LCR 9) exhibited significant positive di, dii and diii estimates of 28.57, 22.86 and 7.80 per cent respectively. While all hybrids exhibited significantly negative heterosis over commercial hybrids.

4.2.3.13. Reducing sugar (Table 55)

Out of eight hybrids, four exhibited significantly positive heterosis over mid parent where three exhibited the same over better parent. The diii estimate ranged from -19.18 (LCR 1 x CLN

2123A) to 16.78 (H 24 x LCR 9) per cent. While none of the hybrids exhibited positive heterosis over commercial hybrids.

4.2.3.14. Pectin (Table 56)

The relative heterosis value for this character ranged from -70.49 (H 24 x CLN 2123A) to 100.00 (H 24 x LCR 9). Except H 24 x LCR 9, all hybrids exhibited significant and negative heterosis over better parent while none of them showed positive heterosis over best parent. The standard heterosis value ranged between -35.71 (H 24 x CLN 2123A) and 135.71 (H 24 x LCR 9) per cent. No hybrid exhibited significant positive heterosis over standard hybrid Ankush.

4.2.3.16. Total phenol (Table 57)

Three hybrids exhibited positive relative heterosis for this trait with highest value being 12.45 per cent in H 24 x LCR 9 whereas only one hybrid (H 24 x LCR 1) exhibited significant and positive heterosis (7.48 percent) over better parent. All hybrids exhibited positive heterosis values over commercial hybrids.

4.2.3.17. OD phenol (Table 58)

The di estimate for this trait ranged between -42.65 (LCR 1 x CLN 2123A) to 32.19 (H 24 x LCR 1) per cent. Only H 24 x LCR 1 exhibited significant positive heterosis over better parent while none of them exhibited the same over best parent. But all hybrids exhibited positive and significant heterosis over commercial hybrid COTH1. LCR 1 x CLN 2123A was the only hybrid exhibited significant negative heterosis over commercial hybrid Ankush.

4.2.3.18. Disease incidence at 75 DAT (Table 59a, 59b and 59c)

4.2.3.18.1. TLCV incidence (Table 59a)

Five hybrids out of eight showed significantly negative heterosis over mid parental value with highest being -39.01 per cent in LE 415 x LCR 1 while four hybrids recorded significantly negative heterosis over better parent. The best parent heterosis was non significant in all the

crosses except one hybrid LE 415 x LCR 1 (-24.58 per cent). The SH estimates over susceptible hybrid COTH 1 ranged between -33.79 (LE 415 x LCR 1) and 4.77 (H 24 x LCR 9) per cent. The cross LE 415 x LCR 1 only had significant negative heterosis over the commercial hybrid Ankush.

4.2.3.18.2. Tv incidence (Table 59b)

Only one of the hybrids showed positive heterosis over mid parental value while two showed positive heterosis (LCR 1 x LE 415) over better parent. The diii estimates was negative and significant in only one cross H 24 x LCR 1 with value being -15.37 per cent. All hybrids exhibited significant negative heterosis over the commercial hybrid Ankush.

4.2.3.18.3. Total disease incidence (Table 59c)

All hybrids showed negative relative heterosis of them five were significant. The dii estimate ranged between -22.17 (H 24 x CLN 2123A) to 0.66 (LCR 1 x LE 415) per cent. The heterosis over commercial hybrids was negative and significant in all the hybrids.

4.3. Combining ability

The general and specific combining ability for 15 characters in the season I and three characters in the season II were furnished in Table 60. GCA, SCA and reciprocal of SCA variances were significant for all the characters studied in both the seasons. The ratio of GCA/SCA in both the seasons ranged from 0.59 (Ascorbic acid) to 15.13 for the character average fruit weight. Out of 15 characters studied in the first season, eleven exhibited higher GCA variance than that of SCA, the remaining characters exhibited higher SCA variance than that of GCA. The RCA variance in the first season ranged between 0.001 and 1086.38. In the second season, out of three characters studied, only one exhibited GCA/SCA ratio of less than one i.e. SCA variance higher than that of GCA.

4.3.1. Combining ability in the Season I

4.3.1.1. Plant height (Table 61)

Among the six parents, CLN 2123A, H 24 and LCR 9 showed significant and positive *gca* of 11.37, 3.70 and 1.63 respectively. The *sca* effect was positive and significant for six crosses with the highest value of 11.48 in LCR 1 x LE 415. Among the reciprocal crosses, *sca* effect ranged from -10.19 (LE 415 x H 24) to 14.39 (H 24 x CLN 2123A).

4.3.1.2. Number of branches per plant (Table 61)

The *gca* effect of parents ranged from -0.66 (LCR 3) to 0.49 (H 24). Five out of 15 direct crosses showed significantly positive *sca* effect ranged from 0.36 (LCR 1 x LCR 3) to 1.27 (LCR 9 x H 24) while in reciprocal crosses, eight out of 15 recorded the same with highest value being 0.75 in the cross H 24 x LCR 9.

4.3.1.3. Days to 50 per cent flowering (Table 62)

Among the six parents, the *gca* ranged from -3.13 in LCR 3 to 2.71 in LCR 9. The *sca* was positive and significant for LCR 3 x CLN 2123A (4.54), H 24 x LE 415 (2.38), CLN 2123A x LE 415 (1.08) and LCR 9 x H 24 (1.00) in case of direct crosses, whereas in reciprocal crosses, CLN 2123A x LCR 1 (3.75), H 24 x LE 415 (2.50), LCR 3 x LCR 1 (2.00) and H 24 x LCR 3 (1.25) recorded the same. Negative and significant *sca* effect in direct and reciprocal crosses was registered in 3 and 5 hybrids respectively.

4.3.1.4. Number of fruits per plant (Table 62)

The *gca* effect for number of fruits per plant ranged from -9.48 (LCR 1) to 8.32 (LCR 3). Seven out of 15 direct crosses registered positive *sca* and six registered significant negative *sca* effect. The *sca* effect of the reciprocal crosses ranged from -16.53 (LE 415 x H 24) to 15.15 (H 24 x CLN 2123A). Among 15 reciprocal crosses, eight were significantly positive and two significantly negative.

4.3.1.5. Fruit weight (Table 63)

The parents LCR 1 and LCR 9 showed positive and significant *gca* of 18.17 and 9.32 for this trait whereas others recorded negative but significant *gca* effect. Among the direct crosses, higher positive *sca* effect of 9.34 was registered in LCR 9 x CLN 2123A followed by H 24 x LE 415 (6.06). Among the reciprocal crosses, positive and significant *sca* was ranging from 0.60 (LE 415 x LCR 3) to 11.13 (LCR 1 x LCR 9). The negative and significant effect was shown by six hybrids ranging from -7.25 in the cross CLN 2123A x LCR 1 to -0.75 in the cross LE 415 x LCR 1.

4.3.1.6. Yield per plant (Table 63)

The *gca* of the parents LCR 3, LE 415 and LCR 9 was negative and significant with values -0.20, -0.12 and -0.08 respectively while in H 24 and LCR 1, it was positive (0.21 and 0.15 respectively) and significant. The *sca* effect ranged from -0.47 (LCR 3 x CLN 2123A) to 0.59 (LCR 1 x LE 415) in case of direct crosses while in reciprocal crosses, the same ranged between -0.57 (LE 415 x H 24) and -0.10 (LE 415 x LCR 9).

4.3.1.7. Total soluble solids (TSS) (Table 64)

The *gca* effect for TSS ranged from -0.11 (LE 415 and LCR 9) to 0.13 (CLN 2123A). Among 15 direct crosses, only five showed positive and significant *sca* with highest of 0.34 in LCR 1 x CLN 2123A. Six recorded negative and significant *sca*, while *sca* of the reciprocal crosses ranged from -0.36 (LCR 3 x LCR 1) to 0.55 (LE 415 x CLN 2123A).

4.3.1.8. Acidity (Table 64)

The *gca* was positive and significant in LCR 3 and CLN 2123A with values being 0.05 and 0.03 respectively. The *sca* of direct crosses varied between -0.03 in the crosses LCR 3 x LE 415 and LCR 1 x LCR 3 to 0.03 in the crosses LCR 1 x H 24 and LCR 3 x CLN 2123A. The *sca*

of the reciprocal crosses was positive and significant in two crosses with highest being 0.03 (LCR 3 x LCR 9 and LE 415 x H 24) and negative and significant with 7 crosses.

4.3.1.9. Ascorbic acid (Table 65)

The parent LCR 3 showed significant positive *gca* effect of 0.80. Negative and significant *sca* effects of -0.95 and -0.82 were registered by H 24 and LE 415 respectively. Among the direct crosses, the highest positive *sca* effect of 4.02 was registered by LCR 1 x H 24, followed by LCR 9 x CLN 2123A (2.79) and LCR 9 x LCR 3 (1.82). The negative and significant *sca* effect was shown by 5 hybrids ranging from -1.82 to -1.50. The *sca* of the reciprocal crosses was positive and significant in seven crosses with highest being 6.42 in the cross LCR 3 x LCR 1 and was negative and significant in three crosses with highest value being -4.09 in the cross H 24 x LCR 9.

4.3.1.10. Lycopene (Table 65)

The *gca* of parents ranged from -0.27 (LCR 1) to 0.47 (H 24). Among 15 direct crosses, seven were positively and seven negatively significant which ranged between -0.58 (LCR 9 x H 24) and 1.34 (LCR 3 x CLN 2123A). Among the reciprocal crosses, the *sca* effect ranged from -0.64 (CLN 2123A x LCR 1) to 0.97 (H 24 x LCR 1). Eight out of 15 reciprocal crosses showed positive and negative *sca* effect.

4.3.1.11. Total phenol (Table 66)

The parent H 24 showed significant positive *gca* (22.17) whereas three parents registered significantly negative *gca*. The *sca* effect of the direct crosses ranged between -37.33 (LCR 1 x LCR 3) and 28.77 (LCR 3 x H 24) whereas the same of reciprocal crosses between -23.92 (LE 415 x H 24) and 33.08 (H 24 x CLN 2123A). Positive and significant *sca* effect was registered in four of direct crosses and five of reciprocal crosses.

4.3.1.12. OD phenol (Table 66)

The *gca* for this trait ranged between -16.50 (LCR 3) to 24.82 (H 24). The *sca* effect of direct crosses ranged from -52.15 (LCR 1 x CLN 2123A) to 46.34 (LCR 1 x H 24) where the same in reciprocal crosses ranged between -22.65 (LE 415 x H 24) and 45.18 (LE 415 x LCR 3). Out of all 30 crosses, twelve exhibited positive and significant *sca* effect and 10 registered negatively significant *sca* effect.

4.3.1.13. Disease incidence at 75 DAT (Table 67 and 68)

4.3.1.13.1. TLCV incidence (Table 67)

The parents CLN 2123A, H 24 and LE 415 recorded negative *gca* of -7.62, -3.56 and -0.31 while the former two were significant. The *sca* effect of direct crosses ranged from -9.22 (LCR 9 x CLN 2123A) to 9.22 (LCR 3 x H 24) while that of reciprocal crosses ranged from -6.81 (H 24 x CLN 2123A) to 8.43 (LE 415 x H 24).

4.3.1.13.2. Tv incidence (Table 67)

The *gca* effect of parents ranged from -3.15 in LCR 1 to 6.56 in CLN 2123A. The *sca* effect of direct crosses was significant and positive in two hybrids CLN 2123A x H 24 (10.02) and LCR 9 x LCR 3 (4.68) while only one hybrid showed significantly negative *sca* of -8.03 (LCR 9 x H 24). The *sca* of the reciprocal crosses ranged from -14.87 (H 24 x CLN 2123A) to 5.45 (CLN 2123A x LCR 1).

4.3.1.13.3. Total disease incidence (Table 68)

All parents recorded non significant *gca* effect ranging from -2.36 (H 24) to 1.79 (LCR 9). Regarding the *sca* effect of direct crosses, three recorded significantly positive *sca* with highest being 8.65 in CLN 2123A x H 24 and three with significantly negative *sca* ranging from -8.22 (LCR 9 x H 24) to -4.87 (LCR 1 x H 24). Reciprocal effect ranged from -12.74 (CLN 2123A x H 24) to 6.79 (LCR 1 x CLN 2123A).

4.3.2. Combining ability in the season II (March – June 2002)

4.3.2.1. Disease incidence at 75 DAT (Table 69 and 70)

4.3.2.1.1. TLCV incidence (Table 69)

Only two parents out of six showed significant *gca* effect of -6.05 and 5.54 in CLN 2123A and LCR 3 respectively. The *sca* effect of the direct crosses ranged between -8.11 (LCR 1 x H 24) and 10.48 (LCR 9 x LCR 1). In the reciprocal crosses, three possessed significantly positive *sca* effect with highest being 11.98 (LE 415 x LCR 9) while only one hybrid CLN 2123A x LCR 9 registered significant and negative *sca* effect of -7.46.

4.3.2.1.2. Tv incidence (Table 69)

The *gca* effect of parents ranged from -2.20 (H 24) to 1.79 (CLN 2123 A). The *sca* effect of direct crosses ranged between -5.12 (LCR 1 x CLN 2123A) and 6.96 (LCR 3 x LE 415), while the *sca* of the reciprocal crosses was positive and significant in one hybrid LCR 3 x LCR 1 (6.51) and negative and significant in four hybrids.

4.3.2.1.3 Total disease incidence (Table 70)

The *gca* effect of parents ranged between -1.84 (H 24) and 1.27 (CLN 2123A). In case of direct crosses, three recorded significantly positive *sca* effect with highest being 6.87 in LCR 3 x LE 415 while three recorded significantly negative *sca* effect. The reciprocal effect ranged from -8.79 (LE 415 x LCR 3) to 6.51 (LCR 3 x LCR 1).

4.4. Hayman's diallel analysis

Similar to combining ability analysis, Hayman's approach was carried out only to 15 characters in the I season *viz.* plant height, number of branches per plant, days to 50 per cent flowering, number of fruits per plant, average fruit weight, yield per plant, TSS, acidity, ascorbic acid, lycopene, total phenol, OD phenol, TLCV, Tv and Total disease incidence at 75 DAT. In the II season, it was restricted to TLCV, Tv and Total disease incidence at 75 DAT.

The validity of the assumptions of the diallel was tested by t^2 test (Table 71). Non significant t^2 values were observed for all characters except two namely number of branches per plant and Tv incidence at 75 DAT in the first season indicating the failure of hypothesis for these

two traits. In the second season, TLCV and total disease incidence at 75 DAT exhibited significant t^2 values.

Another test of significance have revealed that the W_r , V_r regression significantly deviated from zero for five out of 15 characters studied namely number of fruits per plant, Average fruit weight, acidity, TLCV and T_v incidence at 75 DAT in the season I whereas in the season II, T_v incidence at 75 DAT showed significant value.

The deviation of W_r , V_r regression from unity was significant for four out of 15 characters studied in the first season *viz.* number of branches per plant, TSS, T_v and Total disease incidence at 75 DAT whereas in the second season, TLCV and total disease incidence at 75 DAT exhibited significant deviation from the unity indicating the failure of hypothesis for these characters.

4.4.1. Graphical analysis

Information on gene action of six parents' arrays for the 18 characters under study (season I and II) was elicited from (W_r , V_r) graph. The regression coefficients for V_r , W_r are furnished in Table 71. The estimates of variances and covariances for F_{1s} are presented in Table 72.

4.4.1.1. Season I

4.4.1.1.1. Plant height (Fig. 1)

The interception of the regression line of W_r , V_r on Y-ordinate was above the point of origin. The slope of the regression line was more than the unity ($b=1.179 \pm 0.312$). With respect to the position of parental points along the regression line, LCR 1 located nearer to the origin while H 24 located farther from the origin. LCR 3, LE 415, CLN 2123A located around middle. The parents LCR 3 and H 24 located above the regression line whereas others located just below the regression line.

4.4.1.1.2. Number of branches per plant (Fig. 2)

The regression line of W_r , V_r of parental arrays intercepted the Y axis just below the point of origin. The slope of the regression line was less than the unity ($b=0.256 \pm 0.153$). The array points LE 415, LCR 1, CLN 2123A, H 24 and LCR 3 located closer to the origin whereas LCR 9 located far away from the origin. The array points LE 415, CLN 2123A and H 24 were above the regression line, LCR 9 on the line and LCR 3 and LCR 1 above the regression line.

4.4.1.1.3. Days to 50 per cent flowering (Fig. 3)

In the graph, the regression line intercepted the Y axis far above the origin. The slope of the regression line was less than the unity ($b=0.0204 \pm 0.485$). The array points LCR 9, CLN 2123A and LCR 1 were at the middle while others were located far away from the origin. The array points LCR 3, CLN 2123A and LCR 9 were below the regression line while others above the line.

4.4.1.1.4. Number of fruits per plant (Fig. 4)

The regression line of W_r , V_r of the parental arrays intercepted the Y axis below the origin. The slope of the regression line was more than the unity ($b=1.295 \pm 0.275$). The array points LCR 1, LCR 9, LE 415, CLN 2123A and H 24 were at the middle while LCR 3 located farther from the origin. The array points H 24 and CLN 2123A located below the regression line while other parents just above the regression line.

4.4.1.1.5. Average fruit weight (Fig. 5)

The regression line in the W_r , V_r graph intercepted the Y-ordinate well above the origin. The slope of the regression line was less than the unity ($b=0.967 \pm 0.115$). The parental arrays LCR 3, LCR 9 and H 24 located nearer to the origin whereas CLN 2123A and LE 415 located at middle and LCR 1 far away from the origin. The parental arrays LCR 9 and LCR 3 located below the regression line whereas CLN 2123A and LCR 1 on the line.

4.4.1.1.6. Yield per plant (Fig. 6)

The regression line of W_r , V_r graph intercepted the Y axis below the point of origin. The slope of the regression line was more than the unity ($b=0.625 \pm 0.347$). The array point LCR 9 located outside the range of limiting parabola. The array points LCR 3, LCR 1, H 24 and CLN 2123A located around the middle of the regression line whereas LE 415 far away from the origin. Except LCR 3 and LE 415, all other parents located below the regression line.

4.4.1.1.7. TSS (Fig. 7)

The regression line intercepted the Y-ordinate far away from the origin. The slope of the regression line was less than the unity and zero ($b=-0.828 \pm 0.590$). The array points LCR 3, H 24, CLN 2123A, LCR 9 and LE 415 located just at the middle while LCR 1 located far away from the origin. The array points LCR 9, LE 415 located above the middle while LCR 3 on the regression. Other array points had located just below the regression line.

4.4.1.1.8. Acidity (Fig. 8)

The regression line intercepted the Y axis above the point of origin. The slope of the regression line was more than the unity ($b=1.151 \pm 0.087$). The parental array point LE 415 located close to the origin whereas H 24 and LCR 1 at the middle and others farther from the origin. While LE 415 and LCR 9 located just above the regression line, CLN 2123A arranged at middle and others just below the regression line.

4.4.1.1.9. Ascorbic acid (Fig. 9)

The regression line of W_r , V_r graph intercepted the Y axis just above the origin. The slope of the line was just below zero ($b=-0.0007 \pm 0.087$). The parental array points distributed far from the regression line except H 24. LE 415, LCR 3 and LCR 9 located above to the line whereas other parents located below the regression line.

4.4.1.1.10. Lycopene (Fig. 10)

The regression line in the W_r , V_r graph intercepted the Y-ordinate at point just below the origin. The slope of the regression line was less than the unity ($b=0.903 \pm 0.370$). The distribution of array points LE 415 and LCR 9 were closer to the origin whereas LCR 1 and CLN 2123A at the middle and H 24 and LCR 3 far away from the origin. The parental arrays LCR 1 and H 24 located above the regression line, LE 415 on the regression line whereas other arrays below the regression line.

4.4.1.1.11. Total phenol (Fig. 11)

The regression line intercepted the Y-axis below the origin. The slope of the regression line was less than the unity ($b=0.336 \pm 0.278$). The parental array points H 24 and LCR 9 located nearer to the origin, CLN 2123A centred at the middle while LE 415 and LCR 1 located far away from the origin. The array points LCR 9, LE 415 and LCR 1 located above the regression line, CLN 2123A on the regression line while other arrays well below the regression line.

4.4.1.1.12. OD phenol (Fig. 12)

The regression line intercepted the Y-ordinate below the origin. The slope of the regression line was less than the unity ($b=0.753 \pm 0.409$). The array points LCR 9, LCR 3, H 24 and CLN 2123A located at middle position whereas LCR 1 and LE 415 located far away from the origin. While the parental array points H 24 and LCR 1 located just below the regression line and CLN 2123A on the line, others distributed above the regression line.

4.4.1.1.13. TLCV incidence at 75 DAT (Fig. 13)

The regression line intercepted the Y-axis far below the origin. The slope of the regression line was more than the unity ($b=1.241 \pm 0.346$). Except H 24, all the array points located above the regression line. The array point CLN 2123A located near the origin while H 24 around the middle while others located farther from the origin.

4.4.1.1.14. Tv incidence at 75 DAT (Fig. 14)

The regression line intercepted the Y-ordinate just below the origin. The slope of the regression line was less than the unity ($b=0.277 \pm 0.085$). The array point LCR 3 and LCR 1 located closer to the origin, while LCR 9, LE 415 and CLN 2123A at the middle and H 24 far away from the origin. LE 415 located above the regression line while LCR 3, CLN 2123A and H 24 on the line and others below the regression line.

4.4.1.1.15. Total disease incidence at 75 DAT (Fig. 15)

The regression line intercepted the Y-ordinate far above the origin. The slope of the regression line was less than the unity ($b=-1.146 \pm 0.446$). All the array points located far away from the origin except LCR 3 which was lying around middle. While LCR 1 and LCR 9 located below the regression line, all other array points above the regression line.

4.4.1.2. Season II

4.4.1.2.1. TLCV incidence at 75 DAT (Fig. 16)

The interception of Y-ordinate by the regression line was at a point well above the origin. The slope of the regression line was less than the unity ($b=0.055 \pm 0.093$). The parental array points LCR 3, H 24, CLN 2123A and LE 415 located at the middle from the origin whereas LCR 9 and LCR 1 far away from the origin. LE 415 located above the regression line, LCR 3 and LCR 1 on the line while other just below the regression line.

4.4.1.2.2. Tv incidence at 75 DAT (Fig. 17)

The regression line intercepted the Y-axis well below the point of the origin. The array points LCR 1, H 24 located near to the origin while LE 415 located far away from the origin. The array points LCR 1, LCR 9 and LE 415 located above the regression line, while H 24 on the line, others located reasonably below the regression line.

4.4.1.2.3. Total disease incidence at 75 DAT (Fig. 18)

The interception of Y-ordinate by the regression line was at a point well below the origin. The points H 24, CLN 2123A and LCR 3 located near the middle while all others located far away from the origin. The array points LCR 9, LE 415 located above regression line, H 24 on the line while others well below the regression line.

4.4.2. Genetic analysis

The gene action responsible for the inheritance of the characters was studied through diallel analysis. The estimates for genetic parameters *viz.* D, F, H₁, H₂ and h² are furnished in Table 74. The ratios computed from actual values of components of variation are furnished in Table 75. None of the characters studied had r² value nearer to unity. The results of are given character-wise hereunder.

4.4.2.1. Season I (Table 75)

4.4.2.1.1. Plant height

The component of variance due to dominance H_1 (431.560), additive D (199.840), the proportion of dominance due to positive and negative genes in the parents H_2 (328.520), were found to be significant. The mean of covariance of additive and dominance effects over the arrays F (85.941), net dominance h^2 (33.099) and environmental variance E (1.7230) were non significant. The H_1 value was greater than H_2 .

Ratios of the genetic parameters revealed that average degree of dominance $(H_1/D)^{1/2}$ was more than one. Proportion of genes with positive and negative effects in the parents $(H_2/4H_1)$ was 0.190. The ratio of dominant and recessive genes in the parents (K_D/K_R) was more than one (1.343). The number of gene groups controlling this trait and exhibiting dominance (h^2/H_2) was 0.101. The correlation coefficient between Y_r and (W_r+V_r) was -0.214. Heritability in narrow sense was recorded as 56.40 per cent.

4.4.2.1.2. Number of branches per plant

All the six components of variation *viz.* D , H_1 , H_2 , E , F and h^2 recorded non significant values for this character.

The average degree of dominance $(H_1/D)^{1/2}$ was 2.8785. The proportion of genes with positive and negative effects in the parents $(H_2/4H_1)$ was 0.2128. The ratio of dominant and recessive genes in the parents (K_D/K_R) was less than unity. The number of gene groups controlling this character and exhibiting dominance (h^2/H_2) was 0.0252. The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was 0.8741. Heritability in narrow sense was recorded as 38.30 per cent.

4.4.2.1.3. Days to 50 per cent flowering

Two out of six components of variation (D and H_1) were significant. Average degree of dominance $(H_1/D)^{1/2}$ was greater than the unity. The proportion of genes with positive and negative effects $(H_2/4H_1)$ in the parents was not equal to 0.25. K_D/K_R ratio was more than the

unity and the number of gene groups controlling this character was found to be less than one as evidenced by h^2/H_2 ratio (0.0121). The correlation coefficient between parental measurement Y_r and (W_r+V_r) was negative (-0.2587). Heritability in narrow sense was 60.02 per cent.

4.4.2.1.4. Number of fruits per plant

Four out of six components of variation *viz.* D, F, H_1 and H_2 were significant. Average degree of dominance $(H_1/D)^{1/2}$ was more than the unity. Positive and negative alleles are not symmetrically distributed as evident from the ratio of $H_2/4H_1$ (0.1557). The ratio of dominant and recessive genes in the parents (K_D/K_R) was more than the unity (2.6422). The number of gene groups controlling this trait exhibiting dominance (h^2/H_2) was 0.1741. The correlation coefficient between the parental Y_r and (W_r+V_r) was more (0.8432). Narrow sense heritability was recorded high (57.46 per cent).

4.4.2.1.5. Average fruit weight

Four out of six components of variation namely D_1 , F, H_1 and H_2 were significant. The ratios of genetic parameters revealed that $(H_1/D)^{1/2}$ was less than the unity (0.5924). The ratio of dominant and recessive genes in the parents (K_D/K_R) was more than the unity (2.8931). The ratio of h^2/H_2 was 0.1788. The correlation coefficient between Y_r and (W_r+V_r) was 0.7639. Heritability in narrow sense was recorded as 83.43 per cent.

4.4.2.1.6. Yield per plant

The genetic components D, F_1 , H_1 and H_2 were significant while variance due to environment and net dominance effects were non significant. Among the genetic parameters, $(H_1/D)^{1/2}$ and (K_D/K_R) recorded the value of more than the unity. The proportion of genes with positive and negative effect ($H_2/4H_1$) in the parents was 0.1547. The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was negative (-0.5213). The narrow sense heritability was low (17.85 per cent).

4.4.2.1.7. TSS

The components D, H_1 , H_2 and h^2 were significant while F and E showed non significance values. Among the genetic parameters $(H_1/D)^{1/2}$ was more than the unity for this trait (1.7021). The ratio of $H_2/4H_1$ (0.1685) was not equal to 0.25. The ratio of dominant and recessive genes in the parents was more than the unity (3.0229). One group of genes controlled this character, as the ratio h^2/H_2 was less than the unity (-0.0020). The correlation coefficient between Y_r and (W_r+V_r) was -0.4687 indicating equal proportions of dominant genes as positive and negative. The narrow sense heritability was found to be very low (17.29 per cent).

4.4.2.1.8. Acidity

None out of six components of variation was significant. The average degree of dominance $(H_1/D)^{1/2}$ was less than the unity (0.5814). The ratio of dominant and recessive genes (K_D/K_R) in the parents was 2.4383. The $H_2/4H_1$ value (0.1927) was not equal to 0.250. One group of genes controlled this character as the ratio h^2/H_2 was less than the unity (0.1316). The correlation coefficient between Y_r and (W_r+V_r) was 0.0774 indicating equal proportion of dominant genes as positive and negative. The narrow sense heritability was high (79.63 per cent).

4.4.2.1.9. Ascorbic acid

Only dominance variance (H_1) was found to be significant while other five components of variation were non significant. The average degree of dominant $(H_1/D)^{1/2}$ and K_D/K_R was more than one (1.9634 and 2.8117 respectively). The proportion of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. The value of h^2/H_2 was 0.4623 indicating probability of one group of genes controlling the particular trait. The correlation coefficient between the parental measurement Y_r (W_r+V_r) was -0.1353. The heritability in the narrow sense was very low (12.03 per cent).

4.4.2.1.10. Lycopene

The parameters D, F, H_1 and H_2 recorded significant values of 1.432, 1.594, 1.826 and 1.329 respectively. The other components of variation exhibited non-significance. The ratios of genetic parameters revealed that the average degree of dominance $(H_1/D)^{1/2}$ was more than the unity (1.1293) and one group of genes controlled this character, as the ratio of h^2/H_2 was less than the unity (0.4904). The ratio of dominant and recessive genes in the parents (K_D/K_R) was 2.9432. The proportion of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. The correlation coefficient between Y_r and (W_r+V_r) was small (0.4525) indicating equal proportions of dominant genes as positive and negative. The heritability in narrow sense was low (33.08 per cent).

4.4.2.1.11. Total phenol

The proportion of dominance (H_2) due to positive and negative genes in the parents and the variance due to dominance (H_1) were alone found to be significant. Regarding genetic ratios, average degree of dominance $(H_1/D)^{1/2}$ was 2.2607 and only one group of genes controlled this trait and the ratio h^2/H_2 was less than the unity (0.1349). The proportion of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. The K_D/K_R ratio was more than the unity (1.5817). The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was 0.3761. The heritability in narrow sense was recorded low (34.49 per cent).

4.4.2.1.12. OD phenol

Four out of six components of variation namely D, F, H_1 and H_2 were significant. The ratios of $(H_1/D)^{1/2}$ and K_D/K_R were more than the unity (1.3635 and 2.8919 respectively). The number of gene groups controlling the character was found to be one as evidenced by h^2/H_2 ratio (0.5988). The proportions of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. The correlation coefficient between Y_r and (W_r+V_r) was 0.7250. Heritability in narrow sense was found to be low (30.65 per cent).

4.4.2.1.13. TLCV incidence at 75 DAT

All the six components of variation were found to be significant. The average degree of dominance $(H_1/D)^{1/2}$ and K_D was more than the unity (1.3741). The proportion of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. The K_D/K_R ratio was more than the unity (2.2370). The h^2/H_2 ratio was less than one (0.650) indicating single group of genes controlled this trait. The correlation coefficient between Y_r and (W_r+V_r) was 0.4356 indicating that equal proportion of dominant genes as positive and negative. The heritability in the narrow sense was 41.50 per cent.

4.4.2.1.14. Tv incidence at 75 DAT

All the six components of variation namely D, F, H_1 , H_2 , h^2 and E were found to be non significant. The average degree of dominance $(H_1/D)^{1/2}$ was more than the unity (3.7045). The ratio of dominant to recessive genes in the parents (K_D/K_R) was less than the unity (0.5404). The ratio of $H_2/4H_1$ was not equal to 0.25. One group of genes control this trait as it is evidenced by h^2/H_2 ratio (0.0877). The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was 0.5203 indicating equal proportion of dominant genes as positive and negative. Narrow sense heritability was 45.40 per cent.

4.4.2.1.15. Total disease incidence at 75 DAT

Four out of 6 components of variation *viz.* H_1 , H_2 , h^2 and E were significant. The genetic ratios revealed that the average degree of dominance $(H_1/D)^{1/2}$ was very high (10.929) and K_D/K_R value was more than the unity (1.115). Proportion of genes with positive and negative effects was almost equal to 0.25. One block of genes controlled this character as it was evidenced from h^2/H_2 ratio (0.8046). The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was -0.486. Heritability in the narrow sense was very low (11.21 per cent).

4.4.2.2. Season II

4.4.2.2.1. TLCV incidence at 75 DAT

Except environmental component of variation (E), all others exhibited non significant values. The genetic ratios revealed that the average degree of dominance $(H_1/D)^{1/2}$ was more than one (4.1179). The proportion of dominant to recessive genes in the parent was less than the unity (0.1424). The ratio of $H_2/4H_1$ was not equal to 0.25. One group of genes control this trait and exhibit dominance as the h^2/H_2 value was -0.1754. The correlation between the parental measurement Y_r and (W_r+V_r) was 0.3564 indicating equal proportions of dominant genes as positive and negative. The narrow sense heritability was low (38.79 per cent).

4.4.2.2.2. Tv incidence at 75 DAT

Except D and F components, all others genetic parameters namely H_1 , H_2 , h^2 and E had significant values. The average degree of dominance $(H_1/D)^{1/2}$ was more than the unity (2.066) whereas the ratio of dominant and recessive genes (K_D/K_R) in the parents was more than the unity (2.701). The proportion of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. One group of genes controlled this character as the ratio h^2/H_2 was 0.734. The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was -0.876. Heritability in narrow sense was very low (10.70 per cent).

4.4.2.2.3. Total disease incidence at 75 DAT

The components H_1 and H_2 were significant while all other components D, F, h^2 and E were non-significant. The average degree of dominance $(H_1/D)^{1/2}$ was more than the unity (6.7080) and the ratio of dominant and recessive genes in the parents (K_D/K_R) was more than the unity (1.1358). The proportion of genes with positive and negative effects ($H_2/4H_1$) in the parents was not equal to 0.25. One group of genes controlled this character as the ratio h^2/H_2 was 0.4579. The correlation coefficient between the parental measurement Y_r and (W_r+V_r) was -0.8760. Narrow sense heritability was low (31.55 per cent).

4.5. Correlation analysis

Genotypic correlation coefficients were worked out to bring an association between fruit yield per plant and its components in first season involving 30 hybrids and six parents and third season involving 10 hybrids and 5 parents separately and are presented in the Tables 76 and 77.

4.5.1. Season I (Table 76)

4.5.1.1. Plant height

The plant height had positive and significant association with yield whereas it exhibited negative and significant correlation with TLCV at 75 DAT. Its correlation with all other characters was non significant.

4.5.1.2. Number of branches per plant

This trait recorded non significant association with all the characters. With yield it recorded positive association whereas with acidity, it was negatively correlated.

4.5.1.3. Days to 50 per cent flowering

This trait recorded non significant negative association with yield whereas with number of fruits and TSS it expressed significant negative correlation. It exhibited significant positive correlation with average fruit weight and total disease incidence at 75 DAT.

4.5.1.4. Number of fruits per plant

It recorded significant positive association yield and days to 50 per cent flowering while it was significantly and negatively associated with average fruit weight. With all other traits it exhibited non significant correlation.

4.5.1.5. Fruit weight

It recorded positive and significant correlation with yield whereas it had negative and significant correlation with acidity. With all other characters it showed non significant association.

4.5.1.6. TSS

It showed non significant association with any of the characters under study. With yield it recorded non significant negative association.

4.5.1.7. Acidity

It was negatively correlated with yield although the association was non significant. But it was significantly and negatively correlated with average fruit weight and days to 50 per cent flowering. While with all other characters, it yielded non significant association.

4.5.1.8. Ascorbic acid

Although it exhibited positive association with yield, its association was non significant. With all other characters it showed non significant association.

4.5.1.9. Lycopene

Similar to ascorbic acid, it yielded positive non significant association with yield. Lycopene is negatively and significantly correlated with all the three disease incidence traits. It had non significant association with remaining characters under study.

4.5.1.10. Total phenol

Total phenol content was positively and significantly correlated with OD phenol and with TLCV and total disease incidence, it showed negative and significant association. It showed non significant association with remaining characters including yield.

4.5.1.11. OD phenol

It had significantly positive correlation with total phenol, TLCV and total disease incidence. With yield, it recorded non significant positive association. It correlated non-significantly with remaining characters.

4.5.1.12. TLCV incidence at 75 DAT

TLCV incidence was negatively correlated with yield although the correlation was non significant. It had significant negative correlation with Tv incidence at 75 DAT, plant height Total and OD phenol whereas with total disease incidence at 75 DAT, it showed positive association.

4.5.1.13. Tv incidence at 75 DAT

It had negative and significant association with TLCV incidence at 75 DAT and total phenol whereas with total disease incidence at 75 DAT, it showed positive significant association. Tv incidence was negatively associated with yield although non significant.

4.5.1.14. Total disease incidence at 75 DAT

It had positive and significant association with TLCV and Tv incidence at 75 DAT, days to 50 per cent flowering whereas it exhibited significant negative correlation with total and OD phenol. It expressed negative association with yield although the association was non significant.

4.5.2. Season III (Table 77)

4.5.2.1. Plant height

Plant height had positive and significant correlation with number of branches per plant, number of fruits per plant and yield. It had significant negative association with pH and lycopene and TLCV incidence at 75 DAT. It showed non significant association with rest of the characters.

4.5.2.2. Number of branches per plant

It showed positive and significant association with plant height, number of fruits per plant. With yield, it exhibited non significant positive association while it had non significant correlation with all other characters studied.

4.5.2.3. Days to 50 per cent flowering

It had positive and significant correlation with TLCV at 75 DAT, average fruit weight and ascorbic acid while it was negatively and significantly correlated with number of fruits per plant, acidity and reducing sugar. It showed non significant negative correlation with yield.

4.5.2.4. Number of fruits per plant

The number of fruits per plant exhibited significant positive correlation with yield, acidity, total and reducing sugars and number of branches per plant and plant height. While it yielded negative relation with TLCV at 75 DAT, days to 50 per cent flowering and fruit weight. It showed non significant association with remaining characters under study.

4.5.2.5. Average fruit weight

It exhibited positive and significant association with yield and days to 50 flowering. While with number of fruits per plant, acidity and OD phenol, it exhibited significant negative association. It expressed non significant association with rest of the characters under study.

4.5.2.6. pH

It expressed non significant positive association with yield. It registered significant negative correlation with plant height, acidity, pectin and OD phenol. It showed non significant correlation with other characters studied.

4.5.2.7. Acidity

It expressed significant negative correlation with days to 50 per cent flowering, fruit weight, pH and sugar to acid ratio. With OD phenol, it registered positive significant association. It had non significant association remaining characters including the yield.

4.5.2.8. TSS

It is positively and significantly correlated with pectin content whereas negatively but non significantly correlated with yield. It expressed non significant association with remaining characters under study.

4.5.2.9. Ascorbic acid

It experienced significant and positive correlation with days to 50 per cent flowering, total and OD phenol whereas it recorded significant and negative association with lycopene, total and reducing sugar, sugar to acid ratio and pectin. With yield it showed negative and non significant association.

4.5.2.10. Lycopene

Lycopene expressed significant negative correlation with ascorbic acid and plant height. It showed non significant association with remaining characters including yield.

4.5.2.11. Total sugar

It showed positive correlation with reducing sugar, sugar to acid ratio, number of fruits per plant, Tv and total disease incidence at 75 DAT. It expressed significant negative correlation with lycopene. For all other characters, it yielded non significant association including yield.

4.5.2.12. Reducing sugar

It had positive and significant association with total sugar, sugar to acid ratio, number of fruits per plant, Tv and total disease incidence at 75 DAT. It yielded significant negative correlation with plant height and ascorbic acid. It exhibited positive non significant association with yield per plant.

4.5.2.13. Sugar to acid ratio

It had positive and significant association with total and reducing sugar whereas it yielded significantly negative relationship with OD phenol, acidity and ascorbic acid. All other characters expressed non significant association including yield per plant.

4.5.2.14. Pectin

Pectin registered significant and positive correlation with TSS while with ascorbic acid and pH it recorded significantly negative association. It had non significant association with all other characters including yield per plant.

4.5.2.15. Total phenol

It showed positive and significant association with OD phenol and ascorbic acid whereas it was negatively and significantly associated with Tv incidence at 75 DAT. It had non significant association with remaining characters including yield per plant.

4.5.2.16. OD phenol

This trait registered significant positive association with total phenol, ascorbic acid and acidity. Whereas it expressed significant negative association with fruit weight, pH and sugar to acid ratio. It recorded non significant negative association with yield.

4.5.2.17. TLCV incidence at 75 DAT

TLCV incidence was positively and significantly correlated with total disease incidence at 75 DAT, days to 50 per cent flowering, whereas it showed negative and significant association with plant height, number of fruits per plant and yield per plant. With all other characters, it expressed non significant correlation.

4.5.2.18. Tv incidence at 75 DAT

Tv incidence showed significant and positive correlation with total disease incidence at 75 DAT, total sugar and reducing sugar whereas it showed negative and significant correlation with total phenol. It showed non significant negative correlation with yield per plant and OD phenol.

4.5.2.19. Total disease incidence at 75 DAT

This trait recorded positive association with TLCV and Tv incidence at 75 DAT, total and reducing sugar while it expressed significant negative association with yield per plant. With all other characters, it expressed non significant association.

4.6. Path analysis

Path coefficient analysis was carried out at genotypic level to find out the direct and indirect influence of characters on yield per plant (Table 78 and 79).

4.6.1. Season I (Table 78)

4.6.1.1. Plant height

The plant height exerted a positive direct effect on yield. Mainly this character influenced the yield indirectly and positively through number of fruits and fruit weight whereas negatively through TLCV incidence at 75 DAT.

4.6.1.2. Number of branches per plant

This trait exerted a positive direct effect on yield. This character influenced the yield indirectly and positively through number of fruits per plant and total disease incidence at 75 DAT whereas negatively through TLCV incidence at 75 DAT.

4.6.1.3. Days to 50 per cent flowering

This trait exerted a positive direct effect on yield. This trait mainly influenced the yield indirectly and positively through Tv disease incidence at 75 DAT and fruit weight and negatively through total disease incidence and number of fruits per plant.

4.6.1.4. Number of fruits per plant

It exerted higher direct positive effect on yield. Indirectly it influenced the yield positively through total disease incidence at 75 DAT and negatively through average fruit weight and TLCV incidence at 75 DAT.

4.6.1.5. Average fruit weight

The direct effect to yield registered by this trait was high and positive while it influenced the indirectly and negatively through number of fruits per plant.

4.6.1.6. TSS

It showed a positive direct effect on yield. Mainly it influenced the yield indirectly and positively through total disease incidence and total phenol and negatively through fruit weight and Tv incidence at 75 DAT.

4.6.1.7. Acidity

It exerted a negative direct effect on yield. But the same influenced the yield positively through number of fruits per plant and Tv incidence at 75 DAT while negatively through fruit weight and TLCV incidence at 75 DAT.

4.6.1.8. Ascorbic acid

Ascorbic acid exerted a positive direct effect on yield. Its indirect effect on yield was positive through fruit weight and total disease incidence at 75 DAT and was negative through number of fruits per plant.

4.6.1.9. Lycopene

Lycopene exerted a negative direct effect on yield. Its indirect effect on yield was positive through total disease incidence at 75 DAT, number of fruits per plant and total phenol and negative through TLCV incidence at 75 DAT, fruit weight and OD phenol.

4.6.1.10. Total phenol

The direct effect of total phenol on yield was positive. It influenced the yield indirectly and positively through total disease incidence at 75 DAT and number of fruits per plant and negatively through OD phenol, TLCV and Tv incidence at 75 DAT.

4.6.1.11. OD phenol

It exerted a direct negative effect on yield. While it influenced the yield indirectly and positively through total disease incidence on 75 DAT and OD phenol and negatively through Tv and TLCV incidence at 75 DAT and average fruit weight.

4.6.1.12. TLCV incidence at 75 DAT

It exerted a direct positive effect on yield. Mainly it influenced the yield indirectly and negatively through Tv and total disease incidence at 75 DAT and number of fruits per plant.

4.6.1.13. Tv incidence at 75 DAT

It showed a direct positive effect on yield. It influenced the yield indirectly and negatively through TLCV and total disease incidence at 75 DAT.

4.6.1.14. Total disease incidence at 75 DAT

It exerted a direct negative effect on yield. Its indirect effect on yield was positive through TLCV and Tv incidence at 75 DAT and negative through number of fruits per plant and total phenol.

4.6.2. Season III (Table 79)

4.6.2.1. Plant height

Plant height exerted a direct positive effect on yield. Its indirect effect on yield was positive through TLCV incidence at 75 DAT, number of branches per plant and number of fruits per plant while negative through total sugar.

4.6.2.2. Number of branches per plant

Number of branches per plant had a negative direct effect on yield. Its indirect influence on yield was positive through plant height and number of fruits per plant and negative through fruit weight.

4.6.2.3. Days to 50 per cent flowering

It exerted low direct positive effect on yield. It influenced the yield indirectly and positively through fruit weight and total sugar whereas negatively through TLCV incidence at 75 DAT and number of fruits per plant.

4.6.2.4. Number of fruits per plant

It exerted highest direct positive effect on yield and it influenced the yield positively and indirectly through TLCV incidence at 15 DAT and plant height. While its indirect effect on yield was negative through number of branches per plant, fruit weight and total sugar.

4.6.2.5. Average fruit weight

It also exerted high positive and direct effect on yield. Its indirect effect on yield was negative through total disease incidence at 75 DAT, number of fruits per plant and OD phenol.

4.6.2.6. pH

pH of the fruit juice showed a positive direct effect on yield. It exerted its indirect positive effect on yield through number of branches per plant, average fruit weight and total sugar while negative effect through plant height and OD phenol.

4.6.2.7. Acidity

The direct effect of acidity on yield was positive. Its indirect effects on yield was positive through TLCV incidence at 75 DAT, number of fruits per plant and OD phenol while negative through fruit weight and sugar to acid ratio.

4.6.2.8. TSS

The direct effect of TSS on yield was negative. It influenced the yield indirectly and positively through plant height and number of branches per plant and negatively TLCV at 75 DAT and pectin content.

4.6.2.9. Ascorbic acid

Ascorbic acid exerted a direct negative effect on yield. Its indirect effect on yield was positive through total sugar and OD phenol and negative through TLCV incidence at 75 DAT, sugar to acid ratio and total phenol.

4.6.2.10. Lycopene

It exerted a direct positive effect on yield. Its indirect effect on yield was positive through number of fruits per plant, ascorbic acid and sugar to acid ratio while negative through plant height and fruit weight.

4.6.2.11. Total sugar

Total sugar had a direct negative effect on yield. Its indirect effect on yield was positive through total disease incidence at 75 DAT, plant height, number of fruits per plant and total sugar while negative through reducing sugar and OD phenol.

4.6.2.12. Reducing sugar

Reducing sugar exerted a negative direct effect on yield. Its indirect effect on yield was positive through TLCV and total disease incidence at 75 DAT and sugar to acid ratio and negative through fruit weight and total sugar.

4.6.2.13. Sugar to acid ratio

It exerted a direct positive effect on yield. It influenced the yield positively and indirectly through fruit weight and negatively through total sugar and OD phenol.

4.6.2.14. Pectin

Pectin exerted a negative direct effect on yield. Its indirect effect on yield was positive through total disease incidence at 75 DAT, number of branches per plant and ascorbic acid and negative through number of fruits per plant, TSS and total sugar.

4.6.2.15. Total phenol

Total phenol exerted a negative direct effect on yield. It exerted its indirect influence on yield positively through OD phenol, TLCV incidence at 75 DAT and number of fruits per plant and negatively through fruit weight and sugar to acid ratio.

4.6.2.16. OD phenol

OD phenol exerted a direct positive effect on yield. It had indirect effect on yield positively through total sugar and TLCV incidence at 75 DAT and negatively through fruit weight, sugar to acid ratio and total phenol.

4.6.2.17. TLCV incidence at 75 DAT

TLCV incidence exhibited a negative direct effect on yield. It exerted its indirect effect positively through Tv incidence at 75 DAT, number of branches per plant and total sugar and negatively through plant height and number of fruits per plant.

4.6.2.18. Tv incidence at 75 DAT

It exerted a direct positive effect on yield. It influenced the yield indirectly and positively through total disease incidence at 75 DAT, number of branches per plant, total phenol and sugar to acid ratio and negatively through TLCV incidence at 75 DAT, plant height, fruit weight and total sugar.

4.6.2.19. Total disease incidence at 75 DAT

It exerted a direct positive effect on yield. It exerted its indirect effect positively through number of branches per plant, sugar to acid ratio and OD Phenol and negatively through TLCV incidence at 75 DAT, plant height, fruit weight and total sugar.

4.7. Biochemical basis of resistance

4.7.1. Peroxidase (PO) activity (Table 80)

There existed significant differences in peroxidase activity among the genotypes, different hours of inoculation and between viruses inoculated and control treatment. The increase in PO activity was rapid upto 96 hour after inoculation and later on declined. Among the entries, the hybrids H 24 x CLN 2123A recorded maximum mean peroxidase activity of 7.49 followed by H 24 x LCR 1 (7.10) under inoculated conditions. In control, the differences were not so high although they also exhibited significant differences in peroxidase activity among the genotypes. The susceptible check variety CO 3 recorded lowest values in either of the case.

4.7.2. Polyphenol oxidase (PPO) activity (Table 81)

Here also significant differences were noticed among the entries, different hours of inoculation and between control and virus inoculated treatments. Increase in PPO activity was rapid upto 96 hours after inoculation. The hybrid H 24 x LCR 1 exhibited maximum mean peroxidase activity (0.837) followed by H 24 x CLN 2123A (0.806) where as the susceptible check variety recorded the lowest value of 0.608 under inoculated condition.

4.7.3. Phenylalanine ammonia – lyase (PAL) activity (Table 82)

There existed significant differences in PAL activity among the entries and between control and virus inoculated treatments. Out of two hybrids, only H 24 x LCR 1 exhibited significant difference in PAL activity between virus inoculated treatment and control. The PAL activity ranged from 0.510 μ mol (CO 3) to 0.937 μ mol (H 24 x LCR 1) under inoculated condition.

4.7.4. SDS-PAGE analysis (Plate 12)

In the present investigation, crude protein analysis of the plants challenge –inoculated simultaneously with both the viruses by SDS-PAGE expressed 19, 27 and 45 KDa proteins. Only in the hybrid H 24 x CLN 2123A, 27 KDa protein expression was prominent while in other genotypes the expression was not prominent upon challenge-inoculation with both the viruses done simultaneously under greenhouse conditions. Another protein of 19 KDa was prominent in all the genotypes except the hybrid H 24 x LCR 1. Hence even though the banding patterns were similar, differences were found in band intensity.

4.7.5. Isozyme analysis

In the PO isozyme analysis (Plate 10), a thick isoform (PO-3) was common in all the genotypes whereas two isoforms (PO-2 and 4) were more prominent in susceptible check. Out of five isoforms, only 4 were expressed by the hybrids. After 48 hrs of challenge-inoculation no major change in banding pattern was observed. Banding patterns were differentiated only at 96 hrs after graft inoculation. The hybrid H 24 x CLN 2123A exhibited a maximum of six isoforms whereas one of its parents *viz.*, CLN 2123A had only four isoforms.

With respect to PPO (Plate 11), the susceptible check (CO 3) showed five isoforms while all other genotypes had only four isoforms just before inoculation. Similar to PO, clear cut protein expression was visualized only at 96 hours after inoculation. The susceptible check exhibited thick isoforms (PO-1,2,3,4) while two clear cut isoforms were observed in the hybrid in H 24 x LCR 1 which was absent/faint in other genotypes.

CHAPTER V

DISCUSSION

In South India, Tomato Leaf Curl Virus (TLCV) and a Tospovirus (Tv) causing symptoms similar to Tomato Spotted Wilt Virus (TSWV) frequently confused with the latter, form a big menace to tomato cultivation especially if cultivated during January-July. Tospovirus came back on 'economic scene' after 30 years in correlation with the expansion of viral vector thrips. It is not uncommon that the crop infected with any one of these viruses at an earlier stage leaves no saleable produce. Conventional insecticidal application for the control of both these viruses through vector control is totally ineffective. Pesticide contamination of produce and environment has greatly increased in recent times. Even though biological control was conceptually good, it is not very much effective under high input conditions (Traboulsi, 1994). Molecular biologists have been working for almost two decades on the transformation of plants resistant to plant virus and there are only a few successful examples of commercial plant cultivars expressing a high level of resistance to viral diseases. On the other hand, the efforts of several plant breeders with the help of pathologists and entomologists using conventional breeding and virus screening techniques have resulted in the development of a number of worthy cultivars in different crops. These cultivars are the critical components of integrated pest and diseases management programmes and more important, have greatly contributed to the alleviation of poverty in developing countries throughout the world.

In tomato, earlier attempts to develop resistant hybrids / varieties to these viruses were made through distant hybridization involving complicated genetics of resistance with species like *Lycopersicon hirsutum*, *L. pimpinellifolium*, *L. hirsutum* f. *glabratum*, *L. peruvianum* etc. followed by backcrossing with cultivated genotypes and then by selection in the segregating progenies. Though the breeders succeeded in developing these lines resistant to individual virus by utilizing wild species, it was quite laborious, time consuming and lacked many attributes of horticultural and industrial demands. Smith and Gardner (1951) clearly brought out that TSWV

resistance was very difficult to recover and lost in the succeeding generation of back crosses with susceptible parents to improve fruit size. Later it was realized that exploiting cultivated types which exhibited tolerance / resistance against these viral diseases would be a viable method in resistance breeding programmes. The inevitable role of F₁ hybrids having disease resistance was expressed by Finlay (1952, 1953) after studying four sources of TSWV resistant genes. He concluded that it wouldn't be possible to breed a homozygous variety of tomato completely resistant to all the known strains of TSWV but could be achieved only in F₁ hybrids. Rick and Butler (1956) were of the opinion that disease resistance in tomato was mostly inherited as a dominant trait offering unique opportunity for achieving resistance in one generation. They further added that improvement would require more time and would be difficult with other breeding methods. Similarly Kalloo (1986) opined that it is extremely difficult to produce pureline variety of extreme earliness, smoothness, with large number of resistance genes and comparatively large fruited types, but the same can be achieved in F₁ hybrid combinations.

Tomato crop forms a classical example for the horticultural crop plant in which botanical and economic conditions are favourable for exploitation of hybrid vigour i.e., ease of handling flowers, emasculation and production of hybrids and large number of seeds per fruit. Although some lines / hybrids were claimed to be tolerant / resistant to TLCV by some corporate sector companies, they were not popular with farmers for one or other reasons. Moreover none of them showed resistance to tospovirus either singly or in combination with TLCV. This point is more important since breeding for resistance to one virus may aggravate the other virus problem and thereby losses due to disease still continue particularly when two viruses are equally devastating. In the light of such a need, the genetic improvement must be directed towards the identification of good general combiners to find out suitable combinations for exploiting hybrid vigour through identifying heterotic hybrids possessing desirable traits. Hence with all these ideas, an experiment was laid out with the assumption that combined resistance to both the viruses could be possible in F₁ hybrids by integrating different resistant genes from individual parents with simultaneous improvement in horticultural attributes.

5.1. Choice of the parents

Screening of genotypes for respective virus resistance is the first step before hybridization and breeding for resistance. At Tamil Nadu Agricultural University, Ragupathi (1995) isolated a few somaclonal variants from the leaf disc calli of the cultivated variety CO 3, which possessed high degree of resistance or tolerance to TLCV. From the R₆ population, single plants exhibiting high level of field tolerance to TLCV along with appreciable fruit size (~60g) have been identified by earlier workers. These somaclonal variants along with single plant selection from a few OP lines obtained from AVRDC, Taiwan and cultivated variety H 24 (Hisar Anmol) reported to be resistant to TLCV formed the initial material to screening for TLCV both under field and glasshouse conditions. Two other materials namely Stevens received from Dr. C. M. Rick of University of California, Davis and LE 415 from Kerala Agricultural University, Thrissur, India were tested for their field resistance to tospovirus prevailing in South India.

To the surprise, the cultivar Stevens reported to be having a dominant gene *Sw-5* for TSWV resistance was found to be highly susceptible to the tospovirus prevailing in Tamil Nadu which is specific in causing bronzing of stem and ventral surface of the leaf lamina without imparting any necrotic lesion. Similar observation on stable resistance breaking isolates of TSWV was noticed in genotypes of tomato carrying resistance gene *Sw-5* or one of its alleles in Australia (Latham and Jones, 1998). Moreover numerous viral disease symptoms not common and not noticed by tomato breeders in the experimental study area were also found to be present in the cultivar Stevens. It was thought that the inclusion of 'Stevens' in the present breeding investigation might pave way for the introduction of new viral disease(s), if grown continuously. Hence that particular highly acclaimed variety was not taken as parent for hybridization.

Since the genetics of resistance of the genotypes except for H 24 was not known, the criterion that genotypes exhibiting lower incidence of intended virus under high inoculum pressure even though having only medium fruit size was given due weightage so that the probability of losing the resistance was less. Similar opinion on inclusion of parental lines that

possessed good levels of field resistance or rate reducing resistance to TSWV was reported by Boiteux *et al.* (1999).

Based on the above criteria, five genotypes LCR 1, LCR 3, LCR 9, H 24 and CLN 2123A and one line LE 415 were chosen as preferred parents for developing F₁ hybrids with combined resistance to both the viruses. Even though the genotypes were selected with high level of resistance as most important criterion, the required level of geographical and genetic diversity were also seemed to be met out. LCR 1 and LCR 9 originated from same parent (CO 3) hence we can't expect much diversity both genetically and geographically even though they seemed to be with entirely different plant morphology. But H 24 obtained from wild parent hence might satisfy the requirement of genetic diversity. Lines from AVRDC, Taiwan *viz.* LCR 3 and CLN 2123A, latter from multiple crosses involving four parents were also of different plant and fruit characters. Hence the selected lines satisfied the requirement of intermediate level of genetic diversity for exhibition of maximum heterosis as stated by Moll *et al.* (1974).

It was observed that H 24 and CLN 2123A were also found to be free from tospovirus under field condition. Hence to harness this added advantage of these genotypes in bringing out both the disease resistance along with good horticultural traits from other selected genotypes in all possible manner, all the selected parents were crossed in all possible combinations (direct and reciprocal) to get maximum number of cross combinations. This full diallel mating system, one of the biometrical approaches for genetic analysis of traits controlled by polygenes, determine the *gca* of the parents and *sca* of the hybrids and thereby helps in knowing about gene action of the character and building up of a population with favourable genes. Past literature surveyed has adequately shown that there could be considerable differences between direct and reciprocal crosses in tomato.

With such a background, an attempt has been made in the present study to select the best parents and hybrids, measure the extent of heterosis and nature of gene action and to assess the underlying mechanism therein through a full diallel analysis involving both numerical and graphical approaches in two different seasons. Correlation and path analysis will help the

breeders to define the selection indices for the breeding programme. Apart from that, three field experiments were conducted to confirm the findings and for formulating agrotechniques etc.

5.2. Disease incidence

Perusal of all the three seasons studied under unprotected conditions for different viral disease incidence showed that season I recorded higher total disease incidence (87.98 per cent) in the susceptible check (CO 3) (Table 20c) followed by season II (71.34 per cent; Table 27c) and season III (58.01 per cent; Table 32) at 90 DAT, while hybrids and parents had maximum value in season II (41.43 Vs. 33.14 per cent; Table 27c) followed by season I (39.01 Vs. 30.34 per cent; Table 20c) and season III (23.92 Vs. 32.09 per cent; Table 32). This has sufficiently showed that season has got tremendous impact on disease incidence. The differences in the pattern of disease incidence of susceptible check variety, hybrids and their parents in three different seasons might be due to their varying response to the virus diseases prevalent in these seasons. But season III has to be looked in different angle, since it involved only selected eight hybrids and their parents, hence apart from climatic condition prevailing in the respective symptom expression, their inherent genetic resistance could tend to lower the PDI value at 90 DAT. Such seasonal influence on the viral disease incidence in tomato was noticed by several authors (Vijayakumar, 1987; Tomlinson; 1987, Kumar 1988; Saikia and Muniyappa 1989; Jasmine, 1991; Singh and Tripathi, 1991; Chermiti *et al.* 1993; Nainar (1996) and Sankari, 2000).

Coming to individual virus incidence for the three seasons, TLCV incidence at 90 DAT in susceptible check (CO 3) variety was the highest in season II (32.64 per cent; Table 27a) followed by season I (19.63 per cent; Table 20a) and season III (15.91 per cent; Table 32). Almost similar trend was noticed over different seasons for both parents and hybrids (Table 27a, Table 20a and Table 32). This has adequately revealed that the climatic condition prevailing in second season was more congenial for TLCV disease incidence irrespective of the genotypes (i.e.) either susceptible check or parents or hybrids. Hence March-transplanted crop suffered

much due to TLCV incidence. The weather data during season II showed mean maximum and minimum temperature of 34.05°C and 22.83°C and a minimum and maximum relative humidity of 81.75 and 43.25 per cent, with scanty rainfall. This gives a general idea on conducive climatic conditions for rapid spread of this virus by the whitefly vector whose population increase was also correlated with above parameters. Similar findings of these climatic conditions on maximum spread of this virus were noted by Sastry and Singh (1976), Butter and Rataul (1978) and Singh *et al.* (1999). Vijayakumar (1987) and Saikia and Muniyappa (1989) also reported that TLCV incidence was high during summer months in crops planted during February to March in various locations of South India. The comparatively low TLCV incidence during rainy season (season III) might be because of low vector incidence along with the inclusion of hybrids selected for low PDI value for study. This is in corroboration with the findings of Singh and Sastry (1976).

The tospovirus incidence at 90 DAT in susceptible check variety (CO 3) was the highest in season I (54.09 per cent) while it was 38.07 per cent in season II. There was no major change in the mean Tv incidence in hybrids (25.95 vs. 27.12 per cent) and parents (21.59 vs. 20.76 per cent) in both the seasons. Hence it can be concluded that Tv incidence either in hybrids or in parents was at constant in both the season I and II under unprotected conditions in the experimental area. The comparatively lower value of Tv incidence for susceptible check in the season II might be due to peak incidence of TLCV since the check variety was susceptible to both the diseases equally. Competition between both the viruses for the available sites of infection might have caused the lower incidence of Tv in susceptible check in the season II. This is in line with the works of Kassanis (1963). From the above two paragraphs, it is also clear that TLCV infection was more prevalent in season II whereas Tv infection at both season I and II. Higher incidence of TSWV in tomato in January-March planting was also observed by Kumar (1988) and Singh and Tripathi (1991).

In season III, comparatively lower mean incidence of Tv 90 DAT in parents and hybrids (22.24 Vs. 17.44 per cent; Table 32) might be most probably due to selection of hybrids of

exhibiting lower PDI value from previous two experiments than due to rainy season since the susceptible check showed reasonably higher (42.15 per cent; Table 32) PDI value for Tv at 90 DAT

The incidence due to combined infection of both Tv and TLCV was negligible. One or two such observed plants were infected with only bronze symptom-causing Tv not the necrotic one. It might be due to decrease in number of infection sites through changes in metabolism of the plant from the first infection thereby rendering comparatively less number of sites for infection by the second virus or due to depletion of essential host materials thereby arresting the multiplication of second virus (Kassanis, 1963). Interference between viruses may also be due to a concomitant delay in the synthesis of coat protein of suppressed virus and replication of suppressed virus RNA and restricted transport of virus out of infection centres. Eastwell and Kalmer (1997) noticed the same phenomenon between Cowpea Mosaic Virus (CpMV) against Cowpea Severe Mosaic Virus (CpSeMV). Interference among two serologically unrelated viruses i.e., Potato Virus Y (PVY) and Pepper Mottle Virus (PeMV) was also reported by Alegbejo and Nelson (1982). This phenomenon necessitates the need for breeding cultivars resistant to both the viruses since the breeding for resistance to one virus may aggravate the other virus problem. Even within the two distinct symptoms of Tv, those plants previously infected with bronzing symptom were not infected with necrotic symptom but not vice-versa. Elaborate study on these two distinct symptoms needs to be done in the due course for successful management of this disease complex.

Coming to the second aspect, that is the nature of incidence and progress of disease incidence at different stages of crop growth, usually the curve is sigmoidal. During pre-flowering phase (15 DAT), there was practically no incidence in the hybrids in the current investigation. Generally from early flowering phase (30 DAT) onwards, the infection starts rapidly upto peak harvesting stage (75 DAT) and then remained as such. This trend was observed more commonly in season I and II. In season III, this trend was almost disturbed due to the fact that here it involved hybrids selected for lower PDI values from previous two seasons.

The hybrids which exhibited comparatively lower PDI at 75 DAT showed either of the three trends *viz.* slow and gradual rise in PDI from 15 DAT or rapid increase in PDI in either flowering (30 DAT) or fruit formation (45 DAT) phase followed by minor or no increase in PDI in the successive phases or infection only at later stages. These hybrids endure or slow down the epidemics of the disease more efficiently than the susceptible cultivars under similar field conditions. The hybrids H 24 x LCR 1, LE 415 x LCR 3, LCR 9 x H 24, H 24 x LCR 9 and CLN 2123A x LCR 3 recorded lowest total PDI values at 75 DAT i.e. 15.15, 18.54, 20.90, 21.76 and 22.14 per cent respectively in the season I, whereas in the season II, H 24 x CLN 2123A (19.19), LCR 1 x CLN 2123A (22.14), CLN 2123A x LCR 1 (22.96), LCR 9 x H 24 (23.34) and CLN 2123A x LCR 9 (23.37) proved the same. In the season III, out of 8 hybrids studied, H 24 x LCR 1 (14.87), LCR 1 x H 24 (15.15), H 24 x CLN 2123A (20.41), LE 415 x LCR 1 (21.43) and LCR 1 x CLN 2123A (22.00) proved the merit. In these hybrids, one of the above said trends might have yielded this lower PDI values. Since both the diseases attack the crop at any stage, no relation between different stages of crop growth and rate of disease attack was observed. In other words the rate of increase in both the diseases was almost of the same trend.

Severity of expression of symptoms can be very well studied through Coefficient of Infection (CI) values at 75 DAT which was developed for assessing TLCV. It was not developed for Tv which might be due to the fact of outright killing of plants especially if infected with necrotic type in any stage of crop growth. In the first season, the parents LCR 3, CLN 2123A, H 24 and LE 415 and a few hybrids *viz.* LCR 9 x CLN 2123A, LCR 1 x CLN 2123A, H 24 x CLN 2123A, H 24 x LCR 1 recorded zero value of CI for TLCV. Twenty out of 30 hybrids studied had CI value between 0.00 and 4.10 (Highly Resistant), while in second season, 19 showed Highly Resistant reaction. In the season III, all the selected hybrids tested showed Highly Resistant reaction. This itself gives an indication that whether the plant can yield some fruits even after infection or not. In many occasions, in spite of mild incidence of disease in resistant lines, plant growth and fruiting still continued. Hence these hybrids may be profitably grown for economic yield under hot spot areas for TLCV infection. The practicability of using

these hybrids vested with testing them under various conditions and environments. Kalloo and Banerjee (1990b) also noted the same phenomenon in some TLCV resistant lines.

Delayed expression of symptom is yet another mechanism of enduring or slowing down of epidemics of the disease. In the present study, for TLCV incidence such phenomenon was noticed. Hybrids CLN 2123A x LCR 9, CLN 2123A x LCR 1, LCR 3 x CLN 2123A, LCR 9 x LE 415, H 24 x LE 415 and LE 415 x LCR 1 exhibited TLCV symptoms only at first harvest stage (60 DAT) in the season I. In season II, CLN 2123A x LCR 9, H 24 x LCR 1, LCR 1 x CLN 2123A, H 24 x CLN 2123A, LCR 9 x H 24, LCR 9 x LE 415 and CLN 2123A x LE 415 showed the symptoms only at 60 DAT or later. This phenomenon was very well seen in H 24 x CLN 2123A at season III where first symptom expression was seen only on 75 DAT. For Tv, almost all hybrids showed first symptom expression within 45 DAT and none was exhibiting zero PDI in all the three seasons studied. The delayed expression of the symptoms in the hybrid might be due to non-preference by the vector during early stages of growth. Roane (1973) suggested that resistance to the virus was also due to the ability of the host to tolerate the presence of high concentration of virus within its tissue without exhibiting symptoms or without suffering a loss of production. Any one or all such mechanisms may be operating in the parents employed in the present study. Delayed symptom expression of TLCV infected plants resulting in acceptable yields was also reported by Pilowsky and Cohen (1990) and Sankari (2000).

Both the above mentioned mechanisms namely less severity of symptoms expression and delayed symptom expression help for the accurate selection of tolerant tomato plants since earlier the infection, more the loss than the infection at later stages. The works of Ragupathi (1995) and Sankari (2000) in TLCV, Kumar (1988) and Vanitha and Suresh (2002) in TSWV proved the same. Importance of the age of the plant at the time of infection on selection of tolerant plants was also reported by Smith and Gardner (1951) and Vidavsky *et al.* (1998). Based on the results from different seasons, it is safe to conclude that H 24 x CLN 2123A showed delayed and mild symptoms of TLCV.

In the confirmatory trial for the two best performing hybrids planted during peak thrips activity period of mid December 2002, Tv incidence was noticed even at 15 DAT in both the hybrids. At 75 DAT, these two hybrids *viz.*, H 24 x LCR 1, and H 24 x CLN 2123A exhibited 45.71 and 37.50 per cent total disease incidence while the susceptible check exhibited 98.15 per cent infection.

Thus search for total resistance source among the cultivars in India and other tomato growing regions of the world have proved unfruitful. The present study also confirms the non availability of immune sources among the hybrids for both the diseases and the focus must be towards employing this field resistant / tolerant source for disease resistance breeding programmes.

5.3. Evaluation of parents and hybrids in seasons I and II

Selection of parents based on phenotypic expression is easy when the particular character is controlled by a few genes and inherited in a simple way. But for continuously varying traits like yield and its components which are controlled by many genes, selection should be based on *per se* performance and combining ability thereby potentiality of a genotype to be used as parent in hybridization for commercial production of hybrid may be judged (Ceballos, 1998). In addition to the *per se* and *sca*, hybrids can also be judged by the heterosis expressed. The efficiency of such selection depends on the nature and extent of genetic variability and the degree of heritability of such desirable characters.

Since the second season crop was evaluated under severe summer, fruit set was adversely affected in most of the hybrids and parents. Moreover fruit size was also reduced much. Hence observation on yield and other biometrical characters was not taken up with the view that these yields can not be representative of the true potential yield. Hence disease incidence alone was observed. Comparison of mean performance of six parents and 30 hybrids in general revealed that parents had favourable *per se* performance for the characters *viz.* TLCV, Tv and total disease incidence (both the seasons), total and OD phenol, TSS, days to 50 per cent flowering, average

fruit weight than their hybrids. **It implies that majority of hybrids exhibited unfavourable heterotic response and only a few hybrids could be worthwhile to select.**

Before embarking into evaluation aspects, validation of statistical requirements and assumptions needs to be investigated. Significant differences among the genotypes (both parents and hybrids) in both the seasons indicated enough scope for selection. Most of the crosses, although not all, exhibited significant difference from their mid or better or best parental values. In diallel analysis, variances due to *gca*, *sca* and *sca* of reciprocal hybrids were significant for all the characters studied in both the seasons, hence null hypothesis got rejected. It implies that both additive and non additive gene action played a role in the inheritance of these characters.

Apart from that, certain assumptions need validation (Griffing 1956).

1. Normal diploid segregation
2. No reciprocal differences
3. Parental homozygosity
4. No multiple alleles
5. Unlinked gene distribution
6. No epistasis

The validity of these assumptions was examined as follows.

1. Tomato is a self pollinated, true diploid forming species with twelve sets of bivalents having a basic chromosome number of twelve (Winkler 1909; Humphrey, 1934). Hence the assumption of diploid segregation can be considered as satisfied.
2. Significant reciprocal differences were observed for all the eighteen characters under investigation. However, by appropriate choice of Griffings (1956a) method of analysis, the phenomenon of reciprocal difference was overcome. In the analysis of array variance and covariance of genetic analysis, the mean of the direct and reciprocal crosses was used as suggested by Hayman (1954a).
3. Care was taken to achieve homozygosity in the parents by selfing over six generations (done by previous workers).

4. The assumption of no epistasis, no multiple allelism and no linkage could not be clearly assessed in the present material, as it was difficult to evaluate independently each one. However, the assumption of absence of multiple allelism and independent gene distribution was found to be of little consequence (Kempthorne, 1956) and seemed unlikely to disturb the genetic analysis of the data.

5.4. Evaluation of parents based on *per se* and combining ability and hybrids on *per se*, heterosis and combining ability estimates

Plant height is an important component based on which the growth and vigour of the plants are assessed. In general, hybrids involving CLN 2123A as either parent, H 24 as female parent recorded higher plant height. Highest plant height was exhibited by H 24 x CLN 2123A, CLN 2123A x LCR 9 and H 24 x LCR 9. It is worthy to mention here that H 24 recorded lower plant height (53.10 cm) but contributed immensely to improve plant height when used as female parent especially with CLN 2123A, LCR 9 and LE 415. The same hybrids exhibited maximum significant positive heterosis over better parent involving medium x high performance combinations. Heterosis for plant height had also been reported by Kumar *et al.* (1995), Sankari (2000) and Sekar (2001a).

Gca effects also confirmed that parents involved in production of highest *per se* hybrids, possessed significant positive *gca*. Coming to the *sca* effects, the three above mentioned hybrids also showed higher significant (positive) *sca* effects, hence additive x additive gene interaction might be involved in the expression. For their better expression, such combination could be best studied for fixing the favourable alleles in F₂ and later generations. CLN 2123A x LE 415 even though possessed higher positive *sca*, one of its parents (LE 415) had significant negative *gca* and lowest *per se*, hence additive x dominance interaction played a role in the above cross. Another cross with high *sca* viz., LCR 1 x LE 415 had both the parents with significant negative *gca* and lower *per se*. It might be due to non additive gene action of complementary or epistatic nature. Nullifying effects on unfavourable epistatic genes present in

either parent by the genes sponsored might have resulted in such *sca* effect. Both the above two hybrids can be well exploited for heterosis breeding. Existence of such additive and non additive gene action for plant height was also reported by Peter and Rai (1980) Natarajan (1988) and Sekar (2001a).

Number of branches per plant influences the yield to a significant extent through facilitating the production of more number of flowers and in the maintenance of optimum Leaf Area Index. In the present study, LCR 9, CLN 2123A and H 24 had both higher *per se* and significant positive *gca* for this character. The parent LE 415 even though had higher *per se* but had negative *gca* indicating parental *per se* may not be a good criterion for selection of parent to improve the trait. The combination of LCR 9 with CLN 2123A and H 24 (both direct and reciprocal) exhibited high *per se*, high heterosis over best parent. Existence of positive heterosis over best parent was also reported by Kumar *et al.* (1995) Mageswari (1996) and Sankari (2000). All the four crosses mentioned above *viz.* LCR 9 x CLN 2123A, LCR 9 x H 24, CLN 2123A x LCR 9 and H 24 x LCR 9, had significant positive *sca* indicating the role of additive x additive gene action. Such combinations could be used for selecting recombinants as purelines in the later generations. Additive gene action for the trait was also reported by Sekar (2001a). LE 415 even though possessed high *per se*, had negative (medium) *gca*. But three hybrids involving it as female parents had positive significant *sca*, of them two (LE 415 x LCR 1 and LE 415 x LCR 3) had other parent with significant negative *gca* implying the role of complementary gene action or the mutual cancellation of unfavourable epistatic genes present in the parents by the genes sponsored. Another cross (LE 415 x CLN 2123A) had other parent with significant positive *gca* and positive heterobeltiosis indicating the role of additive x dominant gene interaction, which proved its use as good F₁ hybrid for exploitation of heterosis. But its reciprocal cross (CLN 2123A x LE 415) had significantly negative *sca* indicating the role of either maternal effects or genic –cytoplasmic interaction (Jinks, 1964). The existence of non additive gene action for branches per plant was reported by Kalloo *et al.* (1974), Peter and Rai (1980) and Sankari (2000).

Early flowering is a desirable attribute considering the magnitude of per day productivity. A genotype which produces maximum flowers in the early phase of crop duration would be desirable. In the present study, the parents LCR 3 and H 24 recorded earlier flowering which was also manifested in its hybrids. Both the parents also exhibited significant negative *gca* hence proved as good general combiners for earliness. Among the hybrids, LCR 3 x H 24, LE 415 x LCR 3, LCR 3 x LE 415 and LCR 1 x LCR 3 and H 24 x CLN 2123A recorded earliest flowering. These early flowering hybrids could have a greater period of reproductive phase and resulted in a relatively high yield. None of these hybrids showed negative heterosis value over better and best parent. But all have registered negative *sca*. Hence additive x additive interaction might have played a role in LCR 3 x H 24 whose parents had negative *gca*. All other four hybrids mentioned above involved one of the parents with unfavourable *per se* and positive *gca* indicating the role of additive x dominance gene interaction. Hence breeding procedures such as intermating and recurrent selection can be made to harness the fixable additive gene effects without dissipating dominance effects in these hybrids. The role of additive gene action for this character was reported by Banerjee and Kalloo (1989), Aruna (1992) and Sundaram *et al.* (1994), whereas involvement of both additive and dominance gene action was reported by Jawaharlal (1994), Premalakshmi (2001) and Sekar (2001a). Hence for earliness, LCR 3 and H 24 proved to be the best general combiners as they showed their superiority in majority of the crosses.

Number of fruits is the most important yield component closely linked with yield. Among the six parents, LCR 3 recorded 90.78 per cent more fruits than the next best parent CLN 2123A. But it was not able to reflect its superiority except in two crosses (LE 415 x LCR 3 and H 24 x LCR 3) out of ten crosses. But H 24 as a female parent resulted in crosses with significantly high *per se* except in one cross H 24 x LCR 1. The parent LE 415 even though had low *per se*, exhibited positive significant *gca* and eight out of ten crosses in which this genotype was involved as parent exhibited positive *sca*. This confirms that combining ability can't always be judged accurately by their *per se* performance.

Among the hybrids, H 24 x CLN 2123A, H 24 x LE 415 recorded high *per se*, heterosis over better parent and significant positive *sca*. Their parents also had positive *gca*, indicating the role additive x additive gene interaction which would have resulted in more number of fruits in these hybrids. Similar gene interaction also might be operating in LE 415 x LCR 3, although it revealed heterosis only over mid parental values. The role of additive gene action for number of fruits was also reported earlier by Kalloo *et al.* (1974), Aruna (1992) and Premalakshmi (2001).

Even though the cross LE 415 x LCR 3 recorded a quite higher number of fruits (74.20), its reciprocal cross recorded only 44.50 fruits with negative relative heterosis. This clearly showed that reciprocal difference does exist in tomato for this character. Reciprocal differences might be either due to maternal effects or due to genic – cytoplasmic difference. Such reciprocal difference for this character was also reported by Trinklein and Lambeth (1975). Hansen and Bagghat (1977) and Baker *et al.* (1986) reported that even plant to plant genotypic differences that had small phenotypic differences within an inbred line could also result in reciprocal difference.

Fruit weight is yet another important trait contributing directly to the yield. Among the six parents, LCR 1 had highest fruit weight followed by LCR 9. The present study showed parallelism between *per se* and *gca* effects. Hence they proved to be the best combiners. The hybrids LCR 1 x LCR 9, CLN 2123A x LCR 9, LCR 1 x H 24, LCR 1 x CLN 2123A and H 24 x LCR 1 registered highest *per se* indicating the involvement of at least one of the best combiners. None of the hybrids recorded supremacy in heterosis over best parent. Only CLN 2123A x LCR 9 out of these crosses registered positive heterosis over mid and better parent. In the above hybrids, LCR 1 x LCR 9 had positive significant *sca* confirming the role of additive x additive gene interaction. Two hybrids (LCR 1 x CLN 2123A and H 24 x LCR 1) out of five discussed above, exhibited significant negative *sca*. The dominant genes present in low *gca* parent probably by overdominance would have brought down the value drastically in these combinations. In tomato, small fruited character is reported to be dominant over large fruited types (Mac Arthur and Butler, 1938 and Sankari, 2000).

Yield is a complex character and is dependent on its component traits and their inheritance. Nineteen out of 30 hybrids recorded positive heterosis estimates over their respective mid parental values. Heterosis for yield was reported by Mandal *et al.* (1992), Pujari and Kale (1994), Kumar *et al.* (1995) and Indu Rani (2002). Heterosis over the best parent was observed only in four hybrids viz., H 24 x LE 415, H 24 x CLN 2123A, H 24 x LCR 9 and LE 415 x LCR 1. Such high heterotic hybrids mostly involved low x high, medium x medium and low x medium parental combinations. Williams (1959) suggested that heterosis for yield was the consequence of multiplicative relationship between the component characters of the yield complex. Modifiers may also aid in the reflection of these component traits to yield. Yield in tomato is primarily made up of number of fruits, fruit weight and heterosis for total yield can occur in hybrids in which the component characters merely show dominance or intermediate level of expression. For this to occur, the parents must differ with regard to the level of expression of each of the components and neither must have a monopoly at high or low expression in both the unit characters. The result of the present investigation justifies the above statement. This is in line with the works of Aruna and Veeraragavathatham (1996) and Sankari (2000). Perusal of the results of present study also indicated that heterosis in fruit number rather than fruit weight has reflected in heterosis for yield. Better allocation of photoassimilates to the more number of developing fruits not at the expense of fruit weight could have helped to increase the yield ultimately.

The parent LCR 3 had the highest *per se* performance but it exhibited significant negative *gca*. This confirms that combining ability of the parents can't always be judged accurately by their *per se* performance especially for yield which is polygenically controlled. The result was in conformity with the findings of Govindarasu *et al.* (1982) and Premalakshmi (2001). Out of four hybrids which exhibited positive heterosis over the best parent, one of them had the parental combinations having additive x additive gene interaction while other three hybrids had high x low *gca* combination suggesting additive x dominance gene interaction. Additive and non

additive gene actions were also reported by Jamwal *et al.* (1984), Aruna (1992) and Rai *et al.* (1997).

TSS forms an inevitable quality parameter since it is directly related to the output of processed products viz. puree, paste etc. Hence main objective of the breeder interested in quality improvement programme should be to evolve a better hybrid combination with high yield as well as TSS, which is of course a rare blend since both the characters are negatively related. Probable reason for such negative relation could be the lack of availability of sufficient photosynthates to be spared for increasing TSS in the high yielding cultivar particularly having a heavy and concentrated fruit set on the compact vine (Stevens, 1978).

Girdharilal *et al.* (1967) recommended a TSS content of 5.6 per cent as the best for juice purposes in tomato. None of the genotypes either parents or hybrids recorded such a higher value. However, for processing purpose ($> 4.5^\circ$ Brix), LE 415 among the parents, LCR 1 x LCR 9, CLN 2123A x LCR 1, LCR 1 x LCR 3, CLN 2123A x LCR 3 and CLN 2123A x LCR 1 among the hybrids are noteworthy. Physiologically, they have the ability to mobilize more carbohydrates for synthesis of sugars from their total assimilates in spite of diversion of photoassimilates for yield improvement and respiration. None of the above hybrids registered appreciable fruit yield except LCR 1 x CLN 2123A which recorded appreciable fruit yield of 2.52 kg per plant simultaneously possessing higher TSS of 4.52° Brix. This hybrid probably because of its better photosynthetic partitioning and translocation efficiency would have accumulated more dry matter into economic part namely the developing fruits. Stevens and Rudich (1978) opined that genotypes of tomato with better photosynthetic efficiency coupled with partitioning efficiency simultaneously possessing low respiratory rate by the developing fruit could divert more assimilates for TSS. Out of the 30 hybrids studied, only four had positive heterosis over best parent. Hence improvement of TSS content in hybrids will not be so rapid unlike other characters.

In the present study, the parents LE 415, CLN 2123A and H 24 recorded higher TSS which are statistically on par. Although LE 415 recorded highest TSS among the parents it could

not transmit this character to the crosses which was evident from its negative *gca*. Four out of the five mentioned hybrids of noteworthy nature showed additive x additive gene interaction while LCR 1 x LCR 9 exhibited additive x dominance interaction which could be well utilized as F₁ hybrid. Additive gene action for TSS was also reported by Dev and Rattan (1996), Kumar *et al.* (1997), Wang Lei *et al.* (1998) and Sekar (2001b) while non additive gene action by Gibrel (1983) and Kurian (1990). In many of the hybrids, reciprocal difference was quite obvious in terms of *per se* as well as *sca*. Probably cytoplasmic - genic interaction especially cytoplasmic genes of female parent might be the reason for the difference in value of traits under study in direct and reciprocal crosses. Reciprocal difference for TSS was also reported by Ibarbia *et al.* (1969) and Premalakshmi (2001).

Acidity is one of the important traits in tomato for processing. Acidity provides flavour and acts as preservative in the canned product. According to Gould and Berry (1972) citric acid content of 0.35-0.55 per cent is required for processing. The hybrid LCR 3 x CLN 2123A and its reciprocal cross had exhibited highest acidity, despite the fact that direct cross alone had resulted in significant positive heterosis over best parent. The parents LCR 3 and CLN 2123A had both high *per se* and *gca*, suggesting that they are good combiners. The parent H 24 though possessed good acidity *per se*, had zero value of *gca* indicating that it had average performance in ten crosses in which it involved and it is under the influence of the other parent to produce F₁ hybrids. The cross CLN 2123A x LCR 3 recorded significant negative *sca* while its parents had significant positive *gca* indicating the role of non additive gene action of epistatic nature. But its reciprocal cross had significant positive *sca* revealing maternal effect of LCR 3 through cytoplasmic genes or cytoplasmic – genic interaction. Such reciprocal differences were also noticed earlier by Ibarbia *et al.* (1969) for acidity. Involvement of additive as well as non additive gene action of epistatic nature was also reported by Lukyanenko and Lukyanenko (1986) for this trait.

Ascorbic acid, in addition to the contribution to the nutritive value of tomato fruits, has an added role of better retention of colour and flavour of products prepared from tomato. Among

the parents, LCR 3 only had the high *per se* with significantly positive *gca* and proved to be a good combiner. Only five hybrids exhibited significant positive heterosis over the best parent and all of them had exhibited positive *sca* also. Of them, two hybrids viz. LE 415 x LCR 9 and LCR 1 x H 24 had one of their parents with significant negative *gca*. Hence apart from additive x additive, additive x dominant gene interaction also exists. Both the gene actions have the added advantage of selecting recombinants in later generation in addition to exploitation of hybrid vigour. Kalloo *et al.* (1974) reported non additive gene action for the trait.

Lycopene, the pigment that imparts dark colour to ripe tomatoes has been studied in detail with regard to its association in reducing the risk of certain type of cancer and heart diseases. It decides the optimum stage of ripening. Since the use of synthetic colours are banned, processing industry requires the cultivars with high lycopene content for imparting attractive colour. The same criterion holds good for fresh market too. The parents H 24 and CLN 2123A proved to be good combiners. None of the hybrids exhibited positive heterosis over best parent. The hybrids H 24 x LCR 1, CLN 2123A x LCR 3, H 24 x CLN 2123A and LE 415 x LCR 3 had exhibited high *per se*.

The hybrid H 24 x CLN 2123A was found to be a combination of high *gca* x high *gca* leading to high *sca* hybrid suggesting the additive x additive gene interaction for this trait. Hence its parents appeared to be worthy in varietal improvement programme. It is suggested from this study that population involving these parents in multiple crossing programme, can be developed for isolating desirable lines. The cross LE 415 x LCR 3 had both the parents with negative *gca* but resulted in a hybrid with significant positive *sca*. This may be due to epistatic genes present in both the parents and mutual cancellation of epistatic genes resulting in better hybrids. In H 24 x LCR 3 and CLN 2123A x LCR 3 it seems that epistatic genes present in LCR 3, capable of masking the effect of its own genes for production of lycopene probably by overdominance would have brought down the value drastically in these two hybrids thereby result in negative *sca*. The existence of dominant nature of gene action for lycopene was also reported by Bhutani and Kalloo (1983) and Premalakshmi (2001).

Total phenol content in the leaves indicates the degree of resistance to the disease. In the resistant tissues, biochemical reactions leading to the accumulation of phenolics were rapid (Fuchs, 1971). Even though LE 415 had high *per se*, this was not a good combiner when compared to H 24. Only the hybrid H 24 x CLN 2123A registered positive heterosis over the best parent. Significant negative heterosis in other hybrids is in line with the findings of Narayana and Reddy (1980), Singh and Abidi (1988), Sudha (1991) and Sankari (2000). Crosses exhibiting high *per se* (H 24 x CLN 2123A, LCR 3 x H 24, H 24 x LCR 3, and H 24 x LCR 1) had also positive *sca* except one (H 24 x LCR 3). Both additive and epistatic gene actions were inferred for this trait from the present study.

Much variation was seen among the parents and hybrids for OD phenol content in leaves. None of the hybrids showed positive heterosis over the best parent while only two hybrids (LCR 1 x LCR 9 and H 24 x LCR 1) had significant positive heterobeltiosis. LE 415 seemed to be a good parent both in terms of *per se* and *gca*.

Considering the disease incidence aspects in season I, TLCV incidence at 75 DAT was nil in the parents LCR 3, CLN 2123A, H 24 and LE 415, but only CLN 2123A and H 24 proved to be the best combiners by exhibiting significantly negative *gca*. No incidence of TLCV was also noticed in the hybrids H 24 x LCR 1, LCR 9 x CLN 2123A, LCR 1 x CLN 2123A and H 24 x CLN 2123A under unprotected natural conditions. Since both the hybrid and one of the parents (better) or both the parents had zero value, calculation of heterosis over best / better parent is meaningless. These hybrids also exhibited desirable negative *sca*. Out of them, first three hybrids had one parent with positive *gca*, hence indicating the role of dominance x additive interaction while H 24 x CLN 2123A exhibited additive x additive interaction for their Highly Resistant reaction *viz.* Coefficient of Infection value being zero (Kalloo and Banerjee, 1987). Both of these interactions could be exploited very well in F₁ generation.

In the second season, all the parents and hybrids exhibited TLCV incidence except CLN 2123A x LCR 9, LCR 9 x H 24 and LCR 9 x LE 415. Least incidence (below 5.00 per cent) was exhibited in LCR 1 x CLN 2123A and H 24 x CLN 2123A. All these hybrids also exhibited

desired negative heterosis over the best parent (CLN 2123A). The resistance to TLCV in these hybrids could be attributed to the prevention of transmission or prevention of early establishment of viruses by the resistant genes of the hybrids or due to their inherent mechanism to deter the vectors transmitting the disease. According to Valkonen (2002) resistance to viruses may, hypothetically interfere with any steps in the virus infection cycle viz., entry of the virus or disassembly, replication, movement within and between the cells and transport from the site of initial infection to other parts of plant. The genotype CLN 2123A was proved to best general combiner with respect to TLCV disease resistance. All the above mentioned hybrids also exhibited favourable negative *sca*.

Additive x additive gene interaction was involved in the hybrids CLN 2123A x LCR 9, LCR 9 x H 24 and H 24 x CLN 2123A for their Highly Resistant reaction, whereas additive x dominant interaction played role in the other two hybrids for their Highly Resistant reaction. According to Fraser (1986) polygenic resistance might have occurred in two forms. First, resistance could depend on co-operative or cumulative action of many genes, all directly or primarily involved in the mechanism. This could include genes controlling a multiple step pathway leading to a single functional end product, or joint interaction of several genes, perhaps as sub units of a complex enzyme or structures, with some stage of pathogenesis. Effects could be additive, giving more effective resistance with greater number of resistance genes or obligately co-operative, where resistance only occurs with a full complement of genes required. Secondly modifier genes not themselves directly involved in the interaction with the virus might modulate the antiviral activity of one or more major genes. He supported the more probable occurrence of latter rather than former. Resistance to TLCV controlled by polygenes was reported by Berlinger *et al.* (1983) and Kegler (1994) in tomato.

In respect of resistance to Tv, the parents LCR 9, LCR 1 and LCR 3 proved to be the best combiners for reducing Tv incidence in the season I whereas in the season II, LCR 1 and H 24 proved to be best combiners. In the first season, least incidence of Tv was noticed in parents

LCR 9 (14.26 per cent) and LCR 1 (14.29 per cent) while the susceptible check recorded 52.15 per cent infection of Tv. In the second season, LE 415 (8.30 per cent), LCR 9 (18.37 per cent) and H 24 (19.09 per cent) recorded the least incidence while the susceptible check showed 37.09 per cent infection. This indicates the role of environment i.e. different response of existing distinct symptoms of Tosspovirus to environment influence. Besides the above, modifiers may also play a role. Czosnek *et al.* (1990) reported that existence of different strains could cause discrepancies in TYLCV resistance. The influence of modifiers in the expression of resistance to many viral diseases has been reported by several workers (Bagget, 1957; Waswart and Warker, 1961; Martin 1970 and Arumugam, 1977). The parents LCR 9 and H 24 seemed to be less influenced by the above factors since their PDI values remained almost same as compared to that of LE 415 whose PDI values were highly varying in both the seasons (28.57 Vs. 8.33 per cent).

Among the 30 hybrids, only four exhibited favourable negative heterosis over the best parent *viz.*, LE 415 x LCR 3, LCR 9 x H 24, LCR 3 x LCR 1 and CLN 2123A x LCR 3 in the season I. All these hybrids also exhibited favourable negative *sca*. Of which, additive x dominance interaction was involved in three hybrids and one exhibited additive x additive interaction controlling the incidence of Tv at 75 DAT. In the season II, only three hybrids out of 30 showed significant relative heterosis value (H 24 x LCR 3, H 24 x CLN 2123A, and CLN 2123A x LCR 1). Out of these three hybrids, only H 24 x LCR 3 showed favourable negative relative heterosis in the season I. Poor performance of most of the hybrids for virus incidence under unprotected conditions was also reported by Sankari (2000). All the above three hybrids exhibited additive x dominance gene interaction controlling the incidence of Tv at 75 DAT in the season II. Present work confirmed the findings of Kumar (1988) in TSWV. He suggested that simple selection might not be possible since resistance involving additive dominance and duplicate epistasis exists. The difference in PDI of the different hybrids over different seasons might be due to different distinct symptoms caused by this virus, its response to the other virus challenged, environmental factors such as temperature, humidity etc.

Considering the total disease incidence at 75 DAT which stage can be considered as the deciding factor for field resistance since further infection may have little influence on yield loss, in season I, among the parents LCR 3 and CLN 2123A had exhibited lower PDI whereas in the season II, LE 415 and LCR 9 exhibited the same indicating that disease incidence is not so uniform over the seasons as well as with different viruses. The hybrids H 24 x LCR 1, LE 415 x LCR 3, LCR 9 x H 24 and H 24 x LCR 9 recorded lowest total PDI in the season I. In the second season, H 24 x CLN 2123A, LCR 1 x CLN 2123A, CLN 2123A x LCR 1, LCR 9 x H 24 and CLN 2123A x LCR 9 recorded the same indicating the role of CLN 2123A in reducing the incidence of both the viruses since in the second season, both the diseases were equally competent as observed from the susceptible check value.

Mere reduced disease incidence and reduced disease intensity under unprotected conditions and relatively good yield alone couldn't always determine the success of F₁ hybrids, of course both are absolutely important. Apart from them, success of the development of disease resistant F₁ hybrids also depends on two important parameters. Number one is the average fruit weight or size which decides the marketability of the produce. The second important parameter is the number of hybrid seeds that is produced in one fruit by crossing, which actually decides the economic feasibility of hybrid seed production which will in turn bring down the cost of hybrid seed production. In the summer season (season II) even though biometrical observations were not taken up, it as observed that crosses involving LE 415 and H 24 as female parent showed significant fruit set and higher percentage of field establishment which is an added advantage though not a prerequisite to consider in selecting disease resistant hybrids. Hence eight hybrids were short listed for further evaluation based on their performance in both the seasons *viz.* LCR 1 x H 24, LCR 1 x CLN 2123A, LCR 1 x LE 415, H 24 x LCR 1, H 24 x LCR 9, H 24 x CLN 2123A, LE 415 x LCR 1 and CLN 2123A x LCR 9. Even though about 20 - 30 per cent of their population got infected with these viruses, they tend to give around 2.2 to 3.2 kg per plant without any pesticide spray under field conditions.

5.5. Combining ability variance and Graphical analysis

Variances due to GCA and SCA were significant for all the 18 characters studied (both the seasons), indicating the importance of both additive and non additive gene action in the inheritance of these characters. In graphical analysis, the t^2 value was significant for four characters, two in season I (number of branches per plant and Tv incidence at 75 DAT) and another two in the second season (TLCV and total disease incidence at 75 DAT). The deviation of W_r , V_r regression from unity was significant for four out of 15 characters studied in the first season *viz.*, number of branches per plant, TSS, Tv and Total disease incidence at 75 DAT whereas in the second season, TLCV and total disease incidence at 75 DAT exhibited significant deviation from unity indicating the failure of hypothesis for these characters. One of the major causes of such failure might be the role epistatic gene actions since this graphical analysis was primarily intended for additive –dominance model involving independently distributed genes. Also discrepancies in (W_r-V_r) values for parents for these characters further indicated the possibility of epistatic gene action.

Since gene action for the characters which failed in both t^2 and regression coefficient tests, gene action could be obtained only through combining ability analysis and interpreted.

For plant height, GCA variance was more than that of SCA variance indicating the preponderance of additive gene action. This was proved in W_r , V_r graph that partial dominance was in existence. The parent H 24 fell far away from the origin suggesting the involvement of recessive genes in the parent. The component of variance analysis indicated that the expression of plant height was conditioned by additive as well as dominant gene action since D, H_1 and H_2 components were significant. The correlation between $(W_r + V_r)$ and Y_r was negative indicating that dominance was operated in positive direction. Since almost both the additive and dominance nature were operating, heritability was only moderate (56.40 per cent). Moderate heritability for plant height was also reported by Jawaharlal (1994).

Regarding number of branches per plant, GCA / SCA showed the preponderance of additive factors though both additive and non additive gene action were important for the trait

thus suggesting the possibility of its favourable expression in F_1 hybrids. This is in accordance with the results of Sankari (2000) and Sekar (2001a).

Days to 50 per cent flowering was found to be controlled by additive factors as evident from GCA / SCA ratio. This was further additionally supported by the W_r , V_r graph where the regression line cuts Y- axis well above the origin. Still further evidence from fact that the 'D' component was highly significant along with one of the dominance components *viz.* H_1 supports the above inference. Additive nature for this character was also reported by Ponnuswamy and Muthukrishnan (1979) and Banerjee and Kalloo (1989).

GCA / SCA ratio for the trait number of fruits per plant indicated the prominent role of additive gene action suggesting the possibility of effective simple selection for this traits in the later generations so as to fix up the trait. In the graphical analysis, it was found that overdominance was operating as the regression line passed below the origin, cutting the V_r axis, which was also confirmed by average degree of dominance (>1). Hayman (1954) stated that partial dominance along with non allelic interaction might result in overdominant expression. This was additionally proved from the ratios of $H_2/4H_1$ (<0.25), K_D/K_R (>1) and positive F value. Additive gene action for number of fruits per plant was also reported by Sundaram (1986), Aruna (1992) and Premalakshmi (2001).

For the trait fruit weight, the GCA variance was significantly greater in magnitude than that of the SCA indicating the preponderance of additive effects in the expression of this trait. This was confirmed by W_r , V_r graph and average degree of dominance (<1). The heritability in narrow sense very high (83.43 per cent) and the additive genetic variance 'D' component was also significant. All these additionally prove the importance of role of additive gene action in controlling this trait. The parents LCR 3 and LCR 9 possessed more dominant alleles since the array points of these two genotypes were located nearer to the origin while LCR 1 seemed to have recessive genes since its array point fell farther from the origin. The array point of LCR 1 a genotype with the largest fruit size/weight exhibiting the more number of recessive genes further confirms that small fruit size is dominant over large fruit size in tomato. The additive gene action

for this trait was also observed by Singh *et al.* (1988), Kumar *et al.* (1997) and Wang Lei *et al.* (1998).

Combining ability variance suggested that yield was controlled mainly by dominance factors. The W_r , V_r graph depicted the involvement of overdominance which is the cumulative support and complementation between the alleles in the F_1 , contributed by both the male and female parents is observed. The genetic ratio (K_D/K_R) suggested that there was an excess of dominance genes. Moreover heritability was too low (17.85 per cent). It should be noted that in addition F , H_1 and H_2 components of variation, D component also showed significant difference, suggesting the involvement of both the gene actions. This is in corroboration with the findings of Baroncelli *et al.* (1972), Jamwal *et al.* (1984), Rajjadhav and Kale (1985) and Rai *et al.* (1997).

Since the regression coefficient from unity was significant for the character TSS, the trait might be controlled by epistatic gene action. GCA / SCA variance also supported the involvement of non additive gene action for this trait i.e. High TSS is dominant to low TSS controlled by non allelic gene interaction. This finding is in accordance with that of Williams and Gilbert (1964), Stoner and Thompson (1966), Chaudhary (1970), Kalloo *et al.* (1974), Singh *et al.* (1979), Gibrel (1983) and Kurian (1990).

Coming to other quality trait acidity, the ratio of GCA to SCA variance revealed the preponderance of additive gene action. Graphical analysis too revealed that same since the regression line passed well above the origin which was also confirmed by average degree of dominance (<1). Heritability (79.63 per cent), also reiterated the involvement of additive gene action. Although the K_D/K_R value was more than unity, F , H_1 , H_2 and h^2 components were non significant. The involvement of additive gene action for this trait was also reported by Singh and Nandpuri (1975), Gibrel (1983) and Kurian (1990). Medium to high heritability for titrable acidity was also reported by Walkof and Hyde (1963), Stoner and Thompson (1966), Lower and Thompson (1967) and Jawaharlal (1994).

Non additive gene action was observed to be controlling the ascorbic acid content in the fruit as the GCA /SCA was less than unity. It was further confirmed from the significance of H_1 component, the ratio of K_D to K_R (>1) and very low heritability (12.03 per cent). Possibility of overdominance was also evident from the average degree of dominance (1.9634). The correlation between Y_r and (W_r+V_r) was in negative indicating that dominance was operating in positive direction. Existence of such non additive gene action for this trait was also brought out by Chaudhary (1970) and Kalloo *et al* (1974).

Lycopene was found to be under the control of additive factor though both additive and non additive gene actions are important for the trait as evident from the ratio of GCA to SCA variance. Graphical analysis showed that involvement of overdominance in controlling this trait. As the components of variation D , F , H_1 , H_2 were found to be significant, it indicates the role of both additive and dominant gene action. K_D/K_R revealed the involvement of excess of dominant genes. The parents LCR 9 and LE 415 had more dominant genes as they were located nearer to the origin. Role of dominant gene action for this trait was reported by previous workers also (Bhutani and Kalloo, 1983 and Premalakshmi, 2001).

Combining ability variance revealed that total and OD phenol contents in the leaves were controlled by additive genetic factors although both additive and non additive gene actions were important for these traits. Hayman's analysis indicated the presence of overdominance which was evident from graph as well as average degree of dominance for both the characters. The ratios of dominant and recessive genes in the parent indicated that there was an excess of dominant genes involved in this trait. The components of variation H_1 and H_2 were significant for the character total phenol content whereas D , F , H_1 and H_2 were significant for the trait OD phenol indicating the role of dominant factors in the first trait and both additive and dominant factors for the second trait. This result is in line with the findings of Sankari (2000) in the leaves of tomato for TLCV incidence and Indu Rani (2002) in the roots of tomato subjected to root knot nematode infection. H 24 and LCR 9 seemed to have more dominant genes as their array points fell nearer to the origin.

The main objective in the present programme is the development of F₁ hybrids resistant to both TLCV and Tv. So the breeding procedures are to be focused and carefully formulated in increasing the potentiality of this complex trait namely resistance to viral diseases without sacrificing yield. TLCV incidence at 75 DAT was found to be controlled by additive genetic factors in both the seasons as evident from the ratio of GCA to SCA variance (>1). In the second season, assumption of Hayman's diallel analysis failed indicating the role of environmental interaction. In the first season overdominance was evident both from W_r, V_r graph and average degree of dominance. Assymetrical proportion of positive and negative alleles ($H_2/4H_1 < 0.25$), significant positive F value, and more than unity value of K_D/K_R might have caused the inflation of partial dominance into apparent overdominance. CLN 2123A was the only parent with more of dominant genes as its array point fell nearer to the origin while others located farther from the origin.

GCA/ SCA variances suggested the Tv incidence at 75 DAT was controlled by additive factors in the season I whereas by dominant factors in the season II. Such differential gene action might be due to be role of modifiers which react to environmental influence particularly temperature, existence of different distinct symptoms and unstable nature of virus (Watterson, *et al.* 1993). Modifier genes won't themselves directly involve in the interaction with virus but might modulate antiviral activity of one or more major resistant genes. The role of modifiers in the expression of resistance to many viral diseases has been reported by several workers (Martin 1970, Arumugam, 1977, Swiezynski *et al.*, 1981 and Watterson *et al.*, 1989).

Graphical analysis in the second season indicated the existence of overdominance. The components H_1 , H_2 , h^2 were significant and positive indicating the role of dominant genetic factors reducing Tv incidence at 75 DAT, which was also evident from the ratio of K_D to K_R (>1) and low heritability (10.70 per cent). Involvement of dominant factors for Tv resistance was also reported by Juliatta and Maluf (1995).

5.6. Evaluation of selected hybrids in season III (July –October 2002)

Based on the results from previous seasons (season I and II) eight hybrids were selected and tested along with their parents and two hybrids released for commercial cultivation, one COTH 1 of TNAU susceptible to both the diseases, another Ankush, claimed to be resistant to TLCV by a corporate sector company. There existed significant differences among the genotypes (test hybrids, parents and commercial hybrids) for all the characters studied in the season III indicating the scope for further selection among the selected hybrids for commercialization.

In this season, additionally a few more quality traits were observed *viz.* pH of the fruit juice, total and reducing sugars, sugar to acid ratio and pectin with the view to find out their suitability for processing too. One additional operation *i.e.* staking of plants with bamboo poles was done. Comparison of mean performance of selected hybrids in both the seasons (season I and III) have indicated that almost all the biometrical characters exhibited no clear cut differences between both the seasons except TSS and ascorbic acid. Similar observation was also made in parents. In the third season, mean of the selected hybrids was significantly different from that of season I for a few traits *viz.* plant height, number of fruits and yield per plant. But their mean values were far more superior to that of parents in terms of reduced incidence of TLCV, Tv and total disease incidence.

Plant height was significantly high in H 24 x CLN 2123A and H 24 x LCR 9 both in terms of *per se* and standard heterosis. The hybrids CLN 2123A x LCR 9 and H 24 x CLN 2123A, which produced high heterotic expression for this character (100.40 and 104.00 cm respectively) in the season I were not so efficient in the season III. This indicated that these heterozygous populations (F_1 hybrids) expressed superiority phenotypically under unfavourable environmental conditions of season I. However valid conclusion can be drawn only after testing them in different locations and over years in different seasons. Even though staking was done, no significant improvement in plant height was noticed in this season as compared to season I. This might be due to almost sturdy nature of hybrids tested.

Branches per plant were also significantly high in both the above hybrids indicating their general vigour and their potentiality to produce more number of flowers and fruit. Earliest flowering (28 DAT) was noticed in H 24 x CLN 2123A which was on par with best check hybrid Ankush. The same combination also exhibited similar trend in first season too. This hybrid would have been able to store and divert food materials earlier than other hybrids and parents for reproductive phase thereby aid in ripening of greater proportion of fruit at an early phase.

In the case of fruit characteristics, H 24 x CLN 2123A recorded the highest number of fruits (73.33) which is 62.70 per cent higher than the next best hybrid H 24 x LCR 9. It also recorded significant heterosis over both the best parent and the commercial hybrids. This further indicates that small fruited varieties proved to be the best combiners for number of fruits per plant. This is line with the findings of Sankari (2000). Fruit weight is one of the deciding criteria for the popularity of the hybrids. According to Tewari (1997) any fruit weight of more than 50 gram would suit for fresh market as well as for processing industries. In this regard, all hybrids except H 24 x CLN 2123A satisfied the above criteria. Of them, LCR 1 x H 24, LCR 1 x CLN 2123A and H 24 x LCR 1 surpassed the fruit weight of the best commercial hybrid Ankush.

In the plant architecture of H 24 x CLN 2123A, it is quite clear that source is limited to supply required photoassimilates to the large number of developing fruits. An elaborate discussion on this aspect will be made later on. Yield per plant in H 24 x CLN 2123A was higher over the best parent LCR 1 and best check hybrid Ankush. Even though H 24 x CLN 2123A exhibited lowest fruit weight, its highest fruit number contributed to the phenomenal increase in yield per plant.

All the hybrids met out the minimal requirement of pH (<4.5) for processing purpose. Out of eight hybrids, only H 24 x LCR 9 alone recorded pH on par with the best parent CLN 2123A while this hybrid alone recorded significant negative standard heterosis over the best commercial hybrid Ankush. Even though the hybrid H 24 x LCR 9 recorded lowest pH among the selected hybrids, its acidity value was only medium (0.50 per cent). Stevens *et al.* (1977) reported that the relationship between pH and titrable acidity in tomatoes is complex. The low

negative correlation in this hybrid might be due to differential buffering caused primarily by phosphate content of the fruit (Stevens, 1972). The hybrid H 24 x CLN 2123A recorded the highest acidity value of 0.58 per cent among the selected hybrids which was on par with that of the standard commercial hybrid Ankush but it could not surpass one of its parents CLN 2123A which had the highest acidity of 0.60 per cent. This hybrid probably due to its small sized fruits with less pericarp resulted in higher value of acidity. Negative correlation between fruit size and acidity was also reported by Kalloo (1986). The hybrid LCR 1 x LE 415 resulted in lowest acidity of 0.45 per cent which can find its use as salad.

In the processing point of view, TSS content of the fruits forms the major criterion. In the season III, average performance of both the hybrids and parents for this trait was significantly higher than that of both hybrids and parents in the season I. Low sunshine hours per day and comparatively lower temperature prevailing in the season III, could have resulted in more diversion of photoassimilates to the growing sink (fruits) without dissipating much energy towards respiration. This observation was in contrast with the reports of Saimbhi (1970) and Sankari (2000). Despite having highest acidity, the parent CLN 2123A also exhibited highest TSS content (6.04° Brix), which is the rare blend of negatively correlated characters. This was well reflected in the hybrids possessing this genotype as one of the parents *viz.* CLN 2123A x LCR 9 (5.62), LCR 1 x CLN 2123A (5.57) and H 24 x CLN 2123A (5.47) which exhibited high TSS. All the above hybrids exhibited significant positive heterosis over best check hybrid COTH 1. Except LE 415 x LCR 1, all other hybrids will be suited for processing as their TSS value was more than 4.5° Brix. The hybrid CLN 2123A x LCR 9 is to be considered particularly for tomato juice because of its significant highest TSS.

Higher ascorbic acid content in the first season might be due to relatively higher daily sunshine hours during fruit maturity period resulting in increased synthesis of precursors responsible for ascorbic acid synthesis. This finding is line with that of Kohman and Porter (1940) and Hamner *et al.* (1942). Best parent heterosis for the trait ascorbic acid in the fruits was noticed only in two hybrids *viz.* CLN 2123A x LCR 9 and LCR 1 x H 24. The same hybrids

exhibited significant positive heterosis over the commercial hybrids *viz.* COTH 1 and Ankush. For lycopene, heterosis over the best parent (H 24) was noticed only in one hybrid H 24 x LCR 1. But its reciprocal cross exhibited the lowest lycopene value which was also evident in season I indicating the role of maternal effects and /or cytoplasmic-genic interaction.

Sweetness in tomato is largely determined by sugar content. They make an important constituent of flavour in tomato. In the present study reducing sugars contribute around 65 to 85 per cent of total sugars. None of the selected hybrids exhibited heterosis over the commercial hybrid COTH 1 both for the total and reducing sugars.

In the present study, H 24 x LCR 9 and LCR 1 x LE 415 recorded highest total sugars as well as reducing sugars suggesting positive association among these two traits. Sugar to acid ratio is the key factor deciding the flavour quality of tomato fruits. Cultivars with a large locular portion with high concentrations of acids and sugars have better flavour than those with a small locular portion. In the present study the hybrids H 24 x CLN 2123A and H 24 x LCR 9 possessed high sugars and acids, but H 24 x CLN 2123A had only small locular area since the fruit size was small. Hence H 24 x LCR 9 may be suggested as the best fit for above discussed criterion.

Another important criterion which decides the popularity of F₁ hybrids is the firmness at all stages of fruit ripening. Consistency of products from tomato is closely related to the concentration of cellulosic and pectic compounds (Stevens, 1978). There existed wider variation among the hybrids for this character. Pectin content was highest in H 24 x LCR 9 (0.33 per cent) while it was lowest in H 24 x CLN 2123A (0.09 per cent). Even though the parent CLN 2123A the exhibited highest pectin content of 0.45 per cent it is unable to transmit this character to its offspring.

Total phenol content in the leaves of all the selects hybrids was higher than that of commercial hybrids *viz.*, COTH 1 and Ankush, the latter being claimed as resistant to TLCV. Ortho Dihydroxy phenol content was also higher in all the hybrids than the best check hybrid Ankush except in one (LCR 1 x CLN 2123A). It is to be noted that there was no significant

differences between mean values of parents and hybrids for these two characters in both the seasons studied. None of the hybrids exhibited significant positive heterosis over the best parent.

Considering the most important aspect, i.e. disease incidence, selected hybrids exhibited significantly lower PDI values for Tv and total disease incidence at 75 DAT as compared to that of their parents, two commercial hybrids *viz.* COTH 1 and Ankush and susceptible check variety (CO 3). The incidence of TLCV was less during the crop season as it was visualized from the lower PDI values at 75 DAT in susceptible check variety (14.67 per cent) and susceptible check hybrid (8.33 per cent). All the tested hybrids recorded Highly Resistant reaction for TLCV at 75 DAT. Lower incidence of Tv was noticed in H 24 x LCR 1 (10.60 per cent), LCR 1 x H 24 (10.80 per cent) and LCR 1 x CLN 2123A (14.35 per cent). But all the tested hybrids recorded PDI of below 20 for Tv incidence while the susceptible check variety recorded 41.85 per cent incidence. The corporate sector hybrid Ankush was more susceptible (39.25 per cent) than COTH 1 (25.00 per cent) developed and already released by TNAU.

Since, Tv was the major havoc in this season, total PDI also reflected its major share. The hybrids with least incidence of both the diseases were LCR 1 x H 24, H 24 x LCR 1, H 24 x CLN 2123A, LE 415 x LCR 1 and LCR 1 x CLN 2123A.

Based on their relative performance, taking in to consideration of all the three seasons, the hybrids H 24 x LCR 1, H 24 x CLN 2123A, LCR 1 x CLN 2123A, LCR 1 x H 24 and LE 415 x LCR 1 recorded lower mean total PDI of 19.13, 22.13, 23.26, 24.21 and 24.47 per cent respectively, while the susceptible check recorded 70 per cent infection. All the selected hybrids exhibited below 28.00 per cent incidence. Yield is also equally a most important criterion especially when the virus inoculum is high. Hence the hybrids H 24 x CLN 2123A, LCR 1 x LE 415, H 24 x LCR 9 and LE 415 x LCR 1 can fit well as they produced higher yield of 3.16, 2.68, 2.67 and 2.65 kg per plant respectively (average of both season I and III). All the eight hybrids recorded more than 2.30 kg per plant under unprotected conditions while the susceptible check yielded just 0.45 kg per plant. Since the main objective the study is to develop F₁ hybrids with combined resistance to both the diseases along with high yield, the combinations H 24 x LCR 1

and

H 24 x CLN 2123A are the best ones to pursue further.

5.7. Correlation studies

Knowledge about association of various characters helps in selection of genotypes and also suggests the advantage of a selection method for more than one character at a time, which could be explained that improvement of one character results in the simultaneous improvement of all other positively related characters. Since yield is a complex character, direct selection could not be possible. All changes in yield must be accompanied by changes in one or more component characters. This is indicated by varying degree of positive and negative correlation between yield and its components and among themselves. Non existence of negative correlation between two most important traits, if any, identified will be useful for the plant breeder to go for the selection of genotypes / hybrid / hybrid derivatives in the segregating population for high yield coupled with quality factors. In TLCV and Tv diseases resistance breeding programmes, a knowledge of association between the incidence of these diseases and other characters is a must particularly when the breeder wishes to keep a balance between disease resistance and agronomic traits.

Genotypic correlation coefficient provides a measure of a genotypic association between characters eliminating the environmental influence. Genotypic correlation may be due to pleiotropic action of genes or due to linkage or may be due to both. Information on association of characters in season I and III are discussed hereunder.

The nature of association between disease incidence (TLCV/Tv/Total) and yield was in negative direction in both the seasons. The decrease in fruit yield might be due to the decrease in vegetative growth, degeneration of photosynthetic pigments *viz.*, chlorophyll 'a' and 'b' and altered physiology. This is in confirmation with the findings Diener (1963), Sudha (1996) and Mala and Vadivel (1999). But in the first season, the association was non significant. It implies that even though the disease incidence was higher in season I than the season III, some other

characters might have been acting favourable to the increase in yield in the first season. Among the diseases, in the first season, both TLCV and Tv incidence had a negative correlation. It may be due to the competition between these viruses for capturing the infection sites in the plants under test due to comparatively favourable environment for both the viruses compared to season III. In season III, their association is non significant and positive indicating the less competition between them due to the less favourable environment for both of them with respect to their spread by vector.

Both plant height and number of branches per plant had positive correlation with yield in both the seasons. Taller plants produce more number of secondary branches and the resultant increases in plant canopy might have contributed to higher yield. Patil and Bojappa (1993) have observed similar positive association of these characters with yield. Plant height had significant negative association with TLCV incidence in both the seasons whereas with Tv, it had negative association in the season III and non significant positive association in the season I. Since virus infection naturally causes considerable reduction in photosynthetic pigment which normally leads to the reduction in the synthesis of carbohydrates and thereby caused reduction in plant height. According to Sudha (1996), the virus infection caused blockage in the phloem cells hence affected the translocation of sugars resulting in the accumulation of carbohydrates, thereby arresting the apical growth. The above discussions explain the results obtained from both the seasons in the present experiment.

In the present study, earliness is positively correlated with yield although non significant. Early genotypes had a greater period of reproductive phase hence resulted in a relatively higher yield. This is in line with the findings of Aruna and Veeragavathatham (1997). It is also clear that earliness was significantly and positively correlated with fruit number in both the seasons which ultimately resulted in higher yield, while it showed negative correlation with fruit weight suggesting more probable chance of early varieties to yield small fruits. This is line with the reports of Kalloo (1988) and Mohanty (2003). Earliness had negative correlation with TLCV

disease incidence in both the seasons thus resistant genotypes may likely to flower and fruit much earlier before they get infected severely.

Number of fruits per plant was positively and significantly correlated with yield in both the seasons. This was in corroboration with the findings of Mageswari *et al.* (1997) and Das *et al.* (1998). Fruit weight also had significant positive association with yield in both the seasons. Existence of positive association of fruit weight with yield was also reported by earlier workers (Ghosh and Syamal, 1994 and Yadav and Singh, 1998). Of all the characters studied, only the above two characters exhibited highest significant positive correlation with yield in both the seasons. This suggests that a balance in these traits could be made in the selection programme to increase the yield since both these traits are significantly and negatively correlated with each other. This might be attributed to the limited physiological capacity of the plant to provide raw material needed for high quality coupled with high yield which in most cases necessitated greater accumulation of dry matter. This finding is in accordance with that of Stevens (1979), Tikoo (1981), Shoba (1990) and Verma *et al.* (1997). It is quite interesting to note that the correlation (genotypic) between fruit weight and disease incidence (TLCV/Tv/Total) was found to be non significant irrespective of the fact whether the value is positive or negative and it was obvious in both the seasons. This is a welcome phenomenon because the breeder is at liberty to choose genotypes with better fruit weight still having a high level of resistance to such viral diseases.

In the present investigation pH had highly significant negative association with acidity. This has been earlier reported by Stevens (1972) and Abani and Uzo (1984). Lycopene content of the fruit did not exhibit significant correlation with TLCV incidence in both the season. This suggests that at least there is a possibility of developing resistant hybrid with dark red fruit colour. However negative association between TLCV incidence and lycopene content in fruits was reported by Sankari (2000).

Positive association was observed between total sugars and fruit yield in the present study. It indicates the hybrids and parents used in the present study had better photosynthetic, partitioning and translocation efficiency thereby accumulating more dry matter into the

developing fruits. With regard to disease incidence, the total sugar had significant positive genotypic correlation with the incidence of Tv which is also for total disease incidence. However the correlation was not significant with respect to TLCV. Pectin had negative association with fruit yield indicating that fruit yield and pectin content can't be improved simultaneously using the genotypes under study.

Negative and significant association of phenolic content with TLCV, Tv and total disease was noticed in the season I while non significant negative association was observed in season III. This indicates phenolic compounds in the tissue might have inhibited virus multiplication process and inactivated virus. It is also evident that they had a greater role in more conducive periods of disease incidence (season I). Supporting evidence could be obtained from the results of Narayana and Reddy (1980) and Singh and Abidi (1988) for this aspect.

5.8. Path coefficient analysis

When different independent variables influence the particular dependent variable, certain amount of interdependence arises between independent variables. In such situation, it becomes necessary to estimate the degree of direct effect of the independent variable and the indirect effect exerted through other characters. When such complex situation occurs, the correlation alone is insufficient to explain the true association for an effective exploitation of characters. In such situations, path coefficient analysis helps to measure the importance of causal factors involved and permits critical examination of specific forces acting to produce a given correlation both directly and indirectly.

The unexplained variation in genotypic path was 0.132 in season I and 0.307 in season III. It predicted the 86.8 and 69.3 per cent variation in yield at genotypic level in the season I and III respectively. It further indicates that the occurrence of some more factors, not considered here, contributed to fruit yield of tomato. Only in the trait plant height (season III), the correlation coefficient and the direct effect imparted by it with yield were almost equal. This explains the true relationship and a direct selection through this trait will be effective.

In the present investigation, number of fruits per plant followed by average fruit weight had the highest contribution of direct effects on yield per plant in both the seasons. Both characters also had significant positive correlation with yield. Hence direct selection through these characters will also be effective. The highest direct positive effect of fruit weight and number of fruits per plant on yield per plant has also been reported Hazarika and Das (1998), Sankari (2000) and Indu Rani (2002).

Tv incidence at both the season had positive direct effect, but negatively correlated with yield. Its negative indirect effect through reduced fruit weight and total sugars might have brought a cumulative effect in lowering down the yield but not through any one single trait.

With regard to quality characters, sugar to acid ratio exhibited positive direct effect on yield. Its association with yield was also positive. Hence direct selection through this trait would be effective. TSS and Ascorbic acid had both negative direct effect and negative correlation with yield, which indicates that these traits couldn't be improved simultaneously along with.

5.9. Evaluation of best performed hybrids during December 2002-March 2003

Two hybrids which had exhibited least incidence of both the diseases *viz.*, H 24 x LCR 1 and H 24 x CLN 2123A along with their parents and susceptible check variety CO 3 were transplanted during mid December 2002 which was reported as the conducive period for maximum thrips activity as per the results of experiments conducted. They were given special package of practices in order to obtain maximum potential yield under a system of cultivation without the use of inorganic pesticides. Tospovirus incidence was drastically increased upto 60 DAT and then maintained as such. Astonishingly at 75 DAT, both hybrids H 24 x LCR 1 and H 24 x CLN 2123A recorded high total PDI (45.71 and 37.50 per cent respectively). But at the same time the susceptible check variety (CO 3) exhibited 98.15 per cent infection. One aspect which is very clear in all the previous seasons was confirmed in this season i.e. H 24 x CLN 2123A has got the highest level of resistance to TLCV (< 5.00 per cent) in all the four seasons

studied, whereas the other combination H 24 x LCR 1 exhibits incidence of both the diseases equally.

An interesting observation noted was that even though around 40 per cent plants of these hybrids were found infected, their fruit yield was almost comparable with that of the previous seasons. Definitely special packages of practices like *Adhathoda* leaf extract, panchakavya spray, biofertilizer application etc. might have played a role in sustaining the yield in the present study but their exact effect could not be ascertained from the present study since these packages were uniformly given to all the tested genotypes. All the parents yielded around 1.30 kg per plant with virus incidence of around 60 per cent in H 24 and CLN 2123A and 88.88 per cent in LCR 1. There is a special need to mention about the parent CLN 2123A here, even though 60.23 per cent plants were infected with Tv, none of the plants were infected by TLCV. So it proved to be a highly resistant source for single virus resistance breeding programme. It is well visualized that 'tolerance' mechanism was operating in the above two hybrids i.e. these partially diseased hybrids were able to yield an acceptable or expected quantity or quality of fruits. This indicates the worthiness of tolerant hybrids in sustaining the yield in the highly problematic areas *viz.* virus prone fields. Of course, they may act as a potential reservoir of virus inoculum, nevertheless curtailing the multiplication of the virus probably by the specific defense mechanism operating in them, help to maintain the yield at the present time when no highly resistant immune cultivars are available. Previous literature and the present study also indicated the non availability of immune / resistant hybrids for both the viral diseases. Tolerant nature of genotypes infected with viral diseases was also reported by Shoba (1990), Boiteux *et al.* (1999) and Sankari (2000).

5.10. Physical manipulation of source-sink relationship

As discussed earlier, although H 24 x CLN 2123A had satisfied two major selection criteria *viz.*, lower disease incidence and high yield apart from early flowering, high acidity etc. Its lower fruit weight (~42g) appears to hinder its use as commercial hybrid. Tewari (1999) pointed out that any hybrid / variety should have fruit weight of more than 50 g for fresh market

as well as for processing purposes. Here one thing is clear that source is limited to supply the required photoassimilates to the large number of developing fruits (sink) competing with each other ultimately reflecting on fruit size. It was assumed that any success in promoting transport of assimilates to the required level by restricting the abnormal number of sinks (developing fruits) can help to maintain the fruit size to the required level *viz.* level of market acceptability. Hence various physical manipulation methods *viz.*, nipping, flower thinning, truss thinning and alternate truss thinning were attempted.

Plant height remained unaltered in the remaining treatments except nipping treatment. Truss thinning and alternate truss thinning had resulted in the lowest number of fruits owing to the maintenance of only six clusters. Hence they led to the desirable fruit weight of 57.23 and 54.62 g respectively which clearly indicated that source is limited for the supply of the required photosynthates to the large number of developing fruits in the above hybrid. Once the sink load is reduced, automatically it resulted in increased fruit size. Bangerth and Ho (1984) explained that an early – induced fruit may compete better than a later induced one because of its bigger size or it might have suppressed the growth of later induced fruit by producing inhibitors. Early induced fruits were retained in truss thinning treatment hence resulted in big sized fruits than that of alternate truss thinning. Flower thinning (retaining 2 basal flowers / truss) had also resulted in significant improvement in fruit size. The explanation of Bangerth and Ho (1984) here also suits well. Moreover it was reported that proximal fruits contain more IAA but less ABA than the distal fruit throughout the growth (Ho *et al.*, 1983) and thus becoming higher potential sink for importing assimilates. These two reasons may explain the considerable improvement in fruit size (31.67% over control) in flower thinning treatment achieved in the present investigation. The treatment nipping recorded only a marginal increase in fruit weight. This might be partly due to the removal of current photosynthetic apparatus. Ho (1976) reported that the labile assimilate imported by a fruit at any time is a mixture of assimilates currently fixed by the leaves and that remobilized from the reserve in the leaves.

Coming to the another important trait yield per plant, the main interest lies with significant improvement in fruit size above 50 g without sacrificing the total yield much. Nipping treatment exhibited only moderate yield (2.27 kg) per plant. It may be due to only a slight increase in fruit weight (8.2 per cent) and drastic reduction in fruit number owing to the drastic removal of vegetative as well as reproductive sinks. The treatments 'truss thinning' and 'alternate truss thinning' recorded the lowest yield of 1.92 kg per plant possibly due to over elimination of fruiting cluster. Even though fruit weight had increased considerably (~ 40 per cent) in those treatments, that could not replace the reduction in yield because of the fact that number of fruits has been reduced considerably. So in forth coming experiments, it may be worth while to keep three or four additional clusters to sustain the yield or otherwise reduce the intra row space between the plants so as to keep more number of plants thereby compensating the yield loss.

On an average, the hybrid produces 24 flower clusters. Flower thinning leaving only two basal fruits had 37.82 per cent reduction in fruit number (i.e. 47.62) with 31.67 per cent increase in average fruit weight (53.55 g) resulted in contributing nearly 82 per cent of control yield (2.55 vs 3.11 kg). This indicates the potential of stronger strength (i.e basal fruits in a truss) and more efficient partitioning of assimilates to the fruits left indicated by the increased fruit weight.

Polar and equatorial diameter and pericarp (both inner and outer wall) thickness were observed for explaining the way in which increased fruit weight arose. It was well evident by the fact that the control exhibited the lowest values of all these four characters and thereby might have resulted in less weighted fruit. Polar and equatorial diameters were highest in truss thinning treatment possibly explaining the maximum fruit weight in that treatment. Thickness of both inner (septa) and outer walls of pericarp significantly high in all the treatments compared to control. But mere increase in this thickness may not increase the fruit weight as observed from the present study that even though truss thinning treatment had highest fruit weight, it had only moderate improvement in thickness of pericarp. Hence there would be some more factors contributing to the increased fruit weight.

TSS was found to be higher in control suggesting the negative correlation between fruit size and TSS. Ram (1998) reported that tomato fruits grow by cell expansion and not by increase in number of cells. Cell expansion is accompanied by the increase in water content leading to dilution of TSS. Even though treatments with highest yield showed lower TSS values, it at any cost wouldn't affect their processing capabilities as the values were more than the basic requirement of 4.5°Brix for processing. Titrable acidity was found to be negatively associated with fruit size.

From the over all observation, it can be concluded that flower thinning retaining two flowers per cluster can be recommended as it resulted in significant improvement in fruit weight *viz.* 53.55 g against the original fruit weight of 40.60 g in this hybrid with a potential yield of 2.55 kg per plant under pesticide free cultivation. Of course it needs further fine tuning of cultural techniques for further possible improvement of fruit size if any.

5.11. Biochemical basis of resistance

Certain oxidative enzymes of host pathogen interaction defend the host from being diseased (Agrios, 1978). Many enzymes occur frequently in many isoforms and are involved in synthesis of defense substances. Hence biochemical defense mechanism would certainly be helpful in the selection of plant for identifying source of resistance. With this aim, the activity of enzymes related to the induction of disease resistance *viz.* PO, PPO and PAL was measured in two selected hybrids H 24 x LCR 1 and H 24 x CLN 2123A and their parents in comparison with susceptible check variety CO 3. By comparing the results with other selected genotypes, it can help in understanding the resistant mechanisms towards these viral diseases.

Generally, the increase in PO and PPO activity was rapid up to 96 hours after inoculation. Among the genotypes, the hybrids H 24 x CLN 2123A and H 24 x LCR 1 recorded maximum peroxidase activity at 96 hours after challenge-inoculation with both the viruses, which was significantly higher than that of uninoculated control. Susceptible check recorded lowest values in either case. According to Kosuge (1969) and Shankar and Jindal (2001) activity of the

peroxidase and polyphenol oxidase enzymes is directly related to resistance in the host, which could be due to the conversion of the enzymes into quinones, which are toxic to pathogen. Moreover, these oxidative enzymes might have catalyzed the formation of lignin and other oxidative phenols that contribute to the formation of defense barriers for reinforcing the cell structure (Avdiushko *et al.*, 1993). Hence high induction of these defense related enzymes *viz.*, PO and PPO could have helped the hybrid to trigger the defense system thereby induce resistance. Such parallel increase in PO activities with the development of systematic resistance has been observed by Simon and Ross (1970, 1971). Rapid changes in the activities of these enzymes after the stress induction may indicate their possible role in the defense mechanisms.

Phenylalanine ammonia-lyase is reported to be involved in phytoalexin or phenolic compound synthesis. Such enzyme has been correlated with defense against pathogens in several plants (Bashan *et al.* 1985 and Beauodoin-Eagan and Thorpe, 1985). In the present study although both the hybrids investigated (H 24 x LCR 1 and H 24 x CLN 2123A) had significantly highest level of PAL activity before inoculation of viruses, rapid increase in PAL activity when both the viruses were simultaneously inoculated was noticed only in H 24 x LCR 1, while H 24 x CLN 2123A exhibited only a marginal increase in its activity. It may be due to the comparatively early induction of PAL in response to the virus inoculation in the former hybrid than the latter one. High PAL activity in the above hybrid might have produced precursors of phenolics (cinnamic acid) and lignin synthesis thereby aids in forming mechanical and chemical barrier against the invading pathogen (Goldwasser *et al.* 1999). Vasanthi (2001) reported that induction of PAL was high in PGPR (Plant Growth Promoting Rhizobacteria) -treatment. A high level of PAL was noticed by Chen *et al.* (2000) when induced in roots of cucumber plants inoculated with *Pythium aphanidermatum*.

In the present investigation, crude protein analysis of the plants challenge –inoculated simultaneously with both the viruses by SDS-PAGE expressed 19, 27 and 45 KDa proteins. Only in the hybrid H 24 x CLN 2123A, 27 KDa protein expression was prominent while in other genotypes the expression was not prominent upon challenge-inoculation with both the viruses

done simultaneously under greenhouse conditions. Another protein of 19 KDa was prominent in all the genotypes except the hybrid H 24 x LCR 1. Hence even though the banding patterns were similar, differences were found in band intensity. This may be used as a marker for identifying resistant and susceptible genotypes. Ganapathy (1985) reported a significant reduction in number of protein bands in TSWV infected groundnut plants compared to healthy plants. Ragupathi (1995) reported for the absence of 18 KDa protein in AVP (antiviral principle) treated tomato plants challenge - inoculated with TLCV but it was present in untreated TLCV-inoculated control. Vasanthi (2001) observed the expression of 18, 22, 42 and 55 KDa proteins in PGPR-treated plants, but in case of untreated control plants, the expression was not prominent upon challenge –inoculation with TLCV.

5.12. Isozyme analysis

Isozyme variation is used as a powerful tool to complement and to supplement conventional breeding methods (Yndgard and Hoskuldson, 1989). Additional bands or shifts in migration may arise from post translation modification of enzymes. In the present investigation, both PO and PPO isozyme patterns were studied in 3 stages *viz.* just before inoculation, 48 and 96 hours after inoculation. In the PO isozyme analysis, just before virus inoculation, a thick isoform (PO-3) was common in all the genotypes whereas two isoforms (PO-2 and 4) were more prominent in susceptible check. Out of five isoforms, only 4 were expressed by the hybrids. After 48 hrs of inoculation no major change in banding pattern was observed. Banding patterns were differentiated only at 96 hrs after graft inoculation. The hybrid H 24 x CLN 2123A exhibited a maximum of six isoforms whereas one of its parents *viz.*, CLN 2123A had only four isoforms.

From this it is clear that PO induction in resistant hybrid H 24 x CLN 2123A has taken place only after 4 days. The results of the present study of the isozyme profile revealed that

there was appearance of new bands and disappearance of some bands in F₁ hybrids when compared to their parents. The appearance of new bands in genetically reconstituted hybrids can be attributed to the expression of structured genes, which were not expressed previously, or expression of new genes contributed by female parents. The disappearance of some bands in hybrid when compared to their corresponding parents may be due to the negative regulation of gene action or suppression of previously acting genes reconstituted the hybrid genome. Sathiyamurthy (2002) observed an increase in number of bands while studying isozyme pattern in some pepper hybrids.

With respect to PPO, the susceptible check (CO 3) showed five isoforms while all other genotypes had only four isoforms just before inoculation. Similar to PO, clear cut band expression was visualized only at 96 hours after inoculation. The susceptible check exhibited thick isoforms (PO-1,2,3,4) while two clear cut isoforms were observed in the hybrid in H 24 x LCR 1 which was absent/faint in other genotypes.

From the critical analysis of the results, the following cross combinations could be identified as the best ones suitable for growing in areas highly infected with TLCV and a tospovirus with appreciable horticultural attributes under unprotected conditions. The hybrid H 24 x LCR 1 had mean total PDI of only 19.13 with 2.43 kg per plant. The cross combination H 24 x CLN 2123A had 22.13 per cent mean total PDI yielding 3.16 kg per plant. At the same time, the susceptible check (CO 3) exhibited 70 per cent infection and yielded just 0.45 kg per plant. The main advantage of these hybrids is the availability of fruits during the season when there is a severe outbreak of TLCV/Tv or both which normally leads to non availability of fruits in almost all other genotypes. **These hybrids are as good as other hybrids when there is no incidence. At the same time they have the potential to give reasonably good yield under heavy inoculum of viruses when the other commercial hybrids may totally fail. The added advantage would be there is no pesticide residue in the marketable produce.**

This virus tolerance nature of above said hybrids can be used in combination with other cultural practices *viz.*, nursery protection through screens, weed control, physical barriers against

vector movement, reflective mulches etc., in order to get further improvement in productivity especially during peak vector activities. These hybrids are worth test-verifying in different seasons as well as at different locations to confirm their suitability in the areas highly infected with above viruses. Their economic yield could still be increased by standardizing agrotechniques.

Table 83: Best parents with good *per se* and *gca* effects for selected characters

Characters	<i>Per se</i>	<i>gca</i>	<i>Per se and gca</i>
Fruit weight	LCR 1, LCR 9	LCR 1, LCR 9	LCR 1, LCR 9
Yield per plant	LCR 3, LCR 1	LCR 1, H 24	LCR 1
Total phenol	LE 415, H 24	H 24	H 24
OD phenol	LE 415	H 24, LE 415	LE 415
TLCV incidence ^b – Season I	LE 415, CLN 2123A, LCR 3, H 24	H 24, CLN 2123A	H 24, CLN 2123A
TLCV incidence ^b – Season II	LCR 9, CLN 2123A, LCR 1, LCR 3, H 24	CLN 2123A	CLN 2123A
Tv incidence ^b – Season I	LCR 1, LCR 9	LCR 9, LCR 1, LCR 3, H 24	LCR 1, LCR 9, H 24
Tv incidence ^b – Season II	LE 415, LCR 9, H 24	H 24	H 24
Total disease incidence ^{b,c} – Season I	LCR 3	-	-
Total disease incidence ^{b,c} – Season II	LCR 9, LE 415	-	-

^b – low values were considered as best ^c – not *gca* since mostly arithmetic mean of disease incidence

Table 84: Best hybrids with good *per se*, *sca* effects and heterosis^a for selected characters

Characters	<i>Per se</i> and heterosis	<i>Per se</i>, heterosis and <i>sca</i>
Fruit weight	CLN 2123A x LCR 9	CLN 2123A x LCR 9
Yield per plant	H 24 x LE 415, H 24 x CLN 2123A, H 24 x LCR 9, LE 415 x LCR 1	H 24 x LE 415, H 24 x CLN 2123A, H 24 x LCR 9
Total phenol	H 24 x CLN 2123A	H 24 x CLN 2123A
OD phenol	H 24 x LCR 1, LCR 1 x LCR 9	H 24 x LCR 1, LCR 1 x LCR 9
TLCV incidence ^b – Season I	H 24 x LCR 1, LCR 9 x CLN 2123A, LCR 1 x CLN 2123A, H 24 x CLN 2123A	H 24 x LCR 1, LCR 9 x CLN 2123A, H 24 x CLN 2123A,
TLCV incidence ^b – Season II	CLN 2123A x LCR 9, LCR 9 x H 24, LCR 9 x LE 415, LCR 1 x CLN 2123A, H 24 x CLN 2123A	CLN 2123A x LCR 9, LCR 9 x LE 415
Tv incidence ^b – Season I	LE 415 x LCR 3, LCR 9 x H 24, LCR 3 x LCR 1, CLN 2123A x LCR 3	LE 415 x LCR 3, LCR 9 x H 24, CLN 2123A x LCR 3
Tv incidence ^b – Season II	H 24 x CLN 2123A, H 24 x LCR 3, CLN 2123A x LCR 1	H 24 x CLN 2123A
Total disease incidence ^{b,c} – Season I	H 24 x LCR 1, LE 415 x LCR 3, LCR 9 x H 24, H 24 x LCR 9	-
Total disease incidence ^{b,c} – Season II	H 24 x CLN 2123A, LCR 1 x CLN 2123A, CLN 2123A x LCR 1, LCR 9 x H 24, CLN 2123A x LCR 9, H 24 x LCR 9, H 24 x LCR 1	-

^a – if not over diii, then dii/di

^b – low values were considered as best incidence

^c not *sca* since arithmetic mean of disease

Table 85: Best hybrids with mean, heterosis over best parent and gca^d of their parents for selected characters

Characters	Hybrids	Mean	diii (%)	gca^d of the parents
Fruit weight	CLN 2123A x LCR 9	73.00 g	-34.36**	Low x High
Yield per plant	H 24 x LE 415	3.25 kg	22.18**	High x Low
	H 24 x CLN 2123A	3.12 kg	17.11**	High x Medium (+)
	H 24 x LCR 9	2.85 kg	6.95**	High x Low
Total phenol	H 24 x CLN 2123A	307.60 mg/100g	0.56	High x Low
OD phenol	H 24 x LCR 1	247.35 mg/100g	-8.88**	High x Low
	LCR 1 x LCR 9	190.45 mg/100g	-29.84**	Low x Low
TLCV-incidence - season I ^b	H 24 x LCR 1	0.00 %	-	High x Medium (-)
	LCR 9 x CLN 2123A	0.00 %	-	Low x High
	H 24 x CLN 2123A	0.00 %	-	High x High
TLCV incidence - season II ^b	CLN 2123A x LCR 9	0.00 %	-	High x Medium (+)
	LCR 9 x LE 415	0.00 %	-	Medium (+) x Medium (-)
Tv incidence - season I ^b	LE 415 x LCR 3	5.50%	-38.95*	Medium (-) x High
	LCR 9 x H 24	9.14%	-20.76	High x Medium (-)
	CLN 2123A x	13.57%	-2.66	Low x High

	LCR 3			
Tv incidence - season II ^b	H 24 x CLN 2123A	14.65%	34.41	High x Low

Total disease incidence - season I ^{b,c}	H 24 x LCR 1	15.15%	-17.68	-
	LE 415 x LCR 3	18.54%	-8.37	-
	LCR 9 x H 24	20.90%	-2.26	-
	H 24 x LCR 9	21.76%	-0.07	-
Total disease incidence - season II ^{b,c}	H 24 x CLN 2123A	19.19%	- 17.81**	-
	LCR 1 x CLN 2123A	22.14%	- 11.20**	-
	CLN 2123A x LCR 1	22.96%	-9.43*	-
	LCR 9 x H 24	23.34%	-8.60*	-
	CLN 2123A x LCR 9	23.37%	-8.54*	-
	H 24 x LCR 9	26.93%	-1.11	-
	H 24 x LCR 1	27.38%	-0.19	-

^b – Low values were considered as best disease incidence ^c not *sca* since arithmetic mean of

^d – High and low represents those having significant values with positive and negative signs respectively whereas medium represents non significant values

CHAPTER VI

SUMMARY

Investigations were carried out to develop F₁ hybrids in tomato (*Lycopersicon esculentum* Mill.) with combined resistance to Tomato Leaf Curl Virus (TLCV) and the Tospovirus (Tv) prevailing in south India at the Department of Vegetable Crops, Horticultural College and Research Institute, Tamil Nadu Agricultural University, Coimbatore during 2000-2003.

After initial screening for respective virus resistance, the genotypes LCR 9, LCR 1, LCR 3, CLN 2123A and H 24 were chosen as preferred parents for TLCV resistance whereas LE 415 for Tv resistance. All these parents were crossed with each other in both direct and reciprocal fashion to get maximum number of cross combinations. The resultant 30 F₁ hybrids and their parents were evaluated in two seasons under unprotected conditions (without pesticide spray). In the season I (January –April 2002), 18 characters *viz.* plant height, number of branches per plant, days to 50 per cent flowering, number of fruits per plant, fruit weight, yield per plant, total soluble solids, acidity, ascorbic acid, lycopene, total phenol, ortho dihydroxy phenol and percentage of disease incidence (TLCV, Tv and Total) at 15, 30, 45, 60, 75 and 90 DAT were studied. In the season II (March –June 2002) only disease incidence at different stages of crop growth was studied. The research was aimed at estimating the general combining ability of parents, specific combining ability of hybrids, gene action (both Griffings and Hayman's analysis) and extent of heterosis for fifteen characters in the season I whereas in the season II, it was limited to three characters. The association and contribution of these characters to yield were also studied.

Among the 30 hybrids, eight hybrids were short listed for further evaluation mainly based on disease resistance and yield and were tested during July –October 2002 (Season III). A few more quality characters were additionally studied. Both correlation and path analysis were performed. Finally two hybrid combinations, H 24 x LCR 1 and H 24 x CLN 2123A were

selected as the hybrids with field resistance to both the viruses along with high yield. They were subjected to studies on enzyme activity, crude protein (SDS – PAGE) and isozyme analysis along with their parents in comparison with a susceptible check variety CO 3. These two hybrids were further tested for their worthiness in peak thrips activity period (December 2002) along with their parents and susceptible check CO 3. Since the individual fruit weight was less in a resistant hybrid combination H 24 x CLN 2123A, to explore the possibility of improving the fruit size, concurrently an experiment on the effect of various physical manipulation methods on the fruit weight of this hybrid was conducted. The salient findings are presented below.

1. Parents chosen were found to be geographically diverse, genetically different and phenotypically varying in expression.
2. There existed significant differences in total disease incidence at various seasons. It was highest in season I followed by season II and III as observed from the PDI values of susceptible check (CO 3) which was equally susceptible to both the viral disease under study.
3. TLCV incidence was the highest in the season II followed by season I and III, adequately revealing the conducive climatic conditions of summer month for rapid spread of this virus by its vector.
4. Tv incidence in either the parents or hybrids was almost similar in both the season I and II.
5. The incidence due to combined infection of both Tv and TLCV was not common. Within the two distinct symptoms of Tv, those plants previously infected with bronzing symptom were not infected with necrotic symptom but not vice-versa.
6. More than half of number of the hybrids tested showed highly resistant reaction for TLCV incidence at 75 DAT.
7. The hybrid H 24 x CLN 2123A showed the highest level of resistance to TLCV (< 5.00 per cent) at 75 DAT in all the seasons studied.
8. Majority of the hybrids exhibited unfavourable heterotic response for TLCV, Tv and total disease incidence, total and OD phenol, TSS, days to 50 per cent flowering and

average fruit weight. It implies that only a few hybrids could be worthwhile to select for these important characters.

9. There existed significant reciprocal differences in hybrids for most of the characters studied.
10. None of the hybrids exhibited favourable negative heterosis for earliness over best parent (LCR 3).
11. Nineteen out of 30 hybrids recorded positive heterosis estimates over their respective mid parental values for yield per plant. High heterotic hybrids mostly involved low x high, medium x medium and low x medium parental *per se* combinations.
12. The hybrid combination LCR 1 x CLN 2123A recorded appreciable fruit yield of 2.52 kg per plant without any pesticide spray simultaneously possessing higher TSS of 4.52° Brix which might be due to its better photosynthetic as well as translocational efficiency.
13. Influence of modifiers in the expression of resistance and differential response of these viruses to environment was evident as seen from the significant differences in the PDI values (either TLCV/Tv/Total) within the same cross over different seasons studied. Majority of the crosses showed this tendency.
14. The parent H 24 was found to possess good favourable *gca* for ten out 14 characters under study *viz.* plant height, number of branches per plant, days to 50 per cent flowering, number of fruits per plant, yield, lycopene, total and OD phenol, TLCV and Tv incidence at 75 DAT. The parent CLN 2123A proved as best combiner with respect to six out of 14 characters *viz.* plant height, number of branches per plant, TSS, ascorbic acid, lycopene, TLCV incidence. The other parents had proved as good combiner only for less than five characters.
15. Out of six most important characters under study *viz.* fruit weight, yield, total and OD phenol, TLCV and Tv incidence, H 24 showed its worthiness as a best combiner in five characters.
16. The hybrid H 24 x CLN 2123A exhibited high *per se*, heterosis and *sca* for eight characters out of 14 studied and four out of six important characters as mentioned in the previous point.

17. The hybrids having good *per se*, high heterosis and favourable *sca* for the six important traits namely fruit weight, yield per plant, total and OD phenol, TLCV and Tv incidence were seldom high x high *gca* parental combinations.
18. A partial failure in the assumptions of Hayman's analysis was observed for number of branches per plant, TSS, TLCV and Tv and total disease incidence.
19. Additive gene action was observed in the traits *viz.* plant height, days to 50 per cent flowering, number of fruits per plant, fruit weight, acidity and TLCV incidence at 75 DAT. The traits total and OD phenol, number of branches per plant and lycopene exhibited the preponderance of additive factors although both additive and non additive gene action were important, while non additive gene action was observed to be controlling the traits yield, TSS and ascorbic acid.
20. Differential gene action was observed for controlling the Tv incidence in both the seasons suggesting the role of modifiers which react to environmental influence.
21. All the eight hybrids selected for season III exhibited favourable significant negative heterosis over standard commercial hybrids *viz.* COTH 1 and Ankush for both Tv and Total disease incidence at 75 DAT.
22. All the eight hybrids recorded a mean total PDI of 28.00 over three seasons with the yield of more than 2.30 kg per plant. In contrast the susceptible check recorded 70 per cent mean total disease incidence with the fruit yield of merely 0.45 kg per plant.
23. Correlation studies indicated that disease incidence (TLCV /Tv/ Total) was negatively correlated with yield, total and OD phenol in both the season I and II. Correlation between fruit weight and disease incidence (TLCV / Tv/Total) was non significant hence indicating the possibility for choosing genotypes with better fruit weight still having high level of resistance to above viral diseases.
24. Number of fruits per plant and fruit weight exhibited highest significant positive correlation with yield in both the seasons and exerted highest direct effect on it. At the same time, they possessed significant negative correlation with each other.
25. Two best performed hybrids *viz.* H 24 x LCR 1 and H 24 x CLN 2123A when transplanted during mid December 2002 exhibited total PDI of 45.71 and 37.50 respectively (which is inclusive of TLCV and Tv) while the susceptible check (CO 3) recorded 98.15 per cent infection under unprotected condition. But their yield was

almost comparable with that of previous seasons indicating the mechanism of tolerance operating in the above two hybrids. This can be the major advantage of these two hybrids.

26. There existed significant increase in peroxidase, polyphenol oxidase activities in both the resistant hybrids *viz.* H 24 x LCR 1 and H 24 x CLN 2123A upon challenge inoculation of both viruses, while an increase in PAL activity was noticed only in H 24 x LCR 1. The SDS- PAGE analysis indicated the differences in band intensity of these two resistant hybrids with their parents and susceptible check. Isozyme analysis of above entries indicated that clear cut differences in number of isoforms in the resistant hybrids with their parents and susceptible check (CO 3) noticed only at 96 hours of graft inoculation of both the viruses.
27. Attempts to improve the fruit size in the resistant hybrid combination H 24 x CLN 2123A revealed that keeping two flowers per truss had resulted in significant improvement in its average fruit weight (53.55g Vs. 40.60g) and at the same time resulted in less reduction in fruit yield (2.55 Vs. 3.11 kg) per plant.
28. The virus tolerance nature of the best performed hybrids *viz.* H 24 x LCR 1 and H 24 x CLN 2123A can be used in combination with other cultural practices for further improvement in productivity. They can be worth test –verifying in different seasons as well as at different locations to confirm their suitability in the areas highly infected with above viruses.

Table 67: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for TLCV and Tv incidence at 75 DAT in the season I

Parents	TLCV at 75 DAT						Tv at 75 DAT					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	6.33**	1.91	1.24	-9.22**	-3.01	6.75*	-2.10*	4.48	4.68*	2.41	-8.03**	-0.81
LCR 1	-1.95	0.94	8.69**	-2.30	-7.90**	-5.76*	-0.01	-3.15**	-1.72	1.68	-1.16	-1.98
LCR 3	6.39**	-4.60	4.22**	1.67	9.22**	2.00	-1.52	-0.89	-3.12**	-3.84	-2.87	-0.90
CLN 2123A	3.88	5.41	3.73	-7.62**	3.60	5.41*	-4.18	-5.45*	-7.71**	6.56**	10.02**	2.90
H 24	-0.80	-3.88	-2.72	-6.81*	-3.56**	5.36*	2.46	-2.25	0.12	-14.87**	0.31	0.64
LE 415	4.31	0.00	0.22	6.40*	8.43*	-0.31	-4.11	-0.53	-13.23**	-10.17**	0.54	1.51

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	1.153			SE (gi)	0.974		
SE (sij)	2.630			SE (sij)	2.2223		
SE (rij)	3.094			SE (rij)	2.616		
SEd (gi-gj)	2.527	7.254	9.734	SEd (gi-gj)	2.136	6.132	8.228
SEd (sij-sik)	5.649	16.219	21.760	SEd (sij-sik)	4.776	13.711	18.397
SEd (sij-skl)	5.053	14.508	19.464	SEd (sij-skl)	4.272	12.264	16.456
SEd (rij-rkl)	6.188	17.768	23.836	SEd (rij-rkl)	5.231	15.019	20.150

Table 68: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for total disease incidence at 75 DAT in the season I

Parents	Total at 75 DAT					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	1.79	3.66	4.02	-2.30	- 8.22**	3.85
LCR 1	-2.14	-1.91	5.03*	1.46	-4.87*	- 5.00*
LCR 3	3.84	- 6.17*	-0.52	-3.58	-2.19	-0.01
CLN 2123A	-3.07	6.79*	-5.82*	1.39	8.65**	5.69*
H 24	0.30	-4.20	-2.18	- 12.74**	-2.36	2.60
LE 415	2.01	-2.28	- 11.81**	-8.32**	4.88	1.61

** Significance at 0.01 level

* Significance at 0.05 level

		CD (0.05)	CD (0.01)
SE (gi)	1.001		
SE (sij)	2.282		
SE (rij)	2.685		
SEd (gi-gj)	2.193	6.295	8.447
SEd (sij-sik)	4.903	14.076	18.887
SEd (sij-skl)	4.385	12.590	16.891
SEd (rij-rkl)	5.371	15.420	20.689

Table 69: Estimates of *gca* (diagonal values) and *sca* and effects of F₁ for TLCV and Tv incidence at 75 DAT in the season II

Parents	TLCV at 75 DAT						Tv at 75 DAT					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	-0.54	10.48**	3.48	-3.57	-4.87	-6.20*	0.27	1.73	-0.14	5.55**	-3.25	1.10
LCR 1	0.61	1.13	4.97	-4.24	-8.11**	-2.24	-0.52	-1.36	1.39	-5.12*	2.57	-2.45
LCR 3	-4.48	0.16	5.54**	0.35	1.12	-0.14	3.15	6.51**	1.46	-0.97	-3.41	6.96**
CLN 2123A	-7.46*	0.99	8.53*	-6.05**	4.48	-0.45	-8.47**	-0.46	1.11	1.79*	-1.30	2.69
H 24	11.02**	0.00	1.28	-6.37	-1.18	3.72	-4.26	-0.91	-4.41	-5.69*	-2.20*	4.84*
LE 415	11.98**	-2.64	2.50	3.29	4.29	1.10	-6.60**	1.50	-8.93**	-2.98	4.45	0.03

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	1.239			SE (gi)	0.870		
SE (sij)	2.826			SE (sij)	1.985		
SE (rij)	3.326			SE (rij)	2.336		
SEd (gi-gj)	2.176	7.796	10.462	SEd (gi-gj)	1.907	5.476	7.346
SEd (sij-sik)	6.072	17.432	23.389	SEd (sij-sik)	4.264	12.243	16.425
SEd (sij-skl)	5.431	15.592	20.920	SEd (sij-skl)	3.814	10.951	14.692
SEd (rij-rkl)	6.651	19.096	25.619	SEd (rij-rkl)	4.672	13.412	17.997

Table 70: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for Total disease incidence at 75 DAT in the season II

Parents	Total at 75 DAT					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	0.17	0.85	-0.57	5.03**	-0.27**	1.38
LCR 1	-0.62	-1.24	1.27	-5.27**	1.65	-1.54
LCR 3	3.16	6.51**	0.93	-0.47	-3.79*	6.87**
CLN 2123A	-8.52**	0.46	1.12	1.27	-1.50	2.53
H 24	-6.26**	-1.01	-4.41*	-5.87**	-1.84*	4.61**
LE 415	-5.20**	-0.10	-8.79**	-2.98	3.64	0.17

** Significance at 0.01 level

* Significance at 0.05 level

		CD (0.05)	CD (0.01)
SE (gi)	0.375		
SE (sij)	0.856		
SE (rij)	1.007		
SEd (gi-gj)	0.822	2.361	3.166
SEd (sij-sik)	1.839	5.279	7.084
SEd (sij-skl)	1.645	4.723	6.336
SEd (rij-rkl)	2.014	5.783	7.758

Table 61: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for plant height and number of branches per plant in the season I

Parents	Plant height						Number of branches per plant					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	1.63**	-10.98**	-3.71**	7.10**	10.67**	1.43	0.29**	-1.44**	-1.12**	0.92**	1.27**	0.14
LCR 1	-0.53	-1.42**	0.84	-7.40**	-1.23	11.48**	-0.35*	-0.30**	0.36*	0.65**	0.26	0.27
LCR 3	-3.30**	1.00	-9.57**	6.97**	-7.43**	-3.87**	-0.70**	-0.70**	-0.66**	0.53**	-0.08	-0.21
CLN 2123A	14.25**	-3.60**	6.07**	11.37**	8.49**	13.47**	0.40*	0.35*	-0.76**	0.30**	-0.57**	-0.55**
H 24	10.75**	-5.50**	-0.85	14.39**	3.70**	9.87**	0.75**	-0.55**	0.45*	0.29	0.49**	0.01
LE 415	1.70	2.70**	0.80	4.75**	-10.19**	-5.71**	0.20	0.75**	0.50**	0.72**	0.08	-0.12

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	0.349			SE (gi)	0.006		
SE (sij)	0.795			SE (sij)	0.141		
SE (rij)	0.936			SE (rij)	0.166		
SEd (gi-gj)	0.764	2.194	2.943	SEd (gi-gj)	0.134	0.385	0.516
SEd (sij-sik)	1.708	4.904	6.579	SEd (sij-sik)	0.303	0.871	1.167
SEd (sij-skl)	1.528	4.386	5.886	SEd (sij-skl)	0.272	0.781	1.048
SEd (rij-rkl)	1.871	5.373	7.207	SEd (rij-rkl)	0.332	0.952	1.279

Table 62: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for days to 50 per cent flowering and number of fruits per plant in the season I

Parents	Days to 50 per cent flowering						Number of fruits per plant					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	2.71**	-1.79**	0.38	-0.54	1.00*	0.67	-9.03**	0.46	-3.80**	-2.01	5.62**	4.32**
LCR 1	-2.25**	0.04	-0.71	0.38	-0.08	-1.92**	-1.70	-9.48**	-6.45**	5.98**	-5.87**	7.87**
LCR 3	-1.75**	2.00**	-3.13**	4.54**	-0.42	-3.75**	0.25	3.65*	8.32**	-9.00**	-7.03**	5.27**
CLN 2123A	-3.00**	3.75**	0.25	1.04**	-0.08	1.08*	-2.29	4.25**	-8.53**	0.89	13.99**	-6.42**
H 24	-2.00**	0.75	1.25**	-4.75**	-1.50**	2.38**	12.45**	3.41*	11.75**	15.15**	6.60**	8.61**
LE 415	-1.00	0.25	-0.25	0.25	2.50**	0.83**	0.75	2.45	14.85**	10.02**	-16.53**	2.70**

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	0.203			SE (gi)	0.555		
SE (sij)	0.462			SE (sij)	1.266		
SE (rij)	0.544			SE (rij)	1.489		
SEd (gi-gj)	0.445	1.278	1.714	SEd (gi-gj)	1.216	3.490	4.684
SEd (sij-sik)	0.993	2.851	3.825	SEd (sij-sik)	2.719	7.807	10.474
SEd (sij-skl)	0.888	2.459	3.421	SEd (sij-skl)	2.432	6.982	9.368
SEd (rij-rkl)	1.087	3.121	4.187	SEd (rij-rkl)	2.979	8.552	11.475

Table 63: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for fruit weight and yield per plant in the season I

Parents	Fruit weight						Yield per plant					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	9.32**	-8.69**	-3.97**	9.34**	-5.51**	0.36	-0.08**	-0.39**	0.19**	0.20**	0.15*	0.30**
LCR 1	11.13**	18.17**	-12.61**	-5.76**	2.26**	1.25**	0.16*	0.15**	-0.44**	0.23**	-0.10	0.59**
LCR 3	4.50**	8.97**	-13.61**	-1.92**	3.67**	-2.01**	0.19*	0.48**	-0.20**	-0.47**	-0.17*	-0.12
CLN 2123A	4.50**	-7.25**	7.70**	-1.49**	-0.43	0.61*	0.00	-0.03	0.06	0.05	0.52**	-0.30**
H 24	-2.75**	-1.63**	-0.50	-6.47**	-3.88**	6.06**	0.51**	0.17*	0.42**	0.27**	0.21**	0.53**
LE 415	-3.50**	-0.75**	0.60*	-0.55	3.00**	-8.50**	-0.10*	0.06	0.42**	0.40**	-0.57**	-0.12**

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	0.107			SE (gi)	0.003		
SE (sij)	0.244			SE (sij)	0.006		
SE (rij)	0.287			SE (rij)	0.076		
SEd (gi-gj)	0.236	0.679	0.909	SEd (gi-gj)	0.063	0.182	0.243
SEd (sij-sik)	0.525	1.508	2.022	SEd (sij-sik)	0.141	0.406	0.543
SEd (sij-skl)	0.469	1.346	1.807	SEd (sij-skl)	0.126	0.363	0.485
SEd (rij-rkl)	0.574	1.649	2.211	SEd (rij-rkl)	0.148	0.426	0.570

Table 64: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for TSS and acidity in the season I

Parents	TSS						Acidity					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	-0.11**	0.33**	-0.13*	-0.12	-0.02	0.29**	-0.04**	0.02**	0.02**	0.00	-0.01	0.01
LCR 1	0.53**	0.05*	0.20**	0.34**	-0.26**	-0.37**	-0.01*	-0.02**	-0.03**	-0.01	0.03**	0.02**
LCR 3	-0.23**	-0.36**	0.01	0.01	0.24**	-0.15*	0.03**	0.00	0.05**	0.03**	-0.00	-0.03**
CLN 2123A	-0.05	0.10	0.39**	0.13**	-0.03	-0.28**	-0.03**	-0.03**	-0.01*	0.03**	-0.02**	-0.02**
H 24	0.43**	0.00	-0.02	0.10	0.03	-0.16*	-0.04**	-0.03**	-0.00	0.00	0.00	0.01
LE 415	0.30**	-0.30**	-0.04	0.55**	-0.11	-0.11**	-0.01	0.01	-0.01	-0.02**	0.03**	-0.01**

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	0.003			SE (gi)	0.0002		
SE (sij)	0.006			SE (sij)	0.0005		
SE (rij)	0.007			SE (rij)	0.0006		
SEd (gi-gj)	0.063	0.181	0.243	SEd (gi-gj)	0.000	0.000	0.000
SEd (sij-sik)	0.126	0.363	0.485	SEd (sij-sik)	0.000	0.000	0.000
SEd (sij-skl)	0.109	0.315	0.422	SEd (sij-skl)	0.000	0.000	0.000
SEd (rij-rkl)	0.141	0.406	0.543	SEd (rij-rkl)	0.000	0.000	0.000

Table 65: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for ascorbic acid and lycopene in the season I

Parents	Ascorbic acid						Lycopene					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	0.51	0.83	1.82**	2.79**	-1.68*	1.11	-0.15**	0.39**	0.20**	-0.21**	-0.58**	0.32**
LCR 1	0.58	0.05	-1.82**	1.50*	4.02**	-1.76**	0.56**	-0.27**	-0.45**	-0.20**	-0.31**	0.57**
LCR 3	3.49**	6.42**	0.80**	-1.57*	-0.79	0.83	-0.45**	-0.26**	-0.20**	1.34**	-0.45**	0.14**
CLN 2123A	2.91**	0.00	2.33**	0.41	1.91**	-1.12	0.40**	-0.64**	-0.10	0.25**	0.01	-0.31**
H 24	-4.09**	-2.32**	-1.74*	0.58	-0.95**	-1.50*	0.24**	0.97**	-0.46**	0.14*	0.47**	-0.43**
LE 415	4.65**	3.68**	3.49**	-2.33**	0.58	-0.82**	0.46**	-0.48**	0.91**	0.84**	-0.57**	-0.09**

** Significance at 0.01 level * Significance at 0.05 level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	0.277			SE (gi)	0.002		
SE (sij)	0.632			SE (sij)	0.005		
SE (rij)	0.744			SE (rij)	0.006		
SEd (gi-gj)	0.607	1.742	2.338	SEd (gi-gj)	0.045	0.128	0.173
SEd (sij-sik)	1.358	3.899	5.231	SEd (sij-sik)	0.110	0.315	0.424
SEd (sij-skl)	1.215	3.488	4.680	SEd (sij-skl)	0.100	0.287	0.385
SEd (rij-rkl)	1.488	4.272	5.732	SEd (rij-rkl)	0.118	0.340	0.465

Table 66: Estimates of *gca* (diagonal values) and *sca* effects of F₁ for total and OD phenol in the season I

Parents	Total Phenol						OD Phenol					
	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415
LCR 9	-4.47*	11.33*	10.06*	0.61	3.48	-12.90*	-11.37**	35.11**	-3.62	17.39**	-5.16	-15.38**
LCR 1	-0.67	0.39	-37.33**	-26.87**	8.00	26.02**	22.97**	-4.30*	-23.79**	-52.15**	46.34**	-9.78*
LCR 3	3.38	18.40**	-10.68**	-6.53	28.77**	-18.66**	0.10	-2.85	-16.50**	0.62	15.04**	17.66**
CLN 2123A	-10.88	-1.05	29.02**	-10.04**	3.82	-8.78	-3.20	28.02**	42.45**	-7.15**	-13.18**	-19.05**
H 24	5.25	6.88	-1.18	33.08**	22.17**	-27.71**	-16.38**	32.45**	16.40**	9.18	24.82**	-32.55**
LE 415	-13.45*	-8.08	19.42**	19.90**	-23.92**	2.64	16.27**	7.75	45.18**	21.47**	-22.65**	14.50**

** Significance at 1 per cent level

* Significance at 5 per cent level

		CD (0.05)	CD (0.01)			CD (0.05)	CD (0.01)
SE (gi)	2.084			SE (gi)	1.726		
SE (sij)	4.753			SE (sij)	3.936		
SE (rij)	5.593			SE (rij)	4.632		
SEd (gi-gj)	4.567	13.111	17.592	SEd (gi-gj)	3.782	10.858	14.568
SEd (sij-sik)	10.212	29.317	39.337	SEd (sij-sik)	8.457	24.279	32.576
SEd (sij-skl)	9.134	26.222	35.184	SEd (sij-skl)	7.564	21.716	29.137
SEd (rij-rkl)	11.186	32.115	43.088	SEd (rij-rkl)	9.264	26.597	35.685

Table 74: Estimates of genetic parameters in the season I and II

	Characters	Season	D±SE(D)	F±SE(F)	H ₁ ±SE(H ₁)	H ₂ ±SE(H ₂)	h ² ±SE(h ²)	E±SE(E)
1.	Plant height	I	199.840**±39.50	85.947±96.498	431.560*±100.273	328.520*±89.577	33.099±60.245	1.730±14.929
2.	No. of branches per plant	I	0.309±0.445	-0.053±1.088	2.563±1.130	2.182±1.010	0.055±0.679	0.0537±0.168
3.	Days to 50 per cent flowering	I	28.165**±3.771	22.132±9.212	30.641*±9.573	19.961±8.552	0.242±5.752	0.576±1.425
4.	No. of fruits per plant	I	493.470**±35.020	459.030**±85.546	525.090**±88.893	327.043*±79.410	56.935±53.410	4.317±13.235
5.	Average fruit weight	I	997.318**±28.265	574.576**±69.052	349.986**±71.754	219.199*±64.100	39.202±43.111	0.169±10.683
6.	Yield per plant	I	0.465**±0.084	0.913*±0.204	1.443**±0.212	0.893**±0.190	0.239±0.128	0.011±0.032
7.	TSS	I	0.144*±0.044	0.247±0.108	0.418*±0.112	-0.281*±0.100	0.281*±0.067	0.010±0.017
8.	Acidity	I	0.007±0.000	0.003±0.000	0.002±0.000	0.0017±0.000	0.0002±0.000	0.0001±0.000
9.	Ascorbic acid	I	6.716±3.205	12.534±7.830	25.888*±8.137	18.4290±7.269	8.520±4.889	1.390±1.211
10.	Lycopene	I	1.432**±0.191	1.594*±0.466	1.826*±0.484	1.3285*±0.433	0.179±0.291	0.008±0.072
11.	Total phenol	I	485.70±256.042	494.790±625.511	2482.370*±649.990	1906.950*±580.650	935.100±390.520	61.220±96.78
12.	OD phenol	I	3571.87**±631.176	4735.200*±1561.510	6640.910*±1622.610	4454.290*±1449.510	2667.370±390.520	44.110±241.580
13	TLCV DI at 75 DAT	I	160.172**±18.203	168.214*±44.470	302.438**±46.209	197.207**±41.280	128.140**±27.760	19.140*±6.880
14	Tv DI at 75	I	7.107±15.342	-15.711±37.479	97.531±38.946	69.269±334.791	6.075±23.399	13.363±5.799

	DAT							
15.	Total DI at 75 DAT	I	0.797±12.854	0.950±31.402	95.178*±32.631	85.995*±29.150	69.190*±19.605	14.248*±4.858
16.	TLCV DI at 75DAT	II	4.852±20.596	-30.00±50.315	82.280±52.284	67.380±46.706	-11.820±31.413	22.415±7.784
17.	Tv DI at 75 DAT	II	17.957±7.676	34.11±18.753	76.664*±19.487	54.563*±17.408	40.031*±11.708	11.163*±2.901
18.	Total PDI at 75 DAT	II	2.905±11.415	2.478±27.886	130.720*±28.977	105.080*±25.886	48.120±17.410	2.002±4.314

** Significance at 1 per cent level
DI – Disease Incidence

* Significance at 5 per cent level

Table 29b: *Per se* performance of selected hybrids for different characters in the season III

Treatment	pH	Acidity (%)	TSS (°Brix)	Asc. acid (mg/100g)	Lycopene (mg/100g)	Tot. sugar (%)	Reducing sugar (%)	Sugar to acid ratio	Pectin (%)	Total phenol (mg/100g)	OD phenol (mg/100g)
LCR 1 x H 24	3.89	0.55	4.19	28.12	2.52	2.99	2.58	5.49	0.13	276.20	191.30
LCR 1 x LE 415	4.06	0.45	5.15	21.70	4.24	3.57	3.31	8.02	0.19	296.20	165.90
LCR 1 x CLN 2123A	3.88	0.49	5.57	17.36	4.20	2.78	2.36	5.62	0.29	226.23	103.10
CLN 2123A x LCR 9	3.87	0.54	5.62	29.62	3.30	2.92	2.47	5.40	0.28	240.80	120.83
H 24 x LCR 1	3.75	0.54	4.52	26.19	4.75	3.14	2.83	5.77	0.18	309.07	208.29
H 24 x LCR 9	3.57	0.50	4.60	23.59	3.86	3.87	3.41	7.73	0.33	290.30	160.40
H 24 x CLN 2123A	3.71	0.58	5.47	26.80	3.61	3.49	3.08	5.99	0.09	300.83	214.93
LE 415 x LCR 1	3.73	0.54	4.27	23.09	4.02	3.27	2.51	6.03	0.11	269.10	141.17
LCR 1	3.78	0.47	4.85	26.23	3.17	3.59	2.41	7.67	0.23	275.77	125.92
LCR 9	3.76	0.46	5.07	26.08	3.60	3.15	2.74	6.85	0.17	228.73	100.62
CLN 2123A	3.57	0.60	6.04	22.88	3.43	3.57	2.92	6.03	0.45	296.87	233.60
H 24	4.06	0.54	4.66	25.07	4.62	2.87	2.43	5.32	0.16	287.57	189.20
LE 415	3.59	0.53	4.64	27.37	3.55	2.96	2.48	5.57	0.14	301.77	236.10
COTh 1^b	3.87	0.49	4.87	22.71	4.02	4.39	3.91	8.99	0.14	217.03	84.70
Ankush^c	3.75	0.58	4.67	22.22	4.06	3.98	3.79	6.87	0.27	216.73	115.57
CD (0.05)	0.23	0.04	0.09	5.89	0.44	0.25	0.27	0.72	0.04	16.12	11.36
CD (0.01)	0.31	0.05	0.13	7.95	0.59	0.33	0.37	0.98	0.05	21.75	15.32

^b Susceptible check (hybrid) to both the diseases

^c Hybrid from corporate sector claimed to be resistant to TYLCV

Table 29a: *Per se* performance of selected hybrids for different characters in the season III

Treatment	Plant height (cm)	Branches /Plant	Days to 50 per cent flowering	No. of fruits/plant	Fruit weight (g)	Yield/plant (kg)
LCR 1 x H 24	72.82	7.60	30.67	36.80	70.65	2.60
LCR 1 x LE 415	66.43	6.13	32.00	43.93	62.22	2.73
LCR 1 x CLN 2123A	71.67	6.82	31.67	29.23	75.74	2.21
CLN 2123A x LCR 9	77.67	6.54	33.67	38.33	62.74	2.41
H 24 x LCR 1	64.50	6.50	31.00	35.43	66.42	2.35
H 24 x LCR 9	86.60	8.60	32.67	45.07	54.63	2.46
H 24 x CLN 2123A	82.45	8.20	28.00	73.33	43.65	3.20
LE 415 x LCR 1	79.80	7.80	31.00	44.90	57.00	2.56
LCR 1	72.40	6.50	36.00	24.40	117.35	2.86
LCR 9	79.00	7.54	36.67	26.70	71.98	1.92
CLN 2123A	75.57	6.60	28.67	32.73	43.82	1.44
H 24	58.10	7.53	28.67	35.35	39.13	1.38
LE 415	65.23	6.97	36.33	31.40	35.03	1.10
COTH 1^b	69.00	6.53	29.33	46.40	50.37	2.34
Ankush^c	70.10	6.60	28.00	41.33	66.03	2.73
CD (0.05)	3.16	0.27	2.53	2.35	3.49	0.17
CD (0.01)	4.19	0.36	3.41	3.18	4.71	0.23

^b Susceptible check (hybrid) to both the diseases

^c Hybrid from corporate sector claimed to be resistant to TYLCV

Table 71 : Estimates of t^2 , a, b, SEb, b-0/SEb and (1-b)/SEb

S.No	Characters	Season	t^2	a	b	SEb	b-0/SEb	1-b/SEb
1.	Plant height	I	0.3719	12.8624	0.4794	0.3120	1.5363	1.6682
2.	No. of branches per plant	I	7.5792*	-0.0190	0.2565	0.1527	1.6791	4.8684**
3.	Days to 50 per cent flowering	I	0.0003	6.7070	0.2040	0.4855	0.4200	1.6400
4.	No. of fruits per plant	I	3.1830	-51.789	1.2956	0.2749	4.7132**	-1.0754
5.	Average fruit weight	I	0.0026	168.072	0.9674	0.1147	8.4331**	0.2846
6.	Yield per plant	I	0.0339	-0.1536	0.6253	0.3468	1.8029	1.0800
7.	TSS	I	0.8347	0.0820	-0.8276	0.5902	-1.4022	3.0964*
8.	Acidity	I	4.1392	0.0010	1.1509	0.0872	13.1952**	-1.7296
9.	Ascorbic acid	I	1.4017	0.4600	-0.0007	0.8770	-0.0008	1.4100
10.	Lycopene	I	0.2411	-0.0610	0.9030	0.3702	2.4396	0.2620
11.	Total phenol	I	1.0720	-90.6740	0.3360	0.2780	1.208	2.3830
12.	OD phenol	I	0.0841	-441.728	0.7535	0.4091	1.8417	0.6025

13.	TLCV DI at 75 DAT	I	2.1682	-63.9680	1.2410	0.3460	3.5898*	-0.6971
14.	Tv DI at 75 DAT	I	27.4000**	-0.7850	0.2770	0.0850	3.2430*	8.4660**
15.	Total DI at 75 DAT	I	1.5476	39.2670	-1.1464	0.4458	-2.5713	4.8143**
16.	TLCV DI at 75 DAT	II	26.9430**	11.3260	0.0552	0.0927	0.5950	10.1900**
17.	Tv PDI at 75 DAT	II	0.1194	-14.3300	0.7690	0.2450	3.1430*	0.9440
18.	Total DI at 75 DAT	II	6.9472*	-3.9780	0.1515	0.1647	0.9200	5.1500**

** Significance at 1 per cent level

* Significance at 5 per cent level

Table 72: Estimates of variance (Vr) and co variance (Wr) of arrays for parents in the season I and II

S.No	Characters	LCR 9		LCR 1		LCR 3	
		Vr	Wr	Vr	Wr	Vr	Wr
1.	Plant height	180.8990	76.3800	50.668	19.2670	90.5120	124.8670
2.	No. of branches per plant	2.2107	0.5490	0.6120	-0.1380	0.3378	-0.1856
3.	Days to 50 per cent flowering	4.4750	6.6250	7.7170	9.5830	12.6670	3.6170
4.	No. of fruits per plant	97.5213	108.6301	73.6732	69.5744	281.4864	324.3241
5.	Average fruit weight	99.2651	243.0030	57.2620	643.4400	70.3710	187.6160
6.	Yield per plant	0.1130	-0.0399	0.2008	-0.1331	0.1953	0.1234
7.	TSS	0.0820	0.0643	0.1275	-0.0502	.0617	0.0280
8.	Acidity	0.0023	0.0037	0.0011	0.0019	0.0021	0.0031
9.	Ascorbic acid	8.7659	4.8735	6.0122	-4.0076	3.6670	2.5728
10.	Lycopene	0.0326	-0.0747	0.4223	0.6172	0.7998	0.3541
11.	Total phenol	256.0870	132.2350	1165.2430	333.2060	936.0680	-88.1620
12.	OD phenol	684.6440	632.1620	2158.168	-157.3623	564.4070	334.3300
13.	TLCV PDI at 75 DAT	96.1593	63.1260	108.1120	75.6200	113.3240	86.1080
14.	Tv PDI at 75 DAT	33.6030	1.4228	23.5300	4.2921	9.4096	2.6598

15.	Total PDI at 75 DAT	31.6960	0.6510	25.3617	2.6918	23.4865	16.6514
16.	TLCV DI at 75 DAT – Season II	74.2280	13.0500	80.610	15.7770	19.4640	11.696
17.	Tv DI at 75 DAT– Season II	21.8120	9.1460	5.8960	1.8290	17.3310	-13.1820
18.	Total DI at 75 DAT– Season II	37.581	10.8780	60.524	0.8680	32.020	-3.5693

Table 72: Estimates of variance (Vr) and co variance (Wr) of arrays for parents the season I and II (Contd.)

S.No	Characters	CLN 2123A		H 24		LE 415	
		Vr	Wr	Vr	Wr	Vr	Wr
1.	Plant height	154.1180	69.958	227.2800	147.9280	120.7770	33.914
2.	No. of branches per plant	0.5082	0.2040	0.6450	0.3856	0.6450	0.2463
3.	Days to 50 per cent flowering	6.3670	6.9580	11.185	15.0540	14.6190	10.038
4.	No. of fruits per plant	115.0907	61.7930	134.8397	58.6723	151.7913	173.1839
5.	Average fruit weight	167.1440	329.054	127.8670	336.4960	187.7750	390.650
6.	Yield per plant	0.2699	-0.0332	0.2343	-0.0850	0.5180	0.2037
7.	TSS	0.0818	-0.0054	0.0541	-0.0110	0.0991	0.0244
8.	Acidity	0.0021	0.0033	0.009	0.0019	0.0003	0.0014
9.	Ascorbic acid	5.6304	-3.2443	7.4391	-0.6647	3.4567	3.2066
10.	Lycopene	0.4349	0.1167	0.7812	0.9204	0.0527	-0.0210
11.	Total phenol	498.7550	89.9340	219.8020	-181.1510	848.2480	490.092
12.	OD phenol	1427.7850	594.292	597.6380	-411.6120	2938.1340	2665.128
13.	TLCV DI at 75 DAT	23.339	-22.8810	79.8110	-10.6090	87.843	55.9700
14.	Tv DI at 75 DAT	44.0904	11.0547	87.8723	24.3770	28.788	14.4440

15.	Total DI at 75 DAT	36.5678	7.3008	43.1656	-15.5362	32.1254	3.4539
16.	TLCV DI at 75 DAT – Season II	15.8910	7.4060	30.8550	10.8400	33.1020	23.2000
17.	Tv DI at 75 DAT– Season II	19.2360	-8.1170	8.1720	-8.4160	57.2980	32.6550
18.	Total DI at 75 DAT– Season II	18.5400	-4.8994	15.1842	-1.5065	39.8840	5.2290

Table 73: Estimates of variances and co variances for F₁ in the season I

	Characters	Season	VoLo	WoLo ₁	V ₁ L ₁	VoL ₁	(ML ₁ -ML ₀) ²
1.	Plant height	I	201.5710	78.7240	131.375	54.38000	8.5151
2.	No. of branches per plant	I	0.3630	0.1769	0.7627	0.1904	0.0212
3.	Days to 50 per cent flowering	I	28.7420	8.6460	9.5050	4.2260	0.1406
4.	No. of fruits per plant	I	497.7860	132.6960	142.4000	58.4814	14.8332
5.	Average fruit weight	I	997.4870	355.0430	193.281	138.3960	9.8240
6.	Yield per plant	I	0.4757	0.0060	0.2552	0.0264	0.0614
7.	TSS	I	0.1538	0.0120	0.0844	0.0092	0.0012
8.	Acidity	I	0.0068	0.0025	0.0015	0.0010	0.0001
9.	Ascorbic acid	I	8.1060	0.4650	5.8286	0.5261	2.3231
10.	Lycopene	I	1.4396	0.3188	0.4206	0.0846	0.0458
11.	Total phenol	I	546.9250	129.3590	654.0340	146.6850	242.2780
12.	OD phenol	I	3615.9910	609.4890	1395.1290	259.4990	672.9690
13.	TLCV DI at 75 DAT	I	179.3130	41.223	84.7650	25.8920	34.9635
14.	Tv DI at 75 DAT	I	20.4700	9.7080	37.8800	13.8840	3.3750
15.	Total DI at 75 DAT	I	15.0443	2.5354	32.0671	3.4446	19.2764

16.	TLCV DI at 75 DAT	II	27.2669	13.6620	42.3583	13.6619	0.1593
17.	Tv DI at 75 DAT	II	29.1190	2.3110	21.6390	2.4170	11.5580
18.	Total DI at 75 DAT I	II	4.9074	1.1670	33.9550	6.6830	12.3100

Table 75: Ratios of genetic parameters and other parameters in the season I and II

	Characters	Season	$(H_1/D)^{1/2}$	$H_2 / 4H_1$	K_D/K_R	W_r+V_r Vs. Yr (r)	r^2	h^2/H_2	Heritability (NS)
1.	Plant height	I	1.4700	0.1900	1.3430	-0.2140	0.0457	0.1010	0.5640
2.	No. of branches per plant	I	2.8785	0.2128	0.9422	0.8741	0.7640	0.0252	0.3830
3.	Days to 50 per cent flowering	I	1.0430	0.1630	2.2070	-0.2587	0.0609	0.0121	0.6002
4.	No. of fruits per plant	I	1.0315	0.1557	2.6422	0.8432	0.7110	0.1741	0.5746
5.	Average fruit weight	I	0.5924	0.1566	2.8931	0.7639	0.5835	0.1788	0.8343
6.	Yield per plant	I	1.7624	0.1547	3.5186	-0.5213	0.2718	0.2681	0.1785
7.	TSS	I	1.7021	0.1685	3.0229	-0.4687	0.2197	-0.0020	0.1729

8.	Acidity	I	0.5814	0.1927	2.4383	0.0774	0.0060	0.1316	0.7963
9.	Ascorbic acid	I	1.9634	0.1780	2.8117	-0.1353	0.0183	0.4623	0.1203
10.	Lycopene	I	1.1293	0.1819	2.9432	0.4525	0.2048	0.1349	0.3308
11.	Total phenol	I	2.2607	0.1921	1.5817	0.3761	0.1415	0.4904	0.3449
12.	OD phenol	I	1.3635	0.1677	2.8919	0.7250	0.5253	0.5988	0.3065
13.	TLCV DI at 75 DAT	I	1.3741	0.1630	2.2370	0.4356	0.1898	0.6500	0.4150
14.	Tv DI at 75 DAT	I	3.7045	0.178	0.5404	0.5203	0.2707	0.0877	0.4540
15.	Total DI at 75 DAT	I	10.929	0.2260	1.1150	-0.486	0.2360	0.8046	0.1121
16.	TLCV DI at 75 DAT	II	4.1179	0.2047	0.1424	0.3564	0.1270	-0.1754	0.3879
17.	Tv DI at 75 DAT	II	2.0660	0.1780	2.7010	-0.8760	0.7665	0.7340	0.1070
18.	Total DI at 75 DAT	II	6.7080	0.2010	1.1358	-0.0938	0.0088	0.4579	0.3155

DI – Disease Incidence

Table 30: PDI of selected F₁ hybrids in the season III (15 DAT and 30 DAT)

Treatment	15 DAT			30 DAT		
	TLCV	Tv	Total	TLCV	Tv	Total
LCR 1 x H 24	0.00 (0.58)	6.65 (14.94)	6.65 (14.94)	0.00 (0.58)	6.65 (14.94)	6.65 (14.94)
LCR 1 x LE 415	0.00 (0.58)	6.55 (14.83)	6.55 (14.83)	2.38 (8.87)	6.55 (14.83)	8.93 (17.39)
LCR 1 x CLN 2123A	5.55 (13.63)	2.18 (8.49)	7.68 (16.09)	7.68 (16.09)	5.51 (13.58)	13.19 (21.30)
CLN 2123A x LCR 9	3.85 (11.32)	4.55 (12.32)	8.40 (16.85)	8.39 (16.84)	8.39 (16.84)	16.78 (24.18)
H 24 x LCR 1	0.00 (0.58)	4.27 (11.93)	4.26 (11.91)	0.00 (0.58)	6.45 (14.71)	6.45 (14.71)
H 24 x LCR 9	4.16 (11.77)	4.16 (11.77)	11.09 (19.45)	9.17 (17.63)	13.33 (21.41)	22.50 (28.32)
H 24 x CLN 2123A	0.00 (0.58)	2.77 (9.58)	2.77 (9.58)	0.00 (0.58)	4.95 (12.86)	4.95 (12.86)
LE 415 x LCR 1	0.00 (0.58)	1.85 (7.82)	1.85 (7.82)	1.85 (7.82)	3.87 (11.35)	5.72 (13.84)
LCR 1	4.16 (11.77)	0.00 (0.58)	4.16 (11.77)	4.16 (11.77)	4.16 (11.77)	8.32 (16.76)
LCR 9	4.16 (11.77)	4.16 (11.77)	8.32 (16.76)	4.16 (11.77)	4.16 (11.77)	8.32 (16.76)
CLN 2123A	0.00 (0.58)	3.13 (10.19)	3.13 (10.19)	6.46 (14.72)	6.46 (14.72)	12.91 (21.06)
H 24	0.00 (0.58)	2.27 (8.67)	2.27 (8.67)	5.85 (14.00)	8.12 (16.56)	13.97 (21.95)
LE 415	3.57 (10.89)	0.00 (0.58)	3.57 (10.89)	7.74 (16.15)	7.74 (16.15)	15.48 (23.17)
CO TH 1 ^b	0.00 (0.58)	6.25 (14.48)	6.25 (14.48)	2.08 (8.29)	8.33 (16.78)	10.41 (18.82)

Ankush ^c	0.00 (0.58)	4.34 (12.02)	4.34 (12.02)	4.26 (11.91)	6.43 (14.69)	10.69 (19.08)
Co 3 ^d	2.23	12.38	14.61	7.00	23.76	30.76

Figures in parenthesis are arc sine transformed values

CD (0.05) 6.70 9.12 8.96 6.80 4.06 4.71

CD (0.01) 9.30 12.30 12.01 9.18 6.07 6.36

^b Susceptible check (hybrid) to both the diseases

^c Hybrid from corporate sector claimed to be resistant to TYLCV

^d Susceptible check (variety) to both the diseases

Table 31: PDI of selected F₁ hybrids in the season III (45 DAT and 60 DAT)

Treatment	45 DAT			60 DAT		
	TLCV	Tv	Total	TLCV	Tv	Total
LCR 1 x H 24	2.27 (8.67)	6.65 (14.94)	8.92 (17.38)	2.27 (8.67)	10.84 (19.22)	13.07 (21.19)
LCR 1 x LE 415	2.38 (8.87)	10.71 (19.10)	13.09 (21.21)	2.38 (8.87)	18.54 (25.50)	21.68 (27.75)
LCR 1 x CLN 2123A	7.68 (16.09)	7.68 (16.09)	15.36 (23.07)	7.68 (16.09)	11.01 (19.38)	21.68 (27.75)
CLN 2123A x LCR 9	8.39 (16.84)	16.78 (24.18)	25.17 (30.11)	8.39 (16.84)	16.85 (24.24)	25.24 (30.16)
H 24 x LCR 1	2.17 (8.47)	6.34 (14.58)	8.51 (16.96)	2.17 (8.47)	8.42 (16.87)	10.60 (19.00)
H 24 x LCR 9	9.17 (17.63)	13.33 (21.41)	22.50 (28.32)	9.17 (17.63)	13.34 (21.42)	22.46 (28.29)
H 24 x CLN 2123A	0.00 (0.58)	13.28 (21.37)	13.28 (21.37)	0.00 (0.58)	13.28 (21.37)	13.28 (21.37)
LE 415 x LCR 1	1.87 (7.86)	10.71 (19.10)	12.56 (20.76)	1.85 (7.82)	11.85 (20.14)	13.70 (21.72)
LCR 1	6.25 (14.48)	8.33 (16.78)	14.58 (22.45)	6.25 (14.48)	12.5 (20.70)	18.75 (25.66)
LCR 9	6.22 (14.44)	10.42 (18.83)	16.64 (24.07)	6.25 (14.48)	14.58 (22.45)	20.83 (27.15)
CLN 2123A	6.46 (14.72)	16.03 (23.60)	22.49 (28.31)	6.46 (14.72)	20.83 (27.15)	27.29 (31.49)
H 24	5.85 (14.00)	13.30 (21.39)	19.15 (25.95)	5.85 (14.00)	7.89 (16.31)	13.74 (21.76)
LE 415	7.74 (16.15)	11.31 (19.65)	19.05 (25.88)	7.74 (16.15)	19.04 (25.87)	26.78 (31.16)
CO TH 1 ^a	4.16 (11.77)	18.74 (25.65)	22.91 (28.60)	4.17 (11.77)	20.83 (27.15)	25.00 (30.00)
Ankush ^c	6.34	31.95	38.29	6.34	36.14	42.48

	(14.58)	(34.42)	(38.23)	(14.58)	(36.95)	(40.67)
Co 3 ^d	10.19	30.74	40.93	13.70	38.69	52.39

Figures in parenthesis are arc sine transformed values

CD (0.05) 7.76 4.39 5.30 7.87 5.80 5.37

CD (0.01) 10.00 5.93 7.15 10.62 7.83 7.25

^b Susceptible check (hybrid) to both the diseases

^c Hybrid from corporate sector claimed to be resistant to TYLCV

^d Susceptible check (variety) to both the diseases

Table 32: PDI of selected F₁ hybrids in the season III (75 DAT and 90 DAT)

Treatment	75 DAT			90 DAT			CI and reaction TLCV at 75 DAT
	TLCV	Tv	Total	TLCV	Tv	Total	
LCR 1 x H 24	4.36 (12.05)	10.80 (19.19)	15.15 (22.91)	4.36 (12.05)	10.80 (19.19)	15.15 (22.91)	0.54 (HR)
LCR 1 x LE 415	4.65 (12.45)	18.54 (25.50)	23.19 (28.79)	7.03 (15.38)	18.54 (25.50)	25.57 (30.38)	0.58 (HR)
LCR 1 x CLN 2123A	7.65 (16.06)	14.35 (22.26)	22.00 (27.97)	7.65 (16.06)	16.52 (23.98)	27.50 (31.63)	0.96 (HR)
CLN 2123A x LCR 9	8.39 (16.84)	16.78 (24.18)	25.17 (30.11)	8.39 (16.84)	16.78 (24.18)	25.17 (30.11)	1.05 (HR)
H 24 x LCR 1	4.27 (11.93)	10.60 (19.00)	14.87 (22.68)	4.27 (11.93)	12.77 (20.94)	17.04 (24.3)	0.53 (HR)
H 24 x LCR 9	9.12 (17.58)	18.33 (25.35)	27.45 (31.60)	9.12 (17.58)	18.33 (25.35)	27.45 (31.60)	0.52 (HR)
H 24 x CLN 2123A	4.95 (12.86)	15.46 (25.64)	20.41 (26.86)	4.95 (12.86)	17.11 (24.43)	22.06 (28.01)	0.62 (HR)
LE 415 x LCR 1	3.71 (11.11)	18.72 (21.74)	21.43 (27.58)	3.71 (11.11)	18.72 (21.74)	21.43 (27.58)	1.40 (HR)
LCR 1	8.33 (16.78)	14.58 (22.45)	22.91 (28.60)	8.33 (16.78)	16.66 (24.09)	25.0 (30.0)	1.04 (HR)
LCR 9	10.42 (18.83)	18.75 (25.66)	29.17 (32.69)	10.42 (18.83)	18.75 (25.66)	29.17 (32.69)	4.20 (R)
CLN 2123A	6.46 (14.73)	25.63 (30.42)	32.09 (34.51)	9.79 (18.23)	25.63 (30.42)	35.42 (36.52)	0.79 (HR)
H 24	9.41 (17.86)	23.53 (29.02)	32.88 (34.99)	9.41 (17.86)	26.95 (31.27)	36.36 (37.08)	2.95 (HR)
LE 415	11.31 (19.65)	23.21 (28.80)	34.52 (35.98)	11.31 (19.65)	23.21 (28.80)	34.52 (35.98)	4.76 (R)
CO ₁ H 1 ^b	8.33	25.00	33.33	10.42	27.09	37.51	3.64

	(16.78)	(30.00)	(35.26)	(18.83)	(31.36)	(37.77)	(HR)
Ankush ^c	6.34 (14.58)	39.25 (38.79)	45.59 (42.47)	8.51 (16.96)	38.33 (38.25)	46.84 (43.19)	3.17 (HR)
CO 3 ^d	14.67	41.85	56.52	15.91	42.15	58.01	-

Figures in parenthesis are arc sine transformed values

CD (0.05)	4	4.66	4.65	4.20	4.39	4.43	0.73
	.19						
CD (0.01)	5.65	6.03	6.28	5.67	5.93	5.98	0.98

HR – Highly Resistant; R – Resistant

^b Susceptible check (hybrid) to both the diseases

^c Hybrid from corporate sector claimed to be resistant to TYLCV

^d Susceptible check (variety) to both the diseases

Table 43: Heterosis (per cent) for selected F₁ hybrids and parents for plant height in the season III

Cross	di	dii	diii	SH ^x	SH ^y
LCR 1 x H 24	11.60**	0.58	-7.82**	5.54**	3.88**
LCR 1 x LE 415	-3.47*	-8.25**	-15.91**	-3.72*	-5.24**
LCR 1 x CLN 2123A	-3.129	-5.16**	-9.28**	3.87*	2.24
CLN 2123A x LCR 9	0.50	-1.68	-1.68	12.56**	10.80**
H 24 x LCR 1	-1.15	-10.91**	-18.35**	-6.52**	-7.99**
H 24 x LCR 9	26.33**	9.62**	9.62**	25.51**	23.54**
H 24 x CLN 2123A	23.36**	9.10**	4.36**	19.49**	17.62**
LE 415 x LCR 1	15.95**	10.22**	1.01	15.65**	13.84**

** Significance at 1 per cent level

^x Standard heterosis over COH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 44: Heterosis (per cent) for selected F₁ hybrids and parents for number of branches per plant in the season III

Cross	di	dii	diii	SH ^x	SH ^y
LCR 1 x H 24	8.34	0.93	0.80	16.39	15.15
LCR 1 x LE 415	-8.98	-12.05	-18.70	-6.12	-7.12
LCR 1 x CLN 2123A	4.12	3.24	-9.55	4.44	3.33
CLN 2123A x LCR 9	-7.50	-13.26	-13.26	0.15	-0.91

H 24 x LCR 1	-7.34	-13.68	-13.79	-0.46	-1.52
H 24 x LCR 9	14.13	14.06	14.06	31.70	30.30
H 24 x CLN 2123A	16.07	8.99	8.75	25.57	24.24
LE 415 x LCR 1	15.81	3.59	3.45	19.45	18.18

** Significance at 1 per cent level
^x Standard heterosis over COH 1

* Significance at 5 per cent level
^y Standard heterosis over Ankush

Table 45: Heterosis (per cent) for selected F₁ hybrids and parents for days to 50 per cent flowering in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-5.15	6.97*	6.97*	4.57	9.54**
LCR 1 x LE 415	-11.52**	-11.11**	11.61**	9.10**	14.29**
LCR 1 x CLN 2123A	-2.12	10.46**	10.46**	7.98*	13.11**
CLN 2123A x LCR 9	3.06	17.44**	17.44**	14.80**	20.25**
H 24 x LCR 1	-4.19	8.13*	8.13*	5.69	9.68**
H 24 x LCR 9	0.00	13.95**	13.95**	11.38**	16.68**
H 24 x CLN 2123A	2.34	2.34	2.34	-4.53	0.00
LE 415 x LCR 1	-14.28**	-13.89**	8.13*	5.69	9.68**

** Significance at 1 per cent level
^x Standard heterosis over COH 1

* Significance at 5 per cent level
^y Standard heterosis over Ankush

Table 46: Heterosis (per cent) for selected F₁ hybrids and parents for number of fruits per plant in season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	23.18**	4.10	4.10	-20.69**	-10.96**
LCR 1 x LE 415	39.90**	28.52**	24.27**	-5.32**	6.29**
LCR 1 x CLN 2123A	2.33	-10.69**	-17.31**	-37.00**	-29.28**
CLN 2123A x LCR 9	28.99**	17.09**	8.43**	-17.39**	-7.26**
H 24 x LCR 1	18.59**	0.23	0.23	-23.64**	-14.28**
H 24 x LCR 9	45.27**	27.49**	27.49**	-2.87	9.05**
H 24 x CLN 2123A	115.42**	107.44**	107.44**	58.03**	77.43**
LE 415 x LCR 1	60.93**	42.99**	27.02**	-3.23	8.64**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 47: Heterosis (per cent) for selected F₁ hybrids and parents for average fruit weight in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-9.70**	-39.79**	-39.79**	40.26**	6.70**
LCR 1 x LE 415	-22.45**	-46.98**	-46.98**	23.52**	-5.77**
LCR 1 x CLN 2123A	-6.01**	-35.46**	-35.46**	50.37**	14.71**
CLN 2123A x LCR 9	8.36**	-12.84**	-46.53**	24.56**	-4.98*
H 24 x LCR 1	-15.11**	-43.40**	-43.40**	31.86**	0.59
H 24 x LCR 9	-1.66	-24.10**	-53.45**	8.46**	-17.26**
H 24 x CLN 2123A	5.24	-0.39	-62.80**	-13.34**	-33.89**
LE 415 x LCR 1	-25.19**	-51.43**	-51.43**	13.16**	-13.68**

** Significance at 1 per cent level

^x Standard heterosis over COTh 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 48: Heterosis (per cent) for selected F₁ hybrids and parents for yield per plant in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	22.64**	-9.09**	-9.09**	11.11**	-4.76**
LCR 1 x LE 415	37.88**	-4.55**	-4.55*	16.67**	0.00
LCR 1 x CLN 2123A	2.79	-22.72**	-22.72**	-5.55*	-19.05**
CLN 2123A x LCR 9	43.45**	-25.52**	-15.73**	2.99	-11.72**
H 24 x LCR 1	10.85**	-17.83**	-17.83**	0.43	-13.92**
H 24 x LCR 9	49.09**	28.13**	-13.99**	5.13	-9.89**
H 24 x CLN 2123A	126.95**	122.22**	11.88**	36.75**	17.22**
LE 415 x LCR 1	-29.29**	-10.49**	-10.49**	9.40**	-6.23**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 49: Heterosis (per cent) for selected F₁ hybrids and parents for pH of the fruit juice in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-0.77	2.91	8.96**	0.52	3.73
LCR 1 x LE 415	10.18**	13.09**	13.73**	4.91**	8.27**
LCR 1 x CLN 2123A	5.58*	8.68**	8.68**	0.26	3.47
CLN 2123A x LCR 9	5.59**	8.40**	8.40**	0.00	3.20
H 24 x LCR 1	-4.34	-0.79	5.04*	-3.10	0.00
H 24 x LCR 9	-8.69**	-5.05*	0.00	-7.75**	-4.80*
H 24 x CLN 2123A	-2.75	3.92	3.92	-4.13	-1.07
LE 415 x LCR 1	1.22	3.90	4.48	-3.62	-0.53

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 50: Heterosis (per cent) for selected F₁ hybrids and parents for acidity in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	10.00**	3.77	-8.33**	12.24**	-5.17*
LCR 1 x LE 415	-10.00**	-15.09**	-25.00**	-8.16**	-22.41**
LCR 1 x CLN 2123A	-8.41**	-18.33**	-18.33**	0.00	-15.52**
CLN 2123A x LCR 9	1.88	-10.00**	-10.00**	10.20**	-6.89**
H 24 x LCR 1	6.93*	0.00	-10.00**	10.20**	-6.89**
H 24 x LCR 9	0.00	-7.41**	-16.66**	2.04	-13.79**
H 24 x CLN 2123A	1.75	-3.33	-3.33	18.36**	0.00
LE 415 x LCR 1	8.00**	1.88	-10.00**	10.20**	-6.89**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 51: Heterosis (per cent) for selected F₁ hybrids and parents for TSS in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-11.88**	-13.61**	-30.63**	-13.96**	-10.28**
LCR 1 x LE 415	8.54**	6.18**	-14.74**	5.75**	10.28**
LCR 1 x CLN 2123A	2.30**	-7.78**	-7.78**	14.37**	19.27**
CLN 2123A x LCR 9	1.17	-6.95**	-6.95**	15.40**	20.34**
H 24 x LCR 1	-4.94**	-6.80**	-25.17**	-7.19**	-3.21**
H 24 x LCR 9	-5.45**	-9.27**	-23.84**	-5.54**	-1.50*
H 24 x CLN 2123A	2.24**	-9.44**	-9.44**	12.32**	17.13**
LE 415 x LCR 1	-10.01**	-11.96	-29.31**	-12.32**	-8.56**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 52: Heterosis (per cent) for selected F₁ hybrids and parents for ascorbic acid in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	9.63	7.21	7.21	23.82*	26.55**
LCR 1 x LE 415	19.02*	-20.72**	-20.72*	-4.40	-2.34
LCR 1 x CLN 2123A	-29.30**	-33.82**	-36.57**	-23.56*	-21.87*
CLN 2123A x LCR 9	20.99**	13.57	8.22	30.43**	33.30**
H 24 x LCR 1	2.11	-0.15	-4.31	15.32	17.87
H 24 x LCR 9	-7.76	-9.55	-13.81	3.87	6.17
H 24 x CLN 2123A	11.78	6.90	-1.53	18.01	20.61*
LE 415 x LCR 1	-13.84	-15.64	-15.64	1.67	3.92

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 53: Heterosis (per cent) for selected F₁ hybrids and parents for lycopene in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-35.30**	-45.45**	-45.45**	-37.31**	-37.93**
LCR 1 x LE 415	26.19**	19.44**	-8.22*	5.19	4.43
LCR 1 x CLN 2123A	27.27**	22.45**	-9.09*	4.48	3.45
CLN 2123A x LCR 9	-6.12	-0.08	-28.18**	-17.91**	-18.72**
H 24 x LCR 1	21.95**	2.81	2.81	18.10**	16.99**
H 24 x LCR 9	-6.08	-16.45**	-16.45**	-3.98	-4.93
H 24 x CLN 2123A	-10.31*	-21.86**	-21.86**	-10.20*	-11.08**
LE 415 x LCR 1	19.64**	13.24**	-12.99**	0.00	-0.99

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 54: Heterosis (per cent) for selected F₁ hybrids and parents for total sugar in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-7.43	-16.71**	-16.71**	-31.89**	-24.87**
LCR 1 x LE 415	9.01	-0.56	-0.56	-18.68**	-10.30**
LCR 1 x CLN 2123A	-22.35**	-22.56**	-22.56**	-36.67**	-30.15**
CLN 2123A x LCR 9	-13.10*	-18.21**	-18.66**	-33.49**	-26.63**
H 24 x LCR 1	-6.55	-12.53**	-12.53**	-28.47**	-21.11**
H 24 x LCR 9	28.57**	22.86**	7.80	-11.85**	-2.76
H 24 x CLN 2123A	8.39	-2.24	-2.78	-20.50**	-12.31**
LE 415 x LCR 1	-0.15	-8.91	-8.91	-25.51**	-17.84**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 55: Heterosis (per cent) for selected F₁ hybrids and parents for reducing sugar in the season III

Cross	Di	dii	diii	SH^x	SH^y
LCR 1 x H 24	6.61	6.17	-11.64**	-34.02**	-31.93**
LCR 1 x LE 415	35.37**	33.47**	13.35**	-15.35**	-12.66**
LCR 1 x CLN 2123A	-11.44**	-19.18**	-19.18**	-39.64**	-37.73**
CLN 2123A x LCR 9	-12.72**	-15.41**	-15.41**	-36.83**	-34.83**
H 24 x LCR 1	16.94**	16.46**	-3.08	-27.62**	-25.33**
H 24 x LCR 9	31.91**	24.45**	16.78**	-12.78**	-10.03**
H 24 x CLN 2123A	15.14**	5.48	5.48	-21.23**	-18.73**
LE 415 x LCR 1	2.66	1.21	-14.04**	-35.81**	-33.77**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 56: Heterosis (per cent) for selected F₁ hybrids and parents for pectin content in the fruit in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-33.33**	-43.48**	-71.11**	-7.14	-51.85**
LCR 1 x LE 415	2.70	-17.39**	-57.77**	35.71**	-29.63**
LCR 1 x CLN 2123A	-14.70**	-35.56**	-35.56**	107.14**	7.41
CLN 2123A x LCR 9	-9.68*	-37.78**	-37.78**	100.00**	3.70
H 24 x LCR 1	-7.69	-21.70**	-60.00**	28.57**	-33.33**
H 24 x LCR 9	100.00**	94.11**	-26.67**	135.71**	22.22**
H 24 x CLN 2123A	-70.49**	-800.00**	-800.00**	-35.71**	-66.66**
LE 415 x LCR 1	-40.54**	-52.17**	-75.56**	-21.43*	-59.26**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 57: Heterosis (per cent) for selected F₁ hybrids and parents for total phenol in the season II

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-1.94	-3.95	-8.47**	27.26**	27.43**
LCR 1 x LE 415	2.57	-1.85	-1.85	36.48**	36.67**
LCR 1 x CLN 2123A	-20.99**	-23.79**	-25.03**	4.24	4.38
CLN 2123A x LCR 9	-8.40**	-18.89**	-20.20**	10.95**	11.11**
H 24 x LCR 1	9.81**	7.48**	2.42	42.40**	42.61**
H 24 x LCR 9	12.45**	0.949	-3.80	33.76**	33.95**
H 24 x CLN 2123A	2.95	1.33	-0.31	38.61**	38.80**
LE 415 x LCR 1	-6.81**	-10.83**	-10.83**	23.99**	24.16**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 58: Heterosis (per cent) for selected F₁ hybrids and parents for OD phenol in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	21.35	1.11	-18.97**	125.85**	65.53**
LCR 1 x LE 415	-8.35**	-29.73**	-29.73**	95.87**	43.55**
LCR 1 x CLN 2123A	-42.65**	-55.86**	-56.33**	21.70**	-10.79**
CLN 2123A x LCR 9	-27.69**	-48.27**	-48.82**	42.60**	4.55
H 24 x LCR 1	32.19**	10.09**	-11.77**	145.91**	80.23**
H 24 x LCR 9	10.69**	-15.22**	-32.06**	89.37**	38.79**
H 24 x CLN 2123A	1.67	-7.99**	-8.96**	153.75**	85.97**
LE 415 x LCR 1	-22.01**	-40.21**	-40.21**	66.67**	22.15**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 59a: Heterosis^w (per cent) for selected F₁ hybrids and parents for TLCV incidence at 75 DAT in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-30.43**	-28.19**	-18.19	-28.19**	-17.35
LCR 1 x LE 415	-31.65**	-25.80*	-15.48	-25.80*	-14.61
LCR 1 x CLN 2123A	1.94	9.03	9.03	-4.29	10.15
CLN 2123A x LCR 9	0.36	14.32	14.32	0.35	15.50
H 24 x LCR 1	-31.12**	-28.90**	-19.01	-28.90**	-18.18
H 24 x LCR 9	-4.17	-1.57	19.35	4.77	20.58
H 24 x CLN 2123A	-21.08*	-12.70	-12.70	-23.36*	-11.80
LE 415 x LCR 1	-39.01**	-33.79**	-24.58*	-33.79**	-23.80*

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

^w Worked out for arc sine transformed values

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 59b: Heterosis^w (per cent) for selected F₁ hybrids and parents for Tv incidence at 75 DAT in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-25.43**	-14.52	-14.52	-36.03**	-50.53**
LCR 1 x LE 415	-0.49	13.59	13.59	-15.00*	-34.26**
LCR 1 x CLN 2123A	-15.79*	-0.85	-0.85	-25.80**	-42.61**
CLN 2123A x LCR 9	-13.77*	-5.78	7.71	-19.40**	-37.66**
H 24 x LCR 1	-26.17**	-15.37*	-15.37*	-36.66**	-51.02**
H 24 x LCR 9	-7.28	-1.21	12.92	-15.50**	-34.65**
H 24 x CLN 2123A	-22.11**	-20.23**	3.12	-22.83**	-40.32**
LE 415 x LCR 1	0.06	14.21	14.21	14.53	-33.90**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

^w Worked out for arc sine transformed values

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 59c: Heterosis^w (per cent) for selected F₁ hybrids and parents for total disease incidence at 75 DAT in the season III

Cross	di	dii	diii	SH^x	SH^y
LCR 1 x H 24	-27.94**	-19.89**	-19.89**	-35.03**	-46.06**
LCR 1 x LE 415	-9.31	0.66	0.66	-18.35**	-32.21**
LCR 1 x CLN 2123A	-11.36	-2.19	-2.19	-20.67**	-34.14**
CLN 2123A x LCR 9	-10.39	-7.89	5.28	-14.61*	-29.10**
H 24 x LCR 1	-28.67**	-20.70**	-20.70**	-35.68**	-46.60**
H 24 x LCR 9	-6.62	-3.33	10.49	-10.38	-25.59**
H 24 x CLN 2123A	-22.71**	-22.17**	-6.08	-23.8**	-36.76**
LE 415 x LCR 1	-14.59*	-3.57	-3.57	-21.78**	-35.06**

** Significance at 1 per cent level

^x Standard heterosis over COTH 1

^w Worked out for arc sine transformed values

* Significance at 5 per cent level

^y Standard heterosis over Ankush

Table 3: *Per se* performance of parents and hybrids for plant height (cm) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	64.80	55.80	57.70	71.90	71.30	61.70	63.68
LCR 1	54.75	70.50	54.90	72.20	72.60	67.70	64.43
LCR 3	51.10	56.90	54.10	68.75	53.60	46.10	55.29
CLN 2123A	100.40	65.00	80.90	87.10	75.23	53.50	75.01
H 24	92.80	61.60	51.90	104.00	53.10	84.10	78.88
LE 415	65.10	73.10	47.70	63.00	63.73	49.20	62.52
Column Mean (hybrids)	72.83	62.48	58.62	75.97	67.29	62.62	
Mean hybrids	:	66.64		SE	:	1.32	
Mean parents	:	63.13		CD (0.05)	:	5.37	
				CD (0.01)	:	7.21	

Table 4: *Per se* performance of parents and hybrids for branches per plant in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	7.50	5.60	5.90	7.80	8.00	6.80	6.82
LCR 1	4.90	6.00	6.80	7.00	7.70	5.80	6.44
LCR 3	4.50	5.40	5.90	7.63	6.00	5.20	5.75
CLN 2123A	8.60	7.70	6.10	6.30	6.63	5.60	6.93
H 24	9.50	6.60	6.90	7.20	6.80	7.00	7.44
LE 415	7.20	7.30	6.20	7.04	7.15	6.80	6.98
Column Mean (hybrids)	6.94	6.52	6.38	7.33	7.10	6.08	
Mean hybrids	:	6.73		SE	:	0.23	
Mean parents	:	6.55		CD (0.05)	:	0.95	
				CD (0.01)	:	1.28	

Table 5: *Per se* performance of parents and hybrids for days to 50 per cent flowering in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	38.00	35.50	34.00	38.50	36.50	37.50	36.40
LCR 1	31.00	36.50	26.500	30.00	30.00	31.00	29.70
LCR 3	30.50	30.50	26.00	34.50	26.00	26.50	29.60
CLN 2123A	32.50	37.50	35.00	29.00	36.50	35.00	35.30
H 24	32.50	31.50	28.50	27.00	26.50	31.50	30.20
LE 415	35.50	31.50	26.00	35.50	36.50	35.50	33.00
Column Mean (hybrids)	32.40	33.30	30.00	33.10	33.10	32.30	
Mean hybrids	: 32.37		SE		: 0.77		
Mean parents	: 31.92		CD (0.05)		: 3.12		
			CD (0.01)		: 4.188		

Table 6: *Per se* performance of parents and hybrids for number of fruits per plant in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	20.40	26.70	38.30	35.20	33.80	40.30	34.86
LCR 1	23.30	22.10	31.80	36.20	30.90	41.70	32.78
LCR 3	38.80	39.10	80.70	51.80	39.20	44.50	42.68
CLN 2123A	30.63	44.70	34.75	42.30	49.40	30.21	37.94
H 24	58.70	37.73	62.70	79.70	40.95	77.50	63.27
LE 415	41.80	46.60	74.20	50.25	44.44	28.80	51.46
Column Mean (hybrids)	38.65	38.97	48.35	50.63	39.55	46.84	
Mean hybrids	: 43.83			SE	: 2.11		
Mean parents	: 39.21			CD (0.05)	: 8.55		
				CD (0.01)	: 11.47		

Table 7: *Per se* performance of parents and hybrids for fruit weight (g) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	70.50	59.00	46.5	64.00	54.00	56.00	55.90
LCR 1	81.25	111.22	34.30	69.50	69.50	63.00	63.51
LCR 3	55.50	52.24	33.00	26.60	38.00	26.60	39.79
CLN 2123A	73.00	55.00	42.00	46.50	52.00	42.50	52.90
H 24	48.50	66.25	37.00	39.05	37.50	42.00	46.56
LE 415	49.00	61.50	27.80	41.40	48.0	28.05	45.54
Column Mean (hybrids)	61.45	58.80	37.52	48.11	52.30	46.02	
Mean hybrids	: 50.70			SE	: 0.41		
Mean parents	: 54.46			CD (0.05)	: 1.65		
				CD (0.01)	: 2.21		

Table 8: *Per se* performance of parents and hybrids for yield per plant (kg) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	1.44	1.58	1.78	2.22	1.83	2.26	1.93
LCR 1	1.91	2.46	1.09	2.52	2.15	2.62	2.06
LCR 3	2.15	2.05	2.66	1.38	1.49	1.18	1.65
CLN 2123A	2.23	2.46	1.50	1.97	2.57	1.29	2.01
H 24	2.85	2.50	2.32	3.12	1.54	3.25	2.81
LE 415	2.05	2.74	2.06	2.09	2.12	0.81	2.21
Column mean (hybrids)	2.24	2.27	1.75	2.27	2.03	2.12	
Mean hybrids	: 2.11			SE	: 0.11		
Mean parents	: 1.81			CD (0.05)	: 0.435		
				CD (0.01)	: 0.584		

Table 9: *Per se* performance of parents and hybrids for TSS (°Brix) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	3.52	3.84	4.10	4.05	3.57	3.86	3.88
LCR 1	4.90	3.96	4.72	4.52	3.92	3.97	4.41
LCR 3	3.63	4.00	3.94	3.85	4.40	3.88	3.95
CLN 2123A	3.95	4.73	4.63	4.44	4.13	3.29	4.15
H 24	4.43	3.92	4.35	4.32	4.39	3.97	4.20
LE 415	4.46	3.37	3.81	4.38	3.75	4.54	3.95
Column Mean (hybrids)	4.27	3.97	4.32	4.22	3.95	3.79	
Mean hybrids	: 4.09			SE	: 0.10		
Mean parents	: 4.13			CD (0.05)	: 0.398		
				CD (0.01)	: 0.533		

Table 10: *Per se* performance of parents and hybrids for acidity (per cent) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	0.38	0.48	0.50	0.52	0.50	0.47	0.49
LCR 1	0.45	0.44	0.51	0.54	0.55	0.48	0.51
LCR 3	0.55	0.51	0.61	0.62	0.55	0.52	0.55
CLN 2123A	0.47	0.48	0.60	0.57	0.51	0.53	0.52
H 24	0.42	0.50	0.55	0.51	0.50	0.47	0.49
LE 415	0.45	0.50	0.50	0.48	0.54	0.50	0.49
Column Mean (hybrids)	0.47	0.49	0.53	0.53	0.53	0.49	
Mean hybrids	: 0.51			SE	: 0.007		
Mean parents	: 0.50			CD (0.05)	: 0.032		
				CD (0.01)	: 0.043		

Table 11: *Per se* performance of parents and hybrids for ascorbic acid (mg/100g) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	26.75	31.40	30.23	31.40	32.56	26.75	30.47
LCR 1	32.56	27.91	23.20	32.56	36.05	24.39	29.75
LCR 3	37.21	36.05	33.72	27.91	31.40	27.91	32.10
CLN 2123A	37.21	32.56	32.56	27.91	31.40	31.39	33.02
H 24	24.39	31.40	27.91	32.56	26.75	26.75	28.60
LE 415	36.05	31.75	34.89	26.75	27.91	31.40	31.47
Column Mean (hybrids)	33.48	32.63	29.76	30.24	31.86	27.44	
Mean hybrids	: 30.90			SE	: 1.05		
Mean parents	: 29.07			CD (0.05)	: 4.27		
				CD (0.01)	: 5.731		

Table 12: *Per se* performance of parents and hybrids for lycopene (mg/100g) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	2.93	2.77	3.67	2.85	2.86	2.98	3.026
LCR 1	3.90	2.21	2.71	3.78	2.90	4.05	3.47
LCR 3	2.77	2.18	2.19	4.86	3.65	2.32	3.16
CLN 2123A	3.66	2.51	4.65	3.24	3.95	2.37	3.43
H 24	3.34	4.85	2.72	4.23	5.45	3.88	3.80
LE 415	3.91	3.10	4.13	4.06	2.74	2.91	3.59
Column Mean (hybrids)	3.52	3.08	3.58	3.96	3.22	3.12	
Mean hybrids	: 3.41			SE	: 0.08		
Mean parents	: 3.16			CD (0.05)	: 0.342		
				CD (0.01)	: 0.458		

Table 13: Per se performance of parents and hybrids for total phenol (mg/100g) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	237.05	266.50	250.10	255.55	274.50	257.30	260.79
LCR 1	265.15	278.20	192.55	223.10	282.25	295.70	251.75
LCR 3	256.85	229.35	260.90	202.30	300.00	212.45	240.19
CLN 2123A	233.80	221.00	260.35	276.25	241.45	222.50	235.82
H 24	285.00	296.00	297.65	307.60	286.55	279.60	293.17
LE 415	230.40	279.55	251.30	262.30	231.75	305.90	251.06
Column Mean (hybrids)	254.24	258.48	250.39	250.17	265.99	253.51	
Mean hybrids	: 255.46		SE		: 7.91		
Mean parents	: 274.14		CD (0.05)		: 32.11		
			CD (0.01)		: 43.09		

Table 14: *Per se* performance of parents and hybrids for OD phenol (mg/100g) in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	96.95	144.50	116.45	150.10	172.70	119.5	140.65
LCR 1	190.45	143.70	106.30	56.40	182.45	156.2	138.36
LCR 3	116.65	100.60	144.45	82.55	155.00	83.2	107.60
CLN 2123A	143.70	112.45	167.45	200.10	143.35	114.85	136.36
H 24	139.95	247.35	187.80	161.70	187.20	177.45	182.85
LE 415	152.05	140.70	173.55	157.80	132.15	271.45	151.25
Column Mean (hybrids)	148.56	149.12	150.31	121.71	157.13	130.24	
Mean hybrids	:		142.85	SE	:		6.55
Mean parents	:		173.90	CD (0.05)	:		26.59
Susceptible check	:			CD (0.01)	:		35.684

Table 15: *Per se* performance of parents and hybrids for Tv incidence (per cent) 15 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	7.14 (15.50)	4.74 (12.57)	6.85 (15.17)	6.10 (14.30)	1.80 (7.71)	2.18 (8.49)	4.33
LCR 1	12.50 (20.70)	10.71 (19.10)	8.93 (17.38)	5.50 (13.56)	1.93 (7.99)	14.29 (22.21)	8.63
LCR 3	5.63 (13.73)	0.00 (0.58)	7.50 (15.89)	11.54 (19.86)	7.16 (15.52)	16.07 (23.63)	8.08
CLN 2123A	17.31 (24.59)	12.51 (20.71)	6.07 (14.26)	9.52 (17.97)	6.07 (14.26)	22.0 (27.97)	12.79
H 24	6.02 (14.92)	13.57 (21.62)	2.09 (8.31)	5.37 (13.40)	9.64 (18.09)	17.85 (24.99)	8.98
LE 415	4.75 (12.59)	3.60 (10.94)	0.00 (0.58)	3.60 (10.94)	3.57 (10.89)	0.00 (0.58)	3.10
Column Mean (hybrids)	9.24	6.88	4.79	6.42	4.11	14.48	
Mean hybrids	:	7.65		SE	:	3.50	
Mean parents	:	7.42		CD (0.05)	:	13.40	
Susceptible check	:	12.87		CD (0.01)	:	17.98	

Table 16a: *Per se* performance of parents and hybrids for TLCV incidence (per cent) 30 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	0.00 (0.58)	5.36 (13.39)	4.17 (11.78)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	1.91
LCR 1	7.14 (15.50)	7.14 (15.50)	7.14 (15.50)	0.00 (0.58)	0.00 (0.58)	3.57 (10.89)	3.51
LCR 3	11.26 (19.61)	3.79 (11.30)	0.00 (0.58)	0.00 (0.58)	5.36 (13.39)	5.36 (13.39)	5.15
CLN 2123A	0.00 (0.58)	0.00 (0.58)	3.33 (10.51)	0.00 (0.58)	3.57 (10.89)	0.00 (0.58)	1.38
H 24	0.00 (0.58)	0.00 (0.58)	1.79 (7.69)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.36
LE 415	6.08 (14.28)	0.00 (0.58)	1.79 (7.69)	5.36 (13.39)	3.57 (10.89)	0.00 (0.58)	3.36
Column Mean (hybrids)	4.90	1.83	3.64	1.07	2.5	1.79	
Mean hybrids	:	2.62		SE	:	1.01	
Mean parents	:	1.19		CD (0.05)	:	4.088	
Susceptible check	:	9.90		CD (0.01)	:	5.485	

Table 16b: Per se performance of parents and hybrids for Tv incidence at 30 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	7.14 (15.50)	6.55 (14.83)	11.61 (19.92)	29.16 (32.68)	8.00 (16.43)	14.29 (22.21)	13.92
LCR 1	19.65 (26.31)	10.71 (19.10)	8.93 (17.39)	20.62 (27.01)	12.91 (21.06)	19.29 (26.05)	16.28
LCR 3	9.47 (17.92)	9.57 (18.02)	7.14 (15.50)	34.23 (35.81)	12.50 (20.70)	30.36 (33.44)	19.23
CLN 2123A	26.92 (31.25)	14.29 (22.21)	10.00 (18.43)	17.86 (24.99)	29.64 (32.99)	38.00 (38.06)	23.77
H 24	13.89 (21.88)	15.15 (22.91)	9.52 (17.97)	25.00 (30.00)	11.07 (19.43)	20.00 (26.57)	16.71
LE 415	6.14 (14.35)	17.86 (24.99)	5.50 (13.56)	10.12 (18.55)	21.39 (27.55)	14.28 (22.20)	12.20
Column Mean (hybrids)	15.22	12.68	9.11	23.83	16.89	24.39	
Mean hybrids	:	17.02		SE	:	3.11	
Mean parents	:	11.37		CD (0.05)	:	12.64	
Susceptible check	:	17.39		CD (0.01)	:	16.97	

Table 16c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 30 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	7.14 (15.50)	11.94 (20.21)	15.77 (23.40)	29.16 (32.68)	9.79 (18.23)	14.29 (22.21)	16.19
LCR 1	26.79 (31.17)	17.85 (24.99)	16.07 (23.63)	20.62 (27.01)	12.91 (21.06)	28.56 (32.30)	20.99
LCR 3	20.72 (27.08)	21.36 (27.53)	7.14 (15.50)	34.23 (35.81)	19.64 (26.31)	35.71 (36.70)	26.33
CLN 2123A	26.92 (31.25)	14.29 (22.21)	13.81 (21.82)	17.86 (24.99)	38.78 (38.52)	38.00 (38.06)	26.36
H 24	17.59 (24.80)	15.15 (22.91)	11.31 (19.65)	25.00 (30.00)	14.64 (22.50)	20.00 (26.57)	17.81
LE 415	12.25 (20.49)	21.43 (27.58)	13.05 (21.18)	15.47 (23.16)	24.96 (29.97)	14.28 (22.2)	17.43
Column Mean (hybrids)	20.85	16.83	14.00	24.90	21.22	27.31	
Mean hybrids	:	20.85		SE	:	2.58	
Mean parents	:	13.15		CD (0.05)	:	10.48	
Susceptible check	:	28.50		CD (0.01)	:	14.06	

^a includes PDI due to combined infection of both the viruses, if any.

Table 17a: *Per se* performance of parents and hybrids for TLCV incidence (per cent) 45 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	0.00 (0.58)	11.90 (20.18)	6.25 (14.48)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	3.63
LCR 1	7.14 (15.50)	7.14 (15.50)	7.14 (15.00)	0.00 (0.58)	1.93 (7.99)	5.36 (13.39)	4.31
LCR 3	16.88 (24.26)	9.57 (18.02)	0.00 (0.58)	0.00 (0.58)	14.29 (22.21)	7.14 (15.50)	9.58
CLN 2123A	0.00 (0.58)	0.00 (0.58)	3.57 (10.89)	0.00 (0.58)	3.57 (10.89)	2.00 (8.13)	1.83
H 24	1.85 (7.82)	0.00 (0.58)	2.08 (8.29)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.79
LE 415	8.17 (16.61)	0.00 (0.58)	3.57 (10.89)	7.14 (15.50)	3.57 (10.89)	0.00 (0.58)	4.49
Column Mean (hybrids)	6.81	4.29	4.52	1.43	4.67	2.90	
Mean hybrids	:	4.10		SE	:	4.12	
Mean parents	:	1.19		CD (0.05)	:	16.72	
Susceptible check	:	14.14		CD (0.01)	:	22.44	

Table 17b. *Per se* performance of parents and hybrids for Tv incidence (per cent) 45 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	10.72 (19.11)	11.33 (19.67)	11.61 (19.92)	35.41 (36.52)	8.00 (16.43)	18.64 (25.58)	17.00
LCR 1	21.43 (27.58)	10.71 (19.10)	14.29 (22.21)	20.60 (26.99)	17.31 (24.59)	19.29 (26.05)	18.58
LCR 3	9.48 (17.93)	11.36 (19.70)	9.65 (18.10)	34.23 (34.81)	14.29 (22.21)	39.29 (38.82)	21.73
CLN 2123A	26.92 (31.25)	17.86 (24.99)	10.00 (18.43)	23.81 (29.21)	32.14 (34.54)	46.00 (42.71)	26.58
H 24	13.89 (21.88)	15.15 (22.91)	12.50 (20.70)	26.79 (31.17)	15.70 (23.34)	20.00 (26.57)	17.67
LE 415	8.17 (16.61)	17.86 (25.00)	5.50 (13.56)	19.05 (25.88)	24.99 (30.00)	17.86 (25.00)	15.11
Column Mean (hybrids)	15.98	14.72	10.78	27.22	19.35	28.64	
Mean hybrids	:	19.45		SE	:	2.84	
Mean parents	:	14.74		CD (0.05)	:	11.51	
Susceptible check	:	23.32		CD (0.01)	:	15.48	

Table 17c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) 45 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	10.72 (19.11)	23.23 (28.81)	30.36 (33.44)	35.41 (36.52)	15.57 (23.24)	18.64 (25.60)	24.64
LCR 1	28.57 (32.31)	17.85 (24.99)	33.93 (35.63)	25.16 (30.11)	19.23 (26.01)	28.21 (32.08)	27.02
LCR 3	24.36 (29.57)	22.72 (28.47)	9.65 (18.10)	34.23 (35.81)	28.58 (32.32)	50.00 (45.00)	31.98
CLN 2123A	26.92 (31.25)	17.86 (25.0)	22.14 (28.07)	23.81 (29.21)	38.78 (38.52)	48.00 (43.85)	30.74
H 24	17.74 (24.91)	15.15 (22.91)	14.58 (22.45)	26.79 (31.17)	15.70 (23.34)	20.00 (26.57)	18.86
LE 415	16.33 (23.83)	21.43 (27.58)	16.07 (23.62)	26.17 (30.77)	28.56 (32.30)	17.86 (24.99)	21.71
Column Mean (hybrids)	22.79	20.08	23.42	29.55	26.14	32.97	
Mean hybrids	:	25.83		SE	:	2.73	
Mean parents	:	15.93		CD (0.05)	:	11.09	
Susceptible check	:	39.32		CD (0.01)	:	14.87	

^a includes PDI due to combined infection of both the viruses, if any.

Table 18a: *Per se* performance of parents and hybrids for TLCV incidence (per cent) at 60 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	25.00 (30.00)	18.39 (25.39)	8.33 (16.78)	0.00 (0.58)	6.00 (14.18)	10.09 (18.52)	8.56
LCR 1	12.50 (20.70)	10.71 (19.10)	30.20 (33.34)	0.00 (0.58)	3.85 (11.32)	5.36 (13.39)	10.38
LCR 3	29.91 (33.15)	17.36 (24.62)	0.00 (0.58)	5.00 (12.92)	21.43 (27.58)	8.93 (17.39)	16.53
CLN 2123A	1.93 (5.66)	3.57 (7.75)	12.14 (20.32)	0.00 (0.58)	3.57 (10.89)	2.00 (8.13)	4.64
H 24	1.85 (7.99)	0.00 (0.58)	10.42 (18.83)	0.00 (0.58)	0.00 (0.58)	3.57 (10.89)	3.17
LE 415	32.50 (34.77)	3.57 (10.89)	11.13 (19.48)	8.33 (16.78)	14.29 (22.21)	0.00 (0.58)	13.96
Column Mean (hybrids)	15.14	8.58	14.44	2.67	9.83	5.99	
Mean hybrids	:	9.45		SE	:	4.18	
Mean parents	:	5.95		CD (0.05)	:	16.98	
Susceptible check	:	16.50		CD (0.01)	:	22.79	

Table 18b: *Per se* performance of parents and hybrids for Tv incidence (per cent) at 60 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	10.72 (19.11)	19.05 (25.88)	24.50 (29.67)	41.67 (40.20)	8.00 (16.43)	25.93 (30.61)	23.83
LCR 1	21.43 (27.58)	14.28 (22.20)	14.28 (22.20)	23.37 (28.91)	21.16 (27.39)	19.29 (26.05)	19.91
LCR 3	20.73 (27.08)	11.36 (19.70)	21.79 (27.83)	35.39 (36.51)	16.07 (23.63)	39.29 (38.82)	24.57
CLN 2123A	26.92 (31.25)	35.71 (36.70)	10.00 (18.43)	23.81 (29.21)	63.57 (52.87)	60.20 (50.88)	39.28
H 24	13.89 (21.88)	15.15 (22.91)	14.58 (22.45)	26.79 (31.17)	19.65 (26.31)	23.23 (28.81)	18.72
LE 415	16.25 (23.77)	17.86 (25.00)	5.50 (13.56)	22.63 (28.41)	25.0 (30.00)	25.00 (30.00)	17.45
Column Mean (hybrids)	19.84	19.82	13.77	29.97	26.76	35.59	
Mean hybrids	:	23.96		SE	:	2.35	
Mean parents	:	19.21		CD (0.05)	:	9.53	
Susceptible check	:	38.25		CD (0.01)	:	12.78	

Table 18c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 60 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	35.72 (36.70)	39.82 (39.13)	32.83 (34.96)	41.67 (40.20)	18.00 (25.10)	34.67 (36.07)	33.40
LCR 1	33.93 (35.63)	24.99 (29.99)	49.96 (44.98)	25.16 (30.11)	26.93 (31.26)	28.21 (32.08)	32.84
LCR 3	50.64 (45.37)	28.71 (32.40)	21.79 (27.83)	40.39 (39.46)	37.50 (37.76)	49.98 (44.99)	41.44
CLN 2123A	28.81 (32.46)	39.28 (38.82)	22.14 (28.07)	23.81 (29.21)	67.14 (55.02)	62.20 (52.06)	43.91
H 24	17.74 (24.91)	15.15 (22.91)	25.00 (30.00)	26.79 (31.17)	19.65 (26.31)	26.98 (31.29)	22.33
LE 415	48.75 (44.28)	21.43 (27.58)	16.76 (24.17)	30.95 (33.80)	39.29 (38.82)	25.00 (30.00)	31.44
Column Mean (hybrids)	35.97	28.87	29.34	32.99	37.77	40.41	
Mean hybrids	:	34.23		SE	:	3.26	
Mean parents	:	25.16		CD (0.05)	:	13.23	
Susceptible check	:	61.70		CD (0.01)	:	17.75	

^a includes PDI due to combined infection of both the viruses, if any.

Table 19a: *Per se* performance of parents and hybrids for TLCV incidence (per cent) at 75 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	25.01 (30.00)	19.64 (26.31)	12.20 (20.44)	0.00 (0.58)	7.76 (16.17)	15.84 (23.45)	11.09
LCR 1	14.28 (22.21)	14.29 (22.21)	30.36 (33.43)	0.00 (0.58)	3.85 (11.32)	5.36 (13.39)	10.77
LCR 3	29.90 (33.15)	17.36 (24.62)	0.00 (0.58)	5.00 (12.92)	21.43 (27.58)	12.50 (20.70)	17.24
CLN 2123A	3.85 (11.32)	7.15 (15.51)	8.57 (17.02)	0.00 (0.58)	6.07 (14.26)	2.00 (8.13)	5.53
H 24	3.57 (10.89)	0.00 (0.58)	14.25 (22.18)	0.00 (0.58)	0.00 (0.58)	3.57 (10.89)	4.28
LE 415	28.5 (32.27)	5.36 (13.39)	13.05 (21.18)	10.71 (19.10)	17.86 (24.99)	0.00 (0.58)	15.10
Column Mean (hybrids)	16.02	9.90	15.69	3.14	11.39	7.85	
Mean hybrids	:	10.67		SE	:	4.38	
Mean parents	:	6.55		CD (0.05)	:	17.77	
Susceptible check	:	17.8		CD (0.01)	:	23.84	

Table 19b: *Per se* performance of parents and hybrids for Tv incidence (per cent) at 75 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	14.26 (22.21)	22.26 (28.15)	25.30 (30.20)	41.60 (40.16)	9.14 (17.60)	28.11 (32.20)	25.28
LCR 1	23.21 (28.80)	14.29 (22.21)	14.29 (22.21)	23.37 (28.91)	21.16 (27.39)	19.29 (26.05)	20.26
LCR 3	20.73 (20.08)	12.0 (2.27)	21.79 (27.82)	35.39 (36.51)	16.07 (23.63)	41.09 (39.87)	25.06
CLN 2123A	28.85 (32.49)	41.07 (39.86)	13.57 (21.62)	23.81 (29.21)	63.57 (52.87)	62.00 (51.94)	41.81
H 24	18.06 (25.15)	15.15 (22.91)	16.00 (23.58)	26.78 (31.46)	26.79 (31.17)	26.78 (31.16)	20.55
LE 415	16.25 (23.77)	17.86 (25.00)	5.50 (13.56)	25.00 (30.00)	28.57 (32.31)	28.57 (32.31)	18.64
Column Mean (hybrids)	21.42	21.67	14.93	30.43	27.70	35.45	
Mean hybrids	:	25.27		SE	:	3.70	
Mean parents	:	21.59		CD (0.05)	:	15.02	
Susceptible check	:	52.15		CD (0.01)	:	20.15	

Table 19c: *Per se* performance of parents and hybrids for Total disease incidence^a (per cent) at 75 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	39.30 (38.82)	44.64 (41.92)	37.45 (37.73)	41.65 (40.19)	20.90 (27.20)	43.95 (41.53)	37.72
LCR 1	37.5 (37.76)	28.57 (32.34)	50.00 (45.00)	25.65 (30.43)	26.93 (31.26)	30.00 (33.21)	34.02
LCR 3	50.63 (45.36)	29.36 (32.81)	21.79 (27.83)	40.39 (39.46)	37.5 (37.76)	57.16 (49.12)	43.01
CLN 2123A	32.69 (34.87)	48.22 (43.98)	22.14 (28.07)	23.81 (29.21)	69.64 (56.56)	64.00 (53.13)	47.34
H 24	21.76 (27.81)	15.15 (22.91)	30.25 (33.36)	26.78 (31.16)	26.79 (31.17)	30.36 (33.44)	24.86
LE 415	50.84 (45.48)	23.22 (28.81)	18.54 (25.50)	35.71 (36.69)	46.93 (43.24)	28.57 (32.31)	35.05
Column Mean (hybrids)	38.68	32.12	31.68	34.04	40.38	45.09	
Mean hybrids	:	37.00		SE	:	3.80	
Mean parents	:	28.14		CD (0.05)	:	15.34	
Susceptible check	:	83.91		CD (0.01)	:	20.58	

^a includes PDI due to combined infection of both the viruses, if any.

Table 20a: *Per se* performance of parents and hybrids for TLCV incidence (per cent) at 90 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	28.57 (32.31)	22.04 (28.00)	13.99 (21.96)	0.00 (0.58)	7.89 (16.31)	15.84 (23.45)	11.95
LCR 1	17.86 (25.00)	17.86 (25.00)	32.14 (34.54)	0.00 (0.58)	4.43 (12.15)	5.36 (13.39)	11.96
LCR 3	29.99 (33.20)	19.15 (25.95)	1.71 (7.51)	5.00 (12.92)	21.43 (27.58)	12.50 (20.70)	17.61
CLN 2123A	3.85 (11.32)	10.70 (19.09)	8.57 (17.02)	0.00 (0.58)	6.07 (14.26)	2.00 (8.13)	6.24
H 24	6.02 (14.20)	1.79 (7.69)	14.75 (22.59)	0.00 (0.58)	1.26 (6.45)	5.36 (13.39)	5.58
LE 415	34.57 (36.01)	5.36 (13.39)	13.19 (21.30)	17.86 (25.00)	28.57 (32.31)	0.00 (0.58)	19.91
Column Mean (hybrids)	18.46	11.81	16.53	4.57	13.68	8.21	
Mean hybrids	:	12.21		SE	:	4.71	
Mean parents	:	8.23		CD (0.05)	:	19.32	
Susceptible check	:	19.63		CD (0.01)	:	25.93	

Table 20b: *Per se* performance of parents and hybrids for Tv incidence (per cent) at 90 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	14.29 (22.21)	22.62 (28.40)	27.09 (31.36)	41.67 (40.20)	11.14 (19.50)	28.11 (32.02)	26.13
LCR 1	23.21 (28.80)	14.28 (22.21)	14.29 (22.21)	23.38 (28.92)	21.16 (27.39)	19.29 (26.05)	20.27
LCR 3	20.73 (27.08)	12.00 (20.27)	21.79 (27.83)	35.39 (36.51)	17.86 (24.99)	42.89 (40.91)	25.77
CLN 2123A	28.84 (32.48)	44.64 (41.92)	13.57 (21.62)	23.81 (29.21)	70.71 (57.23)	62.00 (51.94)	43.95
H 24	18.06 (24.15)	15.15 (22.91)	16.00 (23.58)	28.57 (32.31)	26.78 (31.16)	26.79 (31.17)	20.91
LE 415	16.25 (23.77)	17.86 (24.99)	5.50 (13.56)	28.57 (32.31)	25.00 (30.00)	28.57 (32.31)	18.64
Column Mean (hybrids)	21.42	22.45	15.29	31.52	29.17	35.82	
Mean hybrids	:	25.95		SE	:	3.37	
Mean parents	:	21.59		CD (0.05)	:	13.95	
Susceptible check	:	54.09		CD (0.01)	:	18.72	

Table 20c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 90 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	42.86 (40.89)	47.03 (43.30)	41.07 (39.86)	41.67 (40.20)	22.75 (28.48)	43.95 (41.53)	39.29
LCR 1	41.07 (39.86)	32.15 (34.54)	53.57 (47.04)	25.71 (30.47)	27.52 (31.64)	30.00 (33.21)	35.57
LCR 3	50.72 (45.41)	31.15 (33.93)	25.36 (30.24)	40.39 (39.46)	39.29 (38.82)	58.96 (50.16)	44.10
CLN 2123A	32.69 (34.87)	55.34 (48.07)	22.14 (28.07)	23.81 (29.21)	76.07 (60.71)	64.00 (53.13)	50.05
H 24	24.07 (29.38)	16.93 (24.29)	30.75 (33.68)	28.57 (32.31)	29.29 (32.77)	32.14 (34.54)	26.49
LE 415	50.82 (45.47)	23.22 (28.80)	18.68 (25.61)	46.43 (42.95)	53.57 (47.05)	28.57 (32.31)	38.54
Column Mean (hybrids)	39.87	34.73	33.24	36.55	43.84	45.81	
Mean hybrids	:	39.01		SE	:	3.89	
Mean parents	:	30.34		CD (0.05)	:	15.79	
Susceptible check	:	87.98		CD (0.01)	:	21.19	

^a includes PDI due to combined infection of both the viruses, if any.

21. Co-efficient of infection (CI) for TLCV incidence at 75 DAT in the season I

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	12.51	8.33	11.84	0.00	10.06	2.03	6.45
LCR 1	4.12	3.58	10.27	0.00	0.45	2.01	3.37
LCR 3	22.89	8.23	0.00	0.63	5.37	3.53	8.13
CLN 2123A	0.48	0.90	1.07	0.00	0.76	0.25	0.69
H 24	1.45	0.00	10.03	0.00	0.00	0.45	2.39
LE 415	17.28	1.78	4.52	2.68	4.02	0.00	6.06
Column Mean (hybrids)	9.24	3.84	7.55	0.66	4.13	1.65	
Mean hybrids	: 4.51			SE	: 2.87		
Mean parents	: 2.68			CD (0.05)	: 11.64		
				CD (0.01)	: 15.62		

Table 22a: *Per se* performance of parents and hybrids for TLCV (per cent) incidence at 15 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00
LCR 1	0.00 (0.58)	2.34 (8.80)	0.00 (0.58)	0.00 (0.58)	1.41 (6.82)	0.00 (0.58)	0.28
LCR 3	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	1.82 (7.75)	0.36
CLN 2123A	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00
H 24	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	1.15 (6.16)	0.23
LE 415	0.00 (0.58)	1.05 (5.88)	3.66 (11.03)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.94
Column Mean (hybrids)	0.00	0.21	0.73	0.00	0.28	0.59	
Mean hybrids	:	0.303		SE	:	3.21	
Mean parents	:	0.39		CD (0.05)	:	NS	
Susceptible check	:	1.20		CD (0.01)	:	NS	

Table 22b: *Per se* performance of parents and hybrids for Tv (per cent) incidence at 15 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	1.47 (6.96)	8.33 (16.78)	0.00 (0.58)	9.99 (18.43)	0.00 (0.58)	3.15 (10.22)	4.29
LCR 1	0.00 (0.58)	4.44 (12.16)	9.68 (18.13)	0.00 (0.58)	0.00 (0.58)	2.33 (8.78)	2.40
LCR 3	0.00 (0.58)	0.00 (0.58)	3.86 (11.38)	0.00 (0.58)	2.13 (8.39)	14.28 (22.20)	3.28
CLN 2123A	12.49 (20.70)	0.95 (5.59)	9.99 (18.43)	1.27 (6.47)	0.00 (0.58)	0.00 (0.58)	4.69
H 24	7.37 (15.75)	2.86 (9.74)	1.15 (6.16)	0.00 (0.58)	1.15 (6.16)	0.00 (0.58)	2.28
LE 415	3.85 (11.32)	2.13 (8.39)	3.66 (11.03)	10.25 (18.67)	1.49 (7.01)	0.00 (0.58)	4.28
Column Mean (hybrids)	4.74	2.85	4.90	4.05	0.72	3.95	
Mean hybrids	:	3.54		SE	:	3.99	
Mean parents	:	2.03		CD (0.05)	:	16.20	
Susceptible check	:	12.0		CD (0.01)	:	21.73	

Table 22c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 15 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	1.47 (6.96)	8.33 (16.78)	0.00 (0.58)	10.00 (18.43)	0.00 (0.58)	12.51 (20.71)	6.17
LCR 1	0.00 (0.58)	8.40 (16.85)	9.72 (18.17)	0.00 (0.58)	2.78 (9.60)	4.55 (12.32)	3.41
LCR 3	0.00 (0.58)	0.00 (0.58)	3.87 (11.34)	0.00 (0.58)	4.17 (11.78)	21.39 (27.55)	5.11
CLN 2123A	12.50 (20.70)	1.88 (7.88)	10.00 (18.43)	1.26 (6.45)	2.68 (9.42)	0.00 (0.58)	5.41
H 24	7.73 (16.14)	5.56 (13.64)	2.28 (8.68)	0.00 (0.58)	1.15 (6.16)	2.28 (8.68)	3.57
LE 415	3.85 (11.32)	6.25 (14.48)	7.33 (15.71)	10.96 (19.33)	2.94 (9.87)	0.00 (0.58)	6.27
Column Mean (hybrids)	4.82	4.40	5.87	4.19	2.51	8.15	
Mean hybrids	:	4.99		SE	:	4.57	
Mean parents	:	2.69		CD (0.05)	:	18.72	
Susceptible check	:	13.20		CD (0.01)	:	25.11	

^a includes PDI due to combined infection of both the viruses, if any.

Table 23a: *Per se* performance of parents and hybrids for TLCV (per cent) incidence at 30 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	0.00 (0.58)	0.00 (0.58)	7.14 (15.50)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	1.43
LCR 1	0.00 (0.58)	6.82 (15.14)	0.00 (0.58)	0.00 (0.58)	2.78 (9.60)	8.33 (16.78)	2.22
LCR 3	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	7.14 (15.50)	1.43
CLN 2123A	0.00 (0.58)	0.00 (0.58)	10.00 (18.43)	0.00 (0.58)	0.00 (0.58)	0.00 (0.58)	2.00
H 24	5.56 (13.64)	0.00 (0.58)	6.25 (14.48)	0.00 (0.58)	3.33 (10.47)	2.28 (8.68)	2.82
LE 415	2.79 (9.60)	2.08 (8.29)	5.34 (13.36)	2.63 (9.32)	0.00 (0.58)	1.67 (7.43)	2.57
Column Mean (hybrids)	1.67	0.42	5.75	0.53	0.56	3.55	
Mean hybrids	:	2.08		SE	:	2.77	
Mean parents	:	1.97		CD (0.05)	:	11.21	
Susceptible check	:	3.93		CD (0.01)	:	15.05	

Table 23b: *Per se* performance of parents and hybrids for Tv (per cent) incidence at 30 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	6.85 (15.17)	8.33 (16.78)	7.14 (15.50)	10.00 (18.43)	3.33 (10.51)	25.00 (30.00)	10.76
LCR 1	0.00 (0.58)	8.89 (17.35)	13.89 (21.88)	0.00 (0.58)	5.56 (13.64)	4.55 (12.32)	4.80
LCR 3	7.14 (15.50)	0.00 (0.58)	11.61 (19.92)	15.00 (22.79)	7.29 (15.66)	22.62 (28.40)	10.41
CLN 2123A	12.50 (20.70)	8.89 (17.35)	10.00 (18.43)	7.78 (16.20)	3.33 (10.51)	5.00 (12.92)	7.94
H 24	9.90 (18.33)	5.56 (13.64)	7.32 (15.70)	0.00 (0.58)	5.61 (13.70)	2.16 (8.45)	4.99
LE 415	9.62 (18.07)	6.80 (15.16)	9.00 (17.45)	25.06 (30.04)	8.82 (17.28)	1.67 (7.43)	11.86
Column Mean (hybrids)	7.83	6.02	9.47	10.01	5.67	11.87	
Mean hybrids	:	8.47		SE	:	4.12	
Mean parents	:	7.07		CD (0.05)	:	17.18	
Susceptible check	:	24.35		CD (0.01)	:	23.05	

Table 23c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 30 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	6.85 (15.17)	8.33 (16.78)	14.28 (22.20)	10.00 (18.43)	3.33 (10.51)	25.00 (30.00)	12.19
LCR 1	0.00 (0.58)	15.71 (23.35)	13.89 (21.88)	0.00 (0.58)	7.83 (16.25)	12.88 (21.03)	6.92
LCR 3	7.14 (15.50)	0.00 (0.58)	11.61 (19.92)	15.00 (22.79)	7.29 (15.66)	29.76 (33.06)	11.84
CLN 2123A	12.50 (20.70)	8.89 (17.35)	20.00 (26.57)	7.78 (16.20)	3.33 (10.51)	5.00 (12.92)	9.94
H 24	15.46 (23.15)	5.56 (13.64)	14.78 (22.61)	0.00 (0.58)	8.94 (17.40)	4.44 (12.16)	8.05
LE 415	12.40 (20.62)	8.88 (17.34)	14.34 (22.25)	27.69 (31.74)	8.82 (17.28)	3.33 (10.51)	14.43
Column Mean (hybrids)	9.50	6.33	15.46	10.54	6.12	15.42	
Mean hybrids	:	10.56		SE	:	4.18	
Mean parents	:	9.04		CD (0.05)	:	17.44	
Susceptible check	:	28.28		CD (0.01)	:	23.40	

^a includes PDI due to combined infection of both the viruses, if any.

Table 24a: *Per se* performance of parents and hybrids for TLCV (per cent) incidence at 45 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	4.68 (12.49)	5.00 (12.92)	11.31 (19.65)	7.14 (15.5)	0.00 (0.58)	0.00 (0.58)	4.69
LCR 1	25.0 (30.00)	6.82 (15.14)	14.72 (22.56)	0.00 (0.58)	5.56 (13.64)	12.88 (21.03)	11.63
LCR 3	7.14 (15.50)	6.25 (14.48)	3.87 (11.35)	5.00 (12.92)	3.13 (10.19)	7.14 (15.50)	5.73
CLN 2123A	0.00 (0.58)	1.85 (7.82)	10.00 (18.43)	0.00 (0.58)	7.17 (15.53)	0.00 (0.58)	3.80
H 24	7.12 (15.48)	0.00 (0.58)	9.38 (17.83)	0.00 (0.58)	3.34 (10.53)	4.54 (12.30)	4.21
LE 415	6.73 (15.04)	4.17 (11.78)	9.33 (17.79)	5.27 (13.27)	7.14 (15.50)	5.00 (12.92)	6.53
Column Mean (hybrids)	9.20	3.45	10.95	3.48	4.60	4.91	
Mean hybrids	:	6.10		SE	:	3.86	
Mean parents	:	3.95		CD (0.05)	:	15.67	
Susceptible check	:	19.08		CD (0.01)	:	21.03	

Table 24b: *Per se* performance of parents and hybrids for Tv (per cent) incidence at 45 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (Hybrids)
LCR 9	9.35 (17.80)	13.33 (21.41)	7.14 (15.50)	17.15 (24.46)	16.67 (24.09)	25.00 (30.00)	15.86
LCR 1	22.50 (28.32)	15.52 (23.20)	13.89 (21.88)	5.00 (12.92)	11.11 (19.47)	4.55 (12.32)	11.41
LCR 3	32.15 (34.54)	18.75 (25.66)	13.74 (21.76)	21.43 (27.58)	10.42 (18.83)	38.10 (38.12)	24.17
CLN 2123A	16.07 (23.63)	8.87 (17.32)	10.00 (18.43)	16.54 (24.00)	6.66 (14.96)	21.11 (27.35)	12.54
H 24	9.90 (18.34)	9.13 (17.59)	7.32 (15.70)	7.32 (15.70)	9.17 (17.63)	6.63 (14.92)	8.06
LE 415	10.58 (18.98)	9.43 (17.86)	24.67 (29.78)	24.56 (29.71)	15.97 (23.55)	6.67 (14.97)	17.04
Column Mean (hybrids)	18.24	11.90	12.60	15.09	12.17	19.08	
Mean hybrids	:	14.85		SE	:	3.18	
Mean parents	:	11.83		CD (0.05)	:	12.90	
Susceptible check	:	30.32		CD (0.01)	:	17.32	

Table 24c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 45 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	9.03 (17.49)	18.33 (25.35)	18.45 (25.44)	24.29 (29.53)	16.67 (24.10)	25.00 (30.00)	20.55
LCR 1	50.00 (45.00)	22.33 (28.20)	28.61 (32.34)	7.78 (16.20)	24.99 (29.99)	17.42 (24.66)	25.76
LCR 3	39.33 (38.84)	25.00 (30.00)	17.44 (24.68)	26.43 (30.94)	13.54 (21.59)	45.26 (42.28)	29.91
CLN 2123A	16.07 (23.63)	10.74 (19.13)	20.00 (26.57)	16.56 (24.01)	13.80 (21.81)	21.11 (27.35)	16.34
H 24	17.02 (24.37)	9.13 (17.59)	16.70 (24.12)	7.32 (15.70)	12.50 (20.70)	11.17 (19.52)	12.27
LE 415	17.31 (24.59)	13.59 (21.63)	34.00 (35.66)	29.83 (33.10)	23.11 (28.73)	11.66 (19.97)	23.57
Column Mean (hybrids)	27.95	15.36	23.55	19.13	18.42	23.99	
Mean hybrids	:	21.40		SE	:	2.77	
Mean parents	:	14.92		CD (0.05)	:	11.23	
Susceptible check	:	49.39		CD (0.01)	:	15.10	

^a includes PDI due to combined infection of both the viruses, if any.

Table 25a: *Per se* performance of parents and hybrids for TLCV (per cent) incidence at 60 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	6.85 (15.17)	10.00 (18.43)	15.49 (23.13)	7.14 (15.50)	0.00 (0.58)	0.00 (0.58)	6.53
LCR 1	25.00 (30.0)	8.99 (17.44)	20.83 (27.15)	0.00 (0.58)	5.56 (13.64)	12.88 (21.03)	12.85
LCR 3	7.14 (15.50)	18.75 (25.66)	9.52 (17.97)	5.00 (12.92)	10.42 (18.83)	14.28 (22.20)	11.12
CLN 2123A	0.00 (0.58)	1.85 (7.817)	20.00 (26.57)	0.00 (0.58)	7.14 (15.50)	5.00 (12.92)	6.80
H 24	9.90 (18.34)	0.00 (0.58)	17.90 (25.03)	4.55 (12.32)	3.33 (10.51)	6.82 (15.14)	7.83
LE 415	9.66 (18.11)	6.25 (14.48)	23.00 (28.66)	5.27 (13.27)	10.08 (18.51)	5.00 (12.92)	10.85
Column Mean (hybrids)	10.34	7.37	19.44	4.39	6.64	7.80	
Mean hybrids	:	9.33		SE	:	4.04	
Mean parents	:	5.62		CD (0.05)	:	16.39	
Susceptible check	:	28.11		CD (0.01)	:	21.99	

Table 25b: *Per se* performance of parents and hybrids for Tv (per cent) incidence at 60 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	16.20 (23.73)	13.33 (21.41)	15.48 (23.17)	51.43 (45.82)	20.00 (26.57)	37.50 (37.76)	27.55
LCR 1	25.00 (30.00)	19.87 (26.47)	18.06 (24.15)	15.00 (22.79)	13.89 (21.88)	12.88 (21.03)	16.97
LCR 3	32.14 (34.54)	25.00 (30.00)	19.35 (26.10)	26.67 (31.09)	21.88 (27.89)	45.24 (42.27)	30.19
CLN 2123A	19.65 (26.31)	10.74 (19.13)	30.00 (33.21)	21.21 (27.42)	13.81 (21.82)	31.11 (33.90)	21.06
H 24	9.90 (18.34)	12.70 (20.88)	9.95 (18.39)	10.10 (18.53)	11.21 (19.56)	9.10 (17.56)	10.35
LE 415	12.50 (20.70)	12.06 (20.32)	16.33 (23.83)	24.56 (29.71)	18.91 (25.78)	6.66 (14.96)	16.87
Column Mean (hybrids)	19.84	14.77	17.96	25.55	17.70	27.17	
Mean hybrids	:	20.50		SE	:	3.27	
Mean parents	:	15.75		CD (0.05)	:	12.92	
Susceptible check	:	31.93		CD (0.01)	:	17.34	

Table 25c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 60 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	23.05 (28.69)	23.33 (28.88)	30.96 (33.81)	58.57 (49.93)	20.00 (26.57)	37.50 (37.76)	34.07
LCR 1	50.00 (45.00)	28.86 (32.49)	38.89 (38.58)	15.00 (22.79)	27.77 (31.80)	25.75 (30.49)	31.48
LCR 3	39.28 (38.81)	43.75 (41.41)	28.87 (32.50)	36.17 (36.97)	32.29 (34.63)	59.52 (50.49)	42.20
CLN 2123A	19.65 (26.31)	12.59 (20.78)	50.00 (45.00)	21.21 (27.42)	20.96 (27.25)	31.11 (33.90)	26.86
H 24	19.80 (26.42)	12.70 (20.88)	27.85 (31.85)	15.15 (22.90)	14.55 (22.42)	15.92 (23.51)	18.28
LE 415	22.16 (28.08)	18.31 (25.38)	39.33 (38.84)	29.83 (33.10)	29.00 (32.58)	11.66 (19.97)	27.73
Column Mean (hybrids)	30.18	22.14	37.41	30.94	26.00	33.96	
Mean hybrids	:	30.11		SE	:	3.53	
Mean parents	:	21.37		CD (0.05)	:	14.32	
Susceptible check	:	60.03		CD (0.01)	:	19.21	

^a includes PDI due to combined infection of both the viruses, if any.

Table 26a: *Per se* performance of parents and hybrids for TLCV (per cent) incidence at 75 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	9.35 (17.80)	23.33 (28.88)	26.79 (31.17)	7.14 (15.50)	0.00 (0.58)	0.00 (0.58)	11.45
LCR 1	25.00 (30.00)	11.26 (19.61)	25.00 (30.00)	3.57 (10.89)	5.56 (13.64)	12.88 (21.03)	14.40
LCR 3	14.28 (22.20)	25.00 (30.0)	11.31 (19.65)	5.00 (12.92)	14.58 (22.45)	14.28 (22.21)	14.63
CLN 2123A	0.00 (0.58)	5.56 (13.64)	20.0 (26.57)	5.00 (12.92)	13.81 (21.82)	5.0 (12.92)	8.87
H 24	14.85 (22.64)	5.56 (13.64)	17.90 (25.03)	4.55 (12.32)	11.21 (19.56)	9.09 (17.55)	10.39
LE 415	17.31 (24.59)	7.28 (15.65)	21.0 (27.27)	7.89 (16.31)	20.07 (26.62)	19.17 (25.97)	14.71
Column Mean (hybrids)	14.29	13.35	22.14	5.63	10.80	8.25	
Mean hybrids	:	12.41		SE	:	4.70	
Mean parents	:	11.22		CD (0.05)	:	19.47	
Susceptible check	:	31.78		CD (0.01)	:	26.12	

Table 26b: *Per se* performance of parents and hybrids for Tv (per cent) incidence at 75 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	18.37 (25.38)	26.67 (31.09)	22.59 (28.38)	51.43 (45.82)	23.34 (28.89)	37.5 (37.76)	32.31
LCR 1	25.0 (30.00)	24.21 (29.47)	18.06 (25.15)	18.57 (25.53)	25.00 (30.00)	17.17 (24.48)	20.76
LCR 3	32.07 (34.49)	37.50 (37.76)	23.52 (29.01)	26.67 (31.09)	25.00 (30.0)	53.70 (47.12)	34.99
CLN 2123A	23.37 (28.91)	17.41 (24.66)	30.00 (33.21)	28.85 (32.49)	31.43 (34.10)	36.67 (37.27)	27.78
H 24	12.08 (20.34)	21.83 (27.85)	13.07 (21.19)	14.65 (22.50)	19.09 (25.91)	22.14 (28.07)	16.75
LE 415	17.31 (24.59)	21.49 (27.62)	24.33 (29.55)	27.19 (31.43)	36.14 (36.95)	8.30 (16.74)	25.29
Column Mean (hybrids)	21.97	24.98	21.61	27.70	28.18	33.44	
Mean hybrids	:	26.31		SE	:	3.30	
Mean parents	:	20.39		CD (0.05)	:	13.37	
Susceptible check	:	37.09		CD (0.01)	:	17.94	

Table 26c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 75 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	27.72 (31.77)	58.00 (45.00)	49.40 (44.66)	58.57 (49.93)	23.34 (28.89)	37.50 (37.76)	43.76
LCR 1	50.00 (45.0)	35.47 (36.55)	43.05 (41.01)	22.14 (28.07)	30.56 (33.56)	30.05 (33.24)	35.16
LCR 3	46.35 (42.91)	67.50 (55.24)	34.83 (36.17)	31.67 (34.25)	39.58 (38.99)	67.98 (55.54)	50.62
CLN 2123A	23.37 (28.91)	22.96 (28.63)	50.00 (45.00)	33.75 (35.52)	45.23 (42.26)	41.67 (40.20)	36.65
H 24	26.93 (31.26)	27.38 (31.55)	30.97 (33.81)	19.19 (25.98)	30.30 (33.40)	31.25 (34.00)	27.14
LE 415	34.61 (36.04)	28.77 (32.44)	45.33 (42.32)	35.08 (36.32)	56.20 (48.56)	27.47 (31.61)	40.00
Column Mean (hybrids)	36.25	39.32	43.75	33.33	39.98	41.69	
Mean hybrids	:	38.89		SE	:	1.42	
Mean parents	:	31.59		CD (0.05)	:	6.375	
Susceptible check	:	68.87		CD (0.01)	:	8.55	

^a includes PDI due to combined infection of both the viruses, if any.

Table 27a: *Per se* performance of parents and hybrids for TLCV (per cent) incidence at 90 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	14.02 (21.99)	23.50 (29.00)	26.79 (31.17)	17.14 (24.46)	0.00 (0.68)	0.00 (0.58)	13.49
LCR 1	25.00 (30.00)	11.26 (19.61)	25.12 (30.08)	8.57 (17.02)	5.56 (13.64)	12.88 (21.03)	15.43
LCR 3	14.28 (22.21)	25.00 (30.00)	11.31 (19.65)	10.00 (18.43)	14.58 (22.44)	14.28 (22.21)	15.63
CLN 2123A	3.57 (10.89)	5.56 (13.64)	20.00 (26.57)	5.00 (12.92)	20.92 (27.22)	5.00 (12.92)	11.01
H 24	14.86 (22.67)	9.72 (18.17)	17.90 (25.03)	7.32 (15.70)	13.49 (21.55)	9.09 (17.55)	11.78
LE 415	19.23 (26.01)	7.28 (15.65)	22.67 (28.43)	7.84 (16.26)	27.31 (31.51)	19.17 (25.97)	16.87
Column Mean (hybrids)	15.39	14.21	22.50	10.17	13.67	8.25	
Mean hybrids	:	14.03		SE	:	3.91	
Mean parents	:	12.38		CD (0.05)	:	16.21	
Susceptible check	:	32.64		CD (0.01)	:	21.75	

Table 27b: *Per se* performance of parents and hybrids for Tv (per cent) incidence at 90 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	18.37 (25.38)	27.12 (31.38)	22.62 (28.40)	51.43 (45.82)	33.33 (35.26)	37.50 (37.76)	34.4
LCR 1	25.00 (30.00)	26.38 (30.90)	18.06 (25.15)	18.56 (25.52)	25.00 (30.00)	21.97 (27.95)	21.72
LCR 3	32.14 (34.54)	37.50 (37.76)	23.52 (29.01)	26.65 (31.08)	25.00 (30.00)	53.57 (47.05)	34.97
CLN 2123A	23.22 (28.81)	17.41 (24.66)	30.00 (33.21)	28.89 (32.51)	31.43 (34.10)	36.67 (37.27)	27.75
H 24	14.86 (22.67)	21.83 (27.85)	13.07 (21.19)	14.65 (22.50)	19.09 (25.91)	24.71 (29.81)	17.82
LE 415	21.15 (27.38)	21.47 (27.60)	24.33 (29.55)	27.19 (31.43)	36.14 (36.95)	8.33 (16.78)	26.06
Column Mean (hybrids)	23.27	25.07	21.62	27.70	30.18	34.88	
Mean hybrids	:	27.12		SE	:	2.68	
Mean parents	:	20.76		CD (0.05)	:	11.86	
Susceptible check	:	38.70		CD (0.01)	:	15.91	

Table 27c: *Per se* performance of parents and hybrids for total disease incidence^a (per cent) at 90 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	32.39 (34.69)	50.62 (45.36)	49.40 (44.66)	68.57 (55.90)	33.33 (35.26)	37.50 (37.76)	47.88
LCR 1	50.00 (45.00)	37.64 (37.84)	43.17 (41.07)	27.14 (31.40)	30.56 (33.56)	34.85 (36.18)	37.14
LCR 3	46.42 (42.95)	67.50 (55.24)	34.83 (36.17)	36.65 (37.26)	42.71 (40.81)	67.85 (55.46)	52.23
CLN 2123A	26.79 (31.17)	22.96 (28.63)	50.00 (45.00)	33.89 (35.60)	52.35 (46.35)	41.67 (40.20)	38.75
H 24	29.71 (33.03)	31.55 (34.17)	30.97 (33.81)	21.97 (27.95)	32.58 (34.81)	34.00 (35.67)	29.64
LE 415	40.38 (39.45)	28.75 (32.42)	47.00 (43.28)	35.08 (36.32)	63.45 (52.80)	27.50 (31.63)	42.93
Column Mean (hybrids)	38.66	40.28	44.11	37.88	44.48	43.17	
Mean hybrids	:	41.43		SE	:	1.11	
Mean parents	:	33.14		CD (0.05)	:	6.70	
Susceptible check	:	71.34		CD (0.01)	:	8.99	

^a includes PDI due to combined infection of both the viruses, if any.

Table 28: Co-efficient of infection (CI) for TLCV incidence at 75 DAT in the season II

Parents	LCR 9	LCR 1	LCR 3	CLN 2123A	H 24	LE 415	Row mean (hybrids)
LCR 9	4.13	17.42	19.64	0.89	0.00	0.00	7.59
LCR 1	12.5	1.41	7.29	0.45	0.70	1.58	4.50
LCR 3	7.14	18.30	2.46	0.63	5.47	7.14	7.74
CLN 2123A	0.00	0.70	2.50	0.96	1.73	0.63	1.11
H 24	2.75	0.70	4.08	0.57	2.52	1.14	1.85
LE 415	13.25	0.92	7.33	3.32	6.85	4.48	6.33
Column Mean (hybrids)	7.13	7.61	8.17	1.17	2.95	2.10	
Mean hybrids	:	4.85		SE	:	1.40	
Mean parents	:	2.66		CD (0.05)	:	5.69	
				CD (0.01)	:	7.64	

Table 1a: Analysis of variance for different characters in the season I

	Characters	Source	df	SS	MSS	F value
1.	Plant height	Genotype	35	14528.42	415.09	118.55**
		Error	35	122.54	3.50	
2.	No. of branches per plant	Genotype	35	75.15	2.147	19.54**
		Error	35	3.84	0.109	
3.	Days to 50 per cent flowering	Genotype	35	1109.38	31.70	26.81**
		Error	35	41.38	1.18	
4.	No. of fruits per plant	Genotype	35	17042.12	486.92	54.88**
		Error	35	310.53	8.87	
5.	Average fruit weight	Genotype	35	21626.37	617.90	1869.43**
		Error	35	11.57	0.33	
6.	Yield per plant	Genotype	35	22.99	0.66	28.63**
		Error	35	0.80	0.023	
7.	TSS	Genotype	35	10.88	0.310	16.21**
		Error	35	0.671	0.019	
8.	Acidity	Genotype	35	0.175	0.005	40.71**
		Error	35	0.004	0.0001	
9.	Ascorbic acid	Genotype	35	965.88	27.60	12.47**
		Error	35	77.49	2.21	
10.	Lycopene	Genotype	35	49.15	1.40	99.16**
		Error	35	0.50	0.014	

11.	Total phenol	Genotype	35	64337.37	1838.21	14.69**
		Error	35	4379.62	125.13	
12.	OD phenol	Genotype	35	131867.98	3767.66	43.90**
		Error	35	3003.71	85.82	
14.	Tv PDI at 15 DAT	Genotype	35	3497.28	99.92	4.09**
		Error	35	856.02	24.46	
15.	Total PDI at 15 DAT	Genotype	35	3497.28	99.92	4.09**
		Error	35	856.02	24.41	
16.	TLCV PDI at 30 DAT	Genotype	35	2720.12	77.72	38.32**
		Error	35	70.99	2.03	
17.	Tv PDI at 30 DAT	Genotype	35	3106.61	88.76	4.57**
		Error	35	678.89	19.39	
18.	Total PDI at 30 DAT	Genotype	35	2539.74	72.56	5.44**
		Error	35	466.63	12.33	
19.	TLCV PDI at 45 DAT	Genotype	35	3853.16	110.09	3.24**
		Error	35	1188.19	33.95	
20.	Tv PDI at 45 DAT	Genotype	35	3486.77	99.62	6.19**
		Error	35	562.85	16.08	
21.	Total PDI at 45 DAT	Genotype	35	2480.88	70.88	4.75**
		Error	35	521.85	14.91	
22.	TLCV PDI at 60 DAT	Genotype	35	7928.00	226.51	6.47**
		Error	35	1224.66	34.99	
23.	Tv PDI at 60 DAT	Genotype	35	4850.48	138.59	12.58**

		Error	35	385.42	11.01	
24.	Total PDI at 60 DAT	Genotype	35	4135.88	118.17	5.56**
		Error	35	743.25	21.24	
25.	TLCV PDI at 75 DAT	Genotype	35	7935.52	226.73	5.92**
		Error	35	1340.49	38.30	
26.	Tv PDI at 75 DAT	Genotype	35	5656.84	161.62	5.91**
		Error	35	957.88	27.37	
27.	Total PDI at 75 DAT	Genotype	35	4424.25	126.41	4.38**
		Error	35	1009.55	28.85	
28.	TLCV PDI at 90 DAT	Genotype	35	8604.43	245.84	5.53**
		Error	35	1554.45	44.41	
29.	Tv PDI at 90 DAT	Genotype	35	5350.35	152.87	6.73**
		Error	35	765.41	22.73	
30.	Total PDI at 90 DAT	Genotype	35	5096.66	145.62	4.81**
		Error	35	1059.86	30.28	
31.	CI for TLCV at 75 DAT	Genotype	35	2066.77	59.05	3.5***
		Error	35	575.44	16.44	

Table 1b: Analysis of variance for different characters in the season II

	Characters	Source	df	SS	MSS	F value
1.	Tv PDI at 15 DAT	Genotype	35	761.12	21.75	1.05**
		Error	35	721.55	20.62	
2.	Total PDI at 15 DAT	Genotype	35	3763.71	107.53	3.37**
		Error	35	1113.96	31.827	
3.	Total PDI at 15 DAT	Genotype	35	3901.57	111.50	2.67**
		Error	35	1461.75	41.76	
4.	TLCV PDI at 30 DAT	Genotype	35	2561.30	73.18	4.78**
		Error	35	535.40	15.30	
5.	Tv PDI at 30 DAT	Genotype	35	4120.67	117.73	3.47**
		Error	35	1187.56	33.93	
6.	Total PDI at 30 DAT	Genotype	35	4955.74	141.59	4.04**
		Error	35	1225.66	35.02	
7.	TLCV PDI at 45 DAT	Genotype	35	3779.11	107.97	3.62**
		Error	35	1042.88	29.80	
8.	Tv PDI at 45 DAT	Genotype	35	2980.16	85.04	4.21**
		Error	35	707.11	20.20	
9.	Total PDI at 45 DAT	Genotype	35	3252.81	92.94	6.05***
		Error	35	537.68	15.36	
10.	TLCV PDI at 60 DAT	Genotype	35	5224.38	149.27	4.58**
		Error	35	1140.48	32.58	
11.	Tv PDI at 60 DAT	Genotype	35	3446.76	98.48	14.61**
		Error	35	747.48	21.36	
12.	Total PDI at 60 DAT	Genotype	35	4575.63	130.73	5.26**
		Error	35	870.44	24.87	
13.	TLCV PDI at 75 DAT	Genotype	35	5397.86	154.22	3.49**
		Error	35	1548.41	44.24	
14.	Tv PDI at 75 DAT	Genotype	35	2846.30	81.32	3.73**
		Error	35	763.81	21.82	
15.	Total PDI at 75 DAT	Genotype	35	3916.31	111.89	27.58**
		Error	35	142.02	4.06	

16.	TLCV PDI at 90 DAT	Genotype	35	4679.66	133.70	4.37**
		Error	35	1069.78	30.57	
17.	Tv PDI at 90 DAT	Genotype	35	2707.71	77.33	5.38**
		Error	35	502.71	14.36	
18.	Total PDI at 90 DAT	Genotype	35	3672.15	104.96	42.29**
		Error	35	86.84	2.48	
19.	CI for TLCV at 75 DAT	Genotype	35	2022.45	57.78	14.69**
		Error	35	137.68	3.93	

** Significance at 1 per cent level

Table 60: Analysis of variance for combining ability (mean squares) in the seasons I and II

	Characters	Season	GCA	SCA	Reciprocal	GCA/SC A
1.	Plant height	I	652.55**	165.99**	100.72**	3.77
2.	Branches per plant	I	2.28**	1.14**	0.60**	2.00
3.	Days to 50 % flowering	I	50.72**	10.56**	9.52**	4.80
4.	No. of fruits per plant	I	701.77**	167.84**	166.31**	4.18
5.	Average fruit weight	I	1660.76**	109.77**	57.52**	15.13
6.	Yield per plant	I	0.32**	0.46**	0.20**	0.70
7.	TSS	I	0.11**	0.15**	0.18**	0.73
8.	Acidity	I	0.012**	0.001**	0.001**	12.68
9.	Ascorbic acid	I	6.31**	10.61**	19.49**	0.59
10.	Lycopene	I	1.02**	0.67**	0.63**	1.52
11.	Total phenol	I	1759.90**	1014.75**	543.23**	1.73
12.	OD phenol	I	3113.93**	2271.27**	1086.38**	1.37
13.	TLCV at 75 DAT	I	310.71**	117.74**	43.20*	2.64
14.	Tv at 75 DAT	I	166.61**	48.00**	85.03**	3.47
15.	Total PDI at 75 DAT	I	41.33*	57.25**	76.45**	0.72
16.	TLCV at 75 DAT	II	171.69**	56.10*	66.60**	3.06
17.	Tv at 75 DAT	II	28.99**	38.45**	46.76**	0.75
18.	Total PDI at 75 DAT	II	80.20**	54.54**	49.27**	1.47

** Significance at 1 per cent level

* Significance at 5 per cent level

Table 34 : Effect of different treatments on various characters of the hybrid H 24 x CLN 2123A

Treatments	Particulars	Plant height (cm)	Bran ches /plant	Fruit No.	Av. fr. wt. (g)	Yield/ plant (kg)	Polar diameter (cm)	Equat orial diameter (cm)	Pericarp thickness (cm)	Septa thickness (cm)	TSS (°Brix)	Total acidity (%)
T ₁	Nipping	65.47	7.67	51.67	44.00	2.27	4.34	5.01	0.48	0.50	5.38	0.69
T ₂	Flower thinning	93.73	8.92	47.62	53.55	2.55	4.03	4.61	0.46	0.45	5.48	0.50
T ₃	Truss thinning	86.13	9.18	33.78	57.23	1.93	4.57	4.92	0.44	0.51	5.32	0.67
T ₄	Alt. truss thinning	90.04	9.00	35.08	54.62	1.92	4.38	4.90	0.58	0.62	5.25	0.65
T ₅	Control	90.67	8.89	76.58	40.67	3.11	3.85	4.19	0.42	0.41	5.97	0.69

Table 35: Heterosis (per cent) for plant height and number of branches in the season I

Cross	Plant height			No. of branches per plant		
	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	-17.52**	-20.85**	-35.94**	-17.04**	-25.33**	-25.33**
LCR 9 x LCR 3	-2.94	-10.96**	-33.75**	-11.94**	-21.33**	-21.33**
LCR 9 x CLN 2123A	-5.33**	-17.45**	-17.45**	13.04**	4.00	4.00
LCR 9 x H 24	20.95**	10.03**	-18.14**	11.89**	6.67*	6.67*
LCR 9 x LE 415	8.25**	-4.78**	-29.16**	-4.90	-9.33**	-9.33**
LCR 1 x LCR 9	-19.07**	-22.34**	-37.14**	-27.41**	-34.67**	-34.67**
LCR 1 x LCR 3	-11.88**	-22.13**	-36.97**	14.29**	13.33**	-9.33**
LCR 1 x CLN 2123A	-8.38**	-17.11**	-17.11**	13.82**	11.11**	-6.67*
LCR 1 x H 24	17.48**	2.98	-16.65**	20.31**	13.24**	2.67
LCR 1 x LE 415	13.12**	-3.97**	-22.27**	-9.37**	-14.71**	-22.67**
LCR 3 x LCR 9	-14.05**	-21.14**	-41.33**	-32.84**	-40.00**	-40.00**
LCR 3 x LCR 1	-8.67**	-19.29**	-34.67**	-9.24*	-10.00**	-28.00**
LCR 3 x CLN 2123A	-2.62	-21.07**	-21.07**	25.00**	21.03**	1.67
LCR 3 x H 24	0.00	-0.92	-38.46**	-5.51	-11.76**	-20.00**
LCR 3 x LE 415	-10.75**	-14.79**	-47.07**	-18.11**	-23.53**	-30.67**
CLN 2123A x LCR 9	32.19**	15.27**	15.27**	24.64**	14.67**	14.67**
CLN 2123A x LCR 1	-17.51**	-25.37**	-25.37**	25.20**	22.22**	2.67
CLN 2123A x LCR 3	-14.59**	-7.12**	-7.12**	0.00	-3.17	-18.67**
CLN 2123A x H 24	7.31**	-13.63**	-13.63**	1.15	-2.57	-11.67**
CLN 2123A x LE 415	-21.50**	-38.58**	-38.58**	-14.50**	-17.65**	-25.33**
H 24 x LCR 9	57.42**	43.21**	6.54**	32.87**	26.67**	26.67**

H 24 x LCR 1	-0.32	-12.62**	-29.28**	3.12	-2.94	-12.00**
H 24 x LCR 3	-3.17	-4.07	-40.41**	8.66**	1.47	-8.00**
H 24 x CLN 2123A	48.36**	19.40**	19.40**	9.92**	5.88	-4.00
H 24 x LE 415	64.42**	58.38**	-3.44*	2.94	2.94	-6.67*
LE 415 x LCR 9	14.21**	0.46	-25.26**	0.70	-4.00	-4.00
LE 415 x LCR 1	22.14**	3.69**	-16.07**	14.06**	7.35*	-2.67
LE 415 x LCR 3	-7.65**	-11.83**	-45.24**	-2.36	-8.82**	-17.33**
LE 415 x CLN 2123A	-7.56**	-27.67**	-27.67**	7.48*	3.53	-6.13*
LE 415 x H 24	24.58**	20.01**	-26.84**	5.15	5.15	-4.67

** Significance at 1% level

* Significance at 5% level

Table 36: Heterosis (per cent) days to 50 per cent flowering and number of fruits in the season I

Cross	Days to 50% flowering			No. of fruits per plant		
	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	-4.70*	-2.74	36.54**	25.65**	20.81**	-66.91**
LCR 9 x LCR 3	6.25**	30.77**	30.77**	-24.23**	-52.54**	-52.54**
LCR 9 x CLN 2123A	14.93**	32.76**	48.08**	12.28*	-16.78**	-56.38**
LCR 9 x H 24	13.18**	37.74**	40.38**	10.19	-17.46**	-58.31**
LCR 9 x LE 415	2.04	5.63**	44.23**	63.82**	39.93**	-50.06**
LCR 1 x LCR 9	-16.78**	-15.07**	19.23**	9.65	5.43	-71.13**
LCR 1 x LCR 3	-15.20**	1.92	1.92	-38.13**	-60.59**	-60.59**
LCR 1 x CLN 2123A	-8.40**	3.45	15.38**	12.42*	-14.42**	-55.14**
LCR 1 x H 24	-4.76*	13.21**	15.38**	-1.98	-24.54**	-61.71**
LCR 1 x LE 415	-13.89**	-12.68**	19.23**	63.85**	44.79**	-48.33**
LCR 3 x LCR 9	-4.69*	-17.31**	-17.31**	-23.24**	-51.92**	-51.92**
LCR 3 x LCR 1	-2.40	-17.31**	-17.31**	-23.93**	-51.55**	-51.55**
LCR 3 x CLN 2123A	25.45**	32.69**	32.69**	-15.77**	-35.81**	-35.81**
LCR 3 x H 24	-0.95	0.00	0.00	-35.55**	-51.43**	-51.43**
LCR 3 x LE 415	-13.82**	1.92	1.92	-18.72**	-44.86**	-44.86**
CLN 2123A x LCR 9	-2.99	12.07**	25.00**	-2.31	-27.60**	-62.05**
CLN 2123A x LCR 1	14.50**	29.31**	44.23**	38.82**	5.67	-44.61**
CLN 2123A x LCR 3	27.27**	34.62**	34.62**	-43.50**	-56.94**	-56.94**
CLN 2123A x H 24	31.53**	37.74**	40.38**	18.68**	16.78**	-38.79**
CLN 2123A x LE 415	8.53**	17.14**	34.66**	-15.62**	-28.58**	-62.57**
H 24 x LCR 9	0.78	22.64**	25.00**	91.36**	43.35**	-27.26**

H 24 x LCR 1	0.00	18.87**	21.15**	19.68**	-7.86	-53.25**
H 24 x LCR 3	8.57**	9.62**	9.62**	3.08	-22.30**	-22.30**
H 24 x CLN 2123A	-2.70	1.89	3.85	91.47**	88.42**	-1.24
H 24 x LE 415	1.61	18.87**	21.15**	122.22**	89.26**	-3.97
LE 415 x LCR 9	-3.40	0.00	36.54**	69.92**	45.14**	-48.20**
LE 415 x LCR 1	-12.50**	-11.27**	21.15**	83.10**	61.81**	-42.26**
LE 415 x LCR 3	-15.45**	0.00	0.00	35.53**	-8.05**	-8.05**
LE 415 x CLN 2123A	10.08**	22.41**	36.54**	41.35**	18.79**	-37.73**
LE 415 x H 24	17.74**	37.74**	40.38**	27.41**	8.51	-44.94**

** Significance at 1% level

* Significance at 5% level

Table 37. Heterosis (per cent) for average fruit weight and yield per plant in the season I

Cross	Average fruit weight			Yield per plant		
	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	-35.06**	-46.95**	-46.95**	-19.23**	-35.98**	-40.79**
LCR 9 x LCR 3	-10.14**	-34.04**	-58.19**	-13.17**	-33.08**	-33.08**
LCR 9 x CLN 2123A	9.40**	-9.22**	-42.45**	30.40**	12.98**	-16.54**
LCR 9 x H 24	0.00	-23.40**	-51.45**	22.48**	18.51**	-31.39**
LCR 9 x LE 415	13.65**	-20.57**	-49.65**	100.44**	56.60**	-15.23**
LCR 1 x LCR 9	-10.57**	-26.94**	-26.94**	-2.31	-22.56**	-28.38**
LCR 1 x LCR 3	-52.43**	-69.16**	-69.16**	-57.42**	-59.02**	-59.02**
LCR 1 x CLN 2123A	-11.87**	-37.51**	-37.51**	13.67**	2.24	-5.45
LCR 1 x H 24	-6.53**	-37.51**	-37.51**	7.50	-12.60**	-19.17**
LCR 1 x LE 415	-9.53**	-43.35**	-43.35**	60.24**	6.50	-1.50
LCR 3 x LCR 9	7.25**	-21.28**	-50.10**	4.88	-19.17**	-19.17**
LCR 3 x LCR 1	-27.55**	-53.03**	-53.03**	-19.92**	-22.93**	-22.93**
LCR 3 x CLN 2123A	-33.08**	-42.80**	-76.08**	-40.54**	-48.31**	-48.31**
LCR 3 x H 24	7.80**	1.33	-65.83**	-29.05**	-43.98**	-43.98**
LCR 3 x LE 415	-12.86**	-19.39**	-76.08**	-32.28**	-55.83**	-55.83**
CLN 2123A x LCR 9	24.79**	3.55**	-34.36**	30.98**	13.49**	-16.17**
CLN 2123A x LCR 1	-30.25**	-50.55**	-50.55**	11.19**	0.00	-7.52*
CLN 2123A x LCR 3	5.66**	-9.68**	-62.24**	-35.35**	-43.80**	-43.80**
CLN 2123A x H 24	23.81**	11.83**	-53.24**	46.65**	30.79**	-3.38
CLN 2123A x LE 415	14.02**	-8.60**	-61.79**	-7.39	-34.61**	-51.69**
H 24 x LCR 9	-10.19**	-31.21**	-56.39**	90.94**	84.74**	6.95

H 24 x LCR 1	-10.90**	-40.43**	-40.43**	25.00**	1.63	-6.02
H 24 x LCR 3	4.96**	-1.33	-66.73**	10.48*	-12.78**	-12.78**
H 24 x CLN 2123A	-7.02**	-16.02**	-64.89**	77.45**	58.52**	17.11**
H 24 x LE 415	28.15**	12.00**	-62.24**	176.60**	111.04**	22.18*
LE 415 x LCR 9	-0.56	-30.50**	-55.94**	82.22**	42.36**	-22.93**
LE 415 x LCR 1	-11.68**	-44.70**	-44.70**	67.58**	11.38**	3.01
LE 415 x LCR 3	-8.93**	-15.76**	-75.00**	18.73**	-22.56**	-22.56**
LE 415 x CLN 2123A	11.07**	-10.97**	-62.77**	50.27**	6.11	-21.62**
LE 415 x H 24	46.45**	28.00**	-56.84**	80.00**	37.34**	-20.49**

** Significance at 1% level

* Significance at 5% level

Table 38: Heterosis (per cent) for TSS and acidity in the season I

Cross	TSS			Acidity		
	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	2.67	-3.03	-15.42**	16.56**	9.20**	-21.49**
LCR 9 x LCR 3	9.92**	4.06	-9.69**	1.52	-17.36**	-17.36**
LCR 9 x CLN 2123A	1.76	-8.78**	-10.79**	10.05**	-7.96**	-14.05**
LCR 9 x H 24	-9.73**	-18.68**	-21.37**	12.50**	-1.00	-18.18**
LCR 9 x LE 415	-4.22	-14.98**	-14.98**	6.29**	-6.06**	-23.14**
LCR 1 x LCR 9	31.02**	23.74**	7.93**	10.43**	3.45*	-25.62**
LCR 1 x LCR 3	19.49**	19.19**	3.96*	-2.88*	-16.53**	-16.53**
LCR 1 x CLN 2123A	7.62**	1.80	-0.44	8.00**	-4.42**	-10.74**
LCR 1 x H 24	-6.11**	-10.71**	-13.66**	17.65**	10.00**	-9.09**
LCR 1 x LE 415	-6.59**	-12.56**	-12.56**	3.23*	-3.03*	-20.66**
LCR 3 x LCR 9	-2.68	-7.87**	-20.04**	11.68**	-9.09**	-9.09**
LCR 3 x LCR 1	1.27	1.01	-11.89**	-2.88**	-16.53**	-16.53**
LCR 3 x CLN 2123A	-8.11**	-13.29**	-15.20**	5.98**	2.48*	2.48*
LCR 3 x H 24	5.64**	0.23	-3.08	-0.45	-9.09**	-9.09**
LCR 3 x LE 415	-8.49**	-14.54**	-14.54**	-5.45**	-14.05**	-14.05**
CLN 2123A x LCR 9	-0.75	-11.04**	-13.00**	-1.59	-17.70**	-23.14**
CLN 2123A x LCR 1	12.62**	6.53**	4.19*	-5.00**	-15.93**	-21.49**
CLN 2123A x LCR 3	10.50**	4.28*	1.98	1.71	-1.65	-1.65
CLN 2123A x H 24	-6.46**	-6.98**	-9.03**	-4.23**	-9.73**	-15.70**
CLN 2123A x LE 415	-26.73**	-27.53**	-27.53**	-0.94	-7.08**	-13.22**
H 24 x LCR 9	12.01**	0.91	-2.42	-4.55**	-16.00**	-30.58**

H 24 x LCR 1	-6.11**	-10.71**	-13.66**	5.88**	-1.00	-18.18**
H 24 x LCR 3	4.44*	-0.91	-4.19*	-1.36	-9.92**	-9.92**
H 24 x CLN 2123A	-2.15	-2.70	-4.85*	-4.23**	-9.73**	-15.70**
H 24 x LE 415	-11.09**	-12.56**	-12.56**	-6.53**	-7.00**	-23.14**
LE 415 x LCR 9	10.67**	-1.76	-1.76	2.86	-9.09**	-25.62**
LE 415 x LCR 1	-20.71**	-25.77**	-25.77**	7.53**	1.01	-17.36**
LE 415 x LCR 3	-10.14**	-16.08**	-16.08**	-9.09**	-17.36**	-17.36**
LE 415 x CLN 2123A	-2.45	-3.52	-3.52	-9.43**	-15.04**	-20.66**
LE 415 x H 24	-16.01**	-17.40**	-17.40**	7.54**	7.00**	-11.57**

** Significance at 1% level

* Significance at 5% level

Table 39: Heterosis (per cent) for ascorbic acid and lycopene in the season I

Cross	Ascorbic acid			Lycopene		
	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	14.88**	12.49**	-6.90*	7.89**	-5.46*	-49.13**
LCR 9 x LCR 3	-0.01	-10.35**	-10.35**	43.36**	25.26**	-32.60**
LCR 9 x CLN 2123A	14.88**	12.49**	-6.90*	-7.62**	-12.04**	-47.66**
LCR 9 x H 24	21.74**	21.74**	-3.44	-31.82**	-47.57**	-47.57**
LCR 9 x LE 415	-8.00*	-14.81**	-20.69**	2.14	1.71	-45.27**
LCR 1 x LCR 9	19.13**	16.64**	-3.45	51.70**	32.94**	-28.47**
LCR 1 x LCR 3	-24.71**	-31.20**	-31.20**	23.09**	22.68**	-50.32**
LCR 1 x CLN 2123A	16.66**	16.66**	-3.44	38.84**	16.67**	-30.58**
LCR 1 x H 24	31.90**	29.15**	6.90*	-24.18**	-46.74**	-46.74**
LCR 1 x LE 415	-17.75**	-22.31**	-27.67**	58.51**	39.41**	-25.62**
LCR 3 x LCR 9	23.08**	10.35**	10.35**	8.01**	-5.63*	-49.22**
LCR 3 x LCR 1	16.97**	6.90*	6.90*	-0.80	-1.13	-59.96**
LCR 3 x CLN 2123A	-9.43**	-17.23**	-17.23**	78.82**	49.85**	-10.84**
LCR 3 x H 24	3.85	-6.90*	-6.90*	-4.52*	-33.06**	-33.06**
LCR 3 x LE 415	-14.27**	-17.23**	-17.23**	-9.13**	-20.31**	-57.48**
CLN 2123A x LCR 9	36.16**	33.32**	10.35**	18.48**	12.81**	-32.87**
CLN 2123A x LCR 1	16.66**	16.66**	-3.44	-7.99**	-22.69**	-53.99**
CLN 2123A x LCR 3	5.66	-3.44	-3.44	71.27**	43.52**	-14.60**
CLN 2123A x H 24	14.88**	12.49**	-6.90*	-9.04**	-27.46**	-27.46**
CLN 2123A x LE 415	5.88	0.00	-6.90*	-22.86**	-26.85**	-56.47**
H 24 x LCR 9	-8.81*	-8.81*	-27.67**	-20.24**	-38.66**	-38.66**

H 24 x LCR 1	14.88**	12.49**	-6.90*	26.67	-11.02**	-11.02**
H 24 x LCR 3	-7.68*	-17.23**	-17.23**	-28.88**	-50.14**	-50.14**
H 24 x CLN 2123A	19.15**	16.66**	-3.44	-2.71	-22.41**	-22.41**
H 24 x LE 415	-8.00*	-14.81**	-20.69**	-7.07**	-28.74**	-28.74**
LE 415 x LCR 9	23.99**	14.81**	6.90*	34.02**	33.45**	-28.19**
LE 415 x LCR 1	7.06*	1.11	-5.86*	21.33**	6.71*	-43.07**
LE 415 x LCR 3	7.15*	3.45	3.45	62.12**	42.17**	-24.15**
LE 415 x CLN 2123A	-9.81**	-14.81**	-20.69**	31.98**	25.15**	-25.53**
LE 415 x H 24	-3.99	-11.10**	-17.23**	-34.37**	-49.68**	-49.68**

** Significance at 1% level

* Significance at 5% level

Table 40: Heterosis (per cent) for total and OD phenol content in the leaves in the season I

Cross	Total phenol			OD phenol		
	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	3.44	-4.21	-12.88**	20.09**	0.56	-46.77**
LCR 9 x LCR 3	0.45	-4.14	-18.24**	-3.52	-19.38**	-57.10**
LCR 9 x CLN 2123A	-0.43	-7.49**	-16.46**	1.06	-24.99**	-44.70**
LCR 9 x H 24	4.85	-4.21	-10.26**	21.56**	-7.75*	-36.38**
LCR 9 x LE 415	-5.22*	-15.89**	-15.89**	-35.12**	-55.98**	-55.98**
LCR 1 x LCR 9	2.92	-4.69	-13.32**	58.28**	32.53**	-29.84**
LCR 1 x LCR 3	-28.57**	-30.79**	-37.05**	-26.22**	-26.41**	-60.84**
LCR 1 x CLN 2123A	-19.52**	-19.81**	-27.07**	-67.19**	-71.81**	-79.22**
LCR 1 x H 24	-0.04	-1.50	-7.73**	10.28**	-2.54	-32.79**
LCR 1 x LE 415	1.25	-3.33	-3.33	-24.75**	-42.46**	-42.46**
LCR 3 x LCR 9	3.16	-1.55	-16.63**	-3.36	-19.25**	-57.03**
LCR 3 x LCR 1	-14.91**	-17.56**	-25.02**	-30.18**	-30.36**	-62.94**
LCR 3 x CLN 2123A	-24.68**	-26.77**	-33.87**	-52.08**	-58.75**	-69.59**
LCR 3 x H 24	9.60**	4.69	-1.93	-6.53	-17.20**	-42.90**
LCR 3 x LE 415	-25.04**	-30.55**	-30.55**	-59.99**	-69.35**	-69.35**
CLN 2123A x LCR 9	-8.90**	-15.37**	-23.57**	-3.25	-28.19**	-47.06**
CLN 2123A x LCR 1	-20.28**	-20.56**	-27.75**	-34.58**	-43.80**	-58.57**
CLN 2123A x LCR 3	-3.06	-5.76*	-14.89**	-2.80	-16.32**	-38.31**
CLN 2123A x H 24	-14.20**	-15.74**	-21.07**	-25.97**	-28.36**	-47.19**
CLN 2123A x LE 415	-23.56**	-27.26**	-27.26**	-51.29**	-57.69**	-57.69**
H 24 x LCR 9	8.86**	-0.54	-6.83**	-1.50	-25.24**	-48.44**

H 24 x LCR 1	4.83	3.30	-3.24	49.50**	32.13**	-8.88**
H 24 x LCR 3	8.74**	3.87	-2.70	13.25**	0.32	-30.82**
H 24 x CLN 2123A	9.31**	7.35**	0.56	-16.50**	-19.19**	-40.43**
H 24 x LE 415	-5.61*	-8.60**	-8.60**	-22.62**	-34.63**	-34.63**
LE 415 x LCR 9	-15.13**	-24.68**	-24.68**	-17.45**	-43.99**	-43.99**
LE 415 x LCR 1	-4.28	-8.61**	-8.61**	-32.22**	-48.17**	-48.17**
LE 415 x LCR 3	-11.33**	-17.85**	-17.85**	-16.54**	-36.07**	-36.07**
LE 415 x CLN 2123A	-9.89**	-14.25**	-14.25**	-33.07**	-41.87**	-41.87**
LE 415 x H 24	-21.77**	-24.24**	-24.24**	-42.37**	-51.32**	-51.32**

** Significance at 1% level

* Significance at 5% level

Table 41: Heterosis^w (per cent) for disease incidence at 75 DAT in the season I

Cross	TLCV			Tv			Total		
	di	dii	diii	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	0.79	18.46	4436.21**	26.74	26.74	26.74	17.87	29.74**	50.63**
LCR 9 x LCR 3	33.68	3424.13**	3424.13**	20.73	35.97*	35.97**	13.22	35.57**	35.57**
LCR 9 x CLN 2123A	-96.21**	0.00	0.00	56.20**	80.82**	80.82**	18.15	37.59**	44.41**
LCR 9 x H 24	5.76	2687.93**	2687.93**	-34.06**	-20.76	-20.76	-22.29*	-12.74	-2.26
LCR 9 x LE 415	53.37*	3943.10**	3943.10**	18.12	44.98**	44.98**	16.77	28.54**	49.23**
LCR 1 x LCR 9	-14.92	0.00	3729.31**	29.67*	29.67*	29.67*	6.17	16.87	35.68**
LCR 1 x LCR 3	193.37**	5663.79**	5663.79**	-11.21	0.00	0.00	49.65**	61.70**	61.70**
LCR 1 x CLN 2123A	-94.91**	0.00	0.00	-12.45	30.17*	30.17 *	-1.07	4.18	9.34
LCR 1 x H 24	-0.66	1851.72**	1851.72**	2.62	23.32	23.32	-1.51	2.79	19.33
LCR 1 x LE 415	17.51	2208.62**	2208.62**	-4.44	17.29	17.29	2.79	2.79	2.79
LCR 3 x LCR 9	116.81**	5615.52**	5615.52**	8.25	21.93	21.93	36.11**	62.99**	62.99**
LCR 3 x LCR 1	116.06**	4144.83**	4144.83**	-18.97	-8.73	-8.73	9.11	17.89	17.89
LCR 3 x CLN 2123A	2127.59**	2127.59**	2127.59**	-28.03*	31.24*	64.39**	38.36**	41.79**	41.79**
LCR 3 x H 24	4655.17**	4655.17**	4655.17**	-19.88	-14.05	6.39	28.00	35.68**	35.68**
LCR 3 x LE 415	3468.97**	3468.97**	3468.97**	32.61**	43.31**	79.51**	63.35**	76.50**	76.50**
CLN 2123A x LCR 9	-25.96	1851.72**	1851.72**	26.37*	46.29**	46.29**	2.51	19.38	25.30*

CLN 2123A x LCR 1	36.11	2574.13**	2574.13**	49.20**	79.47**	79.47**	42.98**	50.56**	58.03**
CLN 2123A x LCR 3	2834.48**	2834.48**	2834.48**	-24.18*	-22.29	-2.66	-1.58	0.86	0.86
CLN 2123A x H 24	2358.62**	2358.62**	2358.62**	75.12**	80.99**	138.00**	87.35**	93.63**	103.23**
CLN 2123A x LE 415	1302.00*	1301.72*	1301.72*	68.86**	77.82**	133.86**	72.72**	81.89**	90.20**
H 24 x LCR 9	-28.78	1777.58**	1777.58**	-5.77	13.24	13.24	-20.53*	-10.78	-0.07
H 24 x LCR 1	-94.91**	0.00	0.00	-14.16	3.15	3.15	-27.82*	-26.50*	-17.68
H 24 x LCR 3	3724.14**	3724.13**	3724.13**	-20.05	-15.24	6.17	13.08	19.87	19.87
H 24 x CLN 2123A	0.00	0.00	0.00	3.21	6.68	40.30**	3.21	6.68	11.96
H 24 x LE 415	515.52	1777.59**	1777.59**	-1.89	-0.03	40.30**	5.36	7.28	20.16
LE 415 x LCR 9	111.05**	5463.79**	5463.79**	-12.80	7.02	7.02	27.88**	40.76**	63.42**
LE 415 x LCR 1	17.51	2208.62**	2208.62**	-8.30	12.56	12.56	-10.83	-10.83	3.52
LE 415 x LCR 3	3551.72**	3551.72**	3551.72**	-54.90**	-51.26**	-38.95*	-15.20	-8.37	-8.37
LE 415 x CLN 2123A	3193.10**	3193.10**	3193.10**	-2.47	2.70	35.07*	19.12	25.44*	31.66**
LE 415 x H 24	4208.62**	4208.62**	4208.62**	1.80	3.66	45.48**	36.23**	38.72**	55.37**

** Significance at 1% level

* Significance at 5% level

^w Worked out for arc sine transformed values

Table 42: Heterosis^w (per cent) for disease incidence at 75 DAT in the season II

Cross	TLCV			Tv			Total		
	di	dii	diii	di	dii	diii	di	dii	diii
LCR 9 x LCR 1	54.39*	62.25*	123.53**	13.36	22.49	85.72**	31.73**	41.64**	42.36**
LCR 9 x LCR 3	66.46**	75.11**	141.25**	4.36	11.82	69.53**	31.47**	40.57**	41.28**
LCR 9 x CLN 2123A	2.92	19.97	19.97	58.35**	80.54**	173.72**	48.39**	57.16**	57.96**
LCR 9 x H 24	-96.90**	-96.74**	-95.36**	12.65	13.83	72.58**	-11.34**	-9.07*	-8.60*
LCR 9 x LE 415	-97.35**	-96.74**	-95.36**	79.30**	125.57**	125.57**	19.15**	19.46**	19.46**
LCR 1 x LCR 9	60.38**	68.54**	132.20**	9.39	18.20	79.21**	31.73**	41.64**	42.36**
LCR 1 x LCR 3	52.83*	52.98*	132.20**	-13.99	-13.31	50.24**	12.79**	13.38**	29.74**
LCR 1 x CLN 2123A	-33.05	-15.71	-15.71	-17.59	-13.37	52.51**	-22.10**	-20.97**	-11.20**
LCR 1 x H 24	-30.35	-30.27	5.57	8.34	15.79	79.21**	-4.05	0.48	6.17
LCR 1 x LE 415	-7.72	-7.24	62.77	5.95	46.24*	46.24*	-2.46	5.16	5.16
LCR 3 x LCR 9	18.56	24.72	71.83*	26.82**	35.89**	106.03**	26.32**	35.06**	35.75**
LCR 3 x LCR 1	52.83*	52.98**	132.00	29.14**	30.16**	125.57**	51.93**	52.72**	74.75**
LCR 3 x CLN 2123A	-20.69	0.00	0.00	1.11	7.17	85.72**	-4.45	-3.58	8.35*
LCR 3 x H 24	14.51	14.78	73.76*	9.25	15.79	79.21**	-0.57	16.74**	23.35**
LCR 3 x LE 415	-2.63	13.03	71.90*	105.90**	181.48**	181.48**	66.34**	75.70	75.70**
CLN 2123A x LCR 9	-96.22	-95.51**	-95.51**	-0.09	13.91	72.70**	-14.07**	-9.00*	-8.54*

CLN 2123A x LCR 1	-16.14	5.57	5.57	-20.40*	-16.32	47.31**	-20.55**	-19.40**	-9.43*
CLN 2123A x LCR 3	63.14*	105.65**	105.65**	8.00	14.48	100.0**	25.54**	26.69**	42.36**
CLN 2123A x H 24	34.36	68.89*	68.89*	16.78	31.61**	103.70**	22.63**	26.53**	33.69**
CLN 2123A x LE 415	-33.56	0.00	0.00	51.40**	122.64**	122.64**	19.77**	27.17**	27.17**
H 24 x LCR 9	21.20	27.19	75.23*	-20.69	-19.86	21.51	-4.07	-1.61	-1.11
H 24 x LCR 1	-30.35	-30.27	5.57	0.58	7.49	66.37**	-9.79**	-5.54	-0.19
H 24 x LCR 3	27.70	27.97	93.73**	-22.87*	-18.22	26.58	-2.80	1.23	6.96
H 24 x CLN 2123A	-24.14	-4.64	-4.64	-22.94*	-13.16	34.41	-24.61**	-22.22**	-17.81**
H 24 x LE 415	-22.91	-10.28	17.63	31.63*	67.68**	67.68**	4.60	7.56	7.56
LE 415 x LCR 9	12.36	38.15	90.33	16.76	46.89**	46.89**	13.73	14.01**	14.01**
LE 415 x LCR 1	-31.33	-20.35	21.13	19.54	64.99**	64.99**	-4.81	2.63	2.63
LE 415 x LCR 3	19.55	38.78	111.07**	29.18*	76.52**	76.52**	24.87**	33.88**	33.88**
LE 415 x CLN 2123A	-16.12	26.24	26.24	27.69*	87.75**	87.75**	8.21*	14.90**	14.90**
LE 415 x H 24	16.93	36.09	106.04**	73.27**	120.73**	120.73**	49.41**	53.62**	53.62**

** Significance at 1% level

* Significance at 5% level

^w Worked out for arc sine transformed values

Table 33: Performance of field –grown best performing hybrids along with parents and check during December 2002 to March 2003

Cross/parents	15 DAT (%)			30 DAT (%)			45 DAT (%)			60 DAT (%)			75 DAT (%)		
	TLCV	Tv	Total	TLCV	Tv	Total	TLCV	Tv	Total	TLCV	Tv	Total	TLCV	Tv	Total
H 24 x LCR 1	0.00	4.29	4.29	1.41	9.86	11.20	1.41	15.71	17.10	7.00	18.57	25.57	25.71	20.00	45.71
H 24 x CLN 2123A	0.00	5.00	5.00	0.00	11.25	11.25	0.00	27.5	27.50	1.25	31.25	32.50	3.75	33.75	37.5
LCR 1	0.00	14.81	14.81	0.00	27.78	27.78	16.67	37.03	53.70	19.08	48.15	67.23	38.88	50.00	88.88
H 24	0.00	16.28	16.28	2.24	31.71	33.95	21.21	33.87	55.08	21.21	38.66	59.87	29.14	34.21	63.35
CLN 2123A	0.00	5.21	5.21	0.00	15.24	15.24	0.00	32.21	32.21	0.00	42.37	42.37	0.00	60.23	60.23
CO 3	0.00	33.33	33.33	0.00	50.00	50.00	20.37	50.00	70.37	33.33	57.41	90.74	40.71	57.41	98.15

Cross /parent	CI for TLCV at 75 DAT	Reaction for TLCV	Number of fruits per plant	Average fruit weight (g)	Yield per plant (kg)	Estimated yield (t) per ha.
H 24 x LCR 1	8.89	Resistant	41.2	63.00	2.60	96.30
H 24 x CLN 2123A	0.47	Highly Resistant	70.70	43.40	3.07	113.70
LCR 1	14.48	Moderately Resistant	13.20	101.27	1.34	49.63
H 24	8.72	Resistant	33.20	38.11	1.27	47.04
CLN 2123A	0.00	Highly Resistant	27.20	45.71	1.24	45.93

Table 80: Peroxidase activity in best –performed hybrids, their parents and check CO 3 (Changes in OD/min/g fresh weight tissue)

SI. No.	Cross/parent	Inoculated							Control							Total mean
		0 hr	24 hr	48 hr	72 hr	96 hr	120 hr	Mean	0 hr	24 hr	48 hr	72 hr	96 hr	120 hr	Mean	
1.	H 24 x CLN 2123A	5.28	5.85	6.67	7.88	8.72	8.19	7.10	4.60	4.77	4.93	5.08	5.07	5.30	4.96	6.03
2.	H 24 x CLN 2123A	6.09	5.69	7.13	7.95	8.84	9.22	7.49	4.54	4.53	4.64	4.89	5.01	5.03	4.77	6.13
3.	H 24	4.92	5.59	6.27	6.81	7.42	7.22	6.37	4.74	4.82	4.90	4.96	5.25	5.09	4.96	5.67
4.	CLN 2123A	4.12	5.77	6.89	7.85	8.69	8.21	6.92	4.26	4.39	4.54	4.66	4.83	4.78	4.58	5.75
5.	LCR 1	4.88	5.28	6.03	6.75	6.90	6.70	6.09	4.52	4.57	4.64	4.74	4.76	4.66	4.65	5.37
6.	CO 3	3.38	3.82	3.98	4.34	4.98	5.02	4.25	3.36	3.46	3.74	3.75	3.77	3.40	3.64	3.95
	Mean	4.78	5.33	6.16	6.93	7.59	7.43	6.37	4.34	4.43	4.57	4.68	4.78	4.77	4.59	5.48

Treatment	SEd	CD (0.05)	CD (0.01)
Genotype	0.051	0.102	0.134
Hour	0.051	0.102	0.134
Virus	0.030	0.059	0.077
Genotype at hour	0.125	0.249	0.327
Hour at virus	0.072	0.144	0.189
Genotype at virus	0.072	0.144	0.189
Genotype at hour at virus	0.177	0.352	0.463

Table 81: Polyphenol oxidase activity in the best-performed hybrids, their parents and check CO 3 (changes in OD/min/g fresh weight tissue)

SI. No.	Cross/parent	Inoculated							Control							Total mean
		0 hr	24 hr	48 hr	72 hr	96 hr	120 hr	Mean	0 hr	24 hr	48 hr	72 hr	96 hr	120 hr	Mean	
1.	H 24 x CLN 2123A	0.610	0.771	0.833	0.880	0.969	0.960	0.837	0.588	0.595	0.596	0.605	0.615	0.619	0.603	0.720
2.	H 24 x CLN 2123A	0.711	0.749	0.798	0.829	0.866	0.885	0.806	0.661	0.674	0.673	0.676	0.684	0.685	0.676	0.741
3.	H 24	0.564	0.611	0.642	0.694	0.735	0.720	0.661	0.546	0.563	0.576	0.586	0.593	0.594	0.576	0.619
4.	CLN 2123A	0.666	0.682	0.740	0.799	0.879	0.872	0.773	0.661	0.672	0.675	0.682	0.689	0.695	0.679	0.726
5.	LCR 1	0.598	0.652	0.683	0.749	0.781	0.781	0.707	0.555	0.563	0.569	0.578	0.587	0.592	0.574	0.641
6.	CO 3	0.488	0.579	0.602	0.638	0.680	0.658	0.608	0.456	0.464	0.478	0.486	0.488	0.499	0.478	0.543
	Mean	0.606	0.674	0.716	0.765	0.818	0.813	0.732	0.578	0.588	0.595	0.602	0.609	0.614	0.598	0.665

Treatment	SEd	CD (0.05)	CD (0.01)
Genotype	0.0024	0.0048	0.0063
Hour	0.0024	0.0048	0.0063
Virus	0.0014	0.0028	0.0036
Genotype at hour	0.0059	0.0117	0.0154
Hour at virus	0.0034	0.0068	0.0089
Genotype at virus	0.0034	0.0068	0.0089
Genotype at hour at virus	0.0084	0.0017	0.0218

Table 82: Phenylalanine ammonia-lyase (PAL) activity in the best-performed hybrids, their parents and check CO 3 (μ mol cinnamic acid formed /min/g fresh weight tissue)

S.No.	Cross /parent	Inoculated	Control	Total mean
1.	H 24 x LCR 1	0.937	0.837	0.887
2.	H 24 x CLN 2123A	0.850	0.837	0.843
3.	H 24	0.740	0.757	0.748
4.	CLN 2123A	0.787	0.637	0.712
5.	LCR 1	0.720	0.543	0.632
6.	CO 3	0.510	0.413	0.462
	Mean	0.757	0.671	0.714

Treatment	SEd	CD (0.05)	CD (0.01)
Genotype	0.0273	0.0564	0.0768
Virus	0.0158	0.0326	0.0444
Genotype at virus	0.0386	0.0797	0.1087

Table 2: Analysis of variance for different characters in the season III

	Characters	Source	df	SS	MSS	F
1.	Plant height	Genotype	14	2425.06	173.22	50.28**
		Error	28	96.46	3.44	
2.	No. of branches per plant	Genotype	14	22.47	1.60	61.49**
		Error	28	0.73	0.03	
3.	Days to 50 per cent flowering	Genotype	14	368.58	26.33	11.53**
		Error	28	63.96	2.28	
4.	No. of fruits per plant	Genotype	14	5769.16	412.08	208.14**
		Error	28	55.43	1.98	
5.	Fruit weight	Genotype	14	16738.75	1195.62	275.01**
		Error	28	121.73	4.35	
6.	Yield per plant	Genotype	14	15.60	1.11	103.30**
		Error	28	0.30	0.01	
7.	pH	Genotype	14	1.00	0.07	3.70**
		Error	28	0.54	0.02	
8.	Acidity	Genotype	14	0.087	0.006	11.21**
		Error	28	0.016	0.001	
9.	TSS	Genotype	14	11.90	0.850	273.78**
		Error	28	0.09	0.003	
10.	Ascorbic acid	Genotype	14	402.19	28.73	2.31*
		Error	28	347.81	12.42	
11.	Lycopene	Genotype	14	13.89	0.99	14.62**
		Error	28	1.90	0.07	
12.	Total phenol	Genotype	14	9.07	0.65	29.87**
		Error	28	0.61	0.02	
13.	Reducing sugar	Genotype	14	11.03	0.79	29.27**
		Error	28	0.75	0.03	
14.	Sugar to acid ratio	Genotype	14	54.56	3.89	20.86**
		Error	28	5.23	0.19	
15.	Pectin	Genotype	14	0.41	0.03	55.46**
		Error	28	0.01	0.005	

16.	Total phenol	Genotype	14	47347.65	3381.97	36.40**
		Error	28	2601.74	92.92	
17.	OD phenol	Genotype	14	107578.18	7684.16	166.70**
		Error	28	1290.64	46.09	
18.	TLCV PDI at 15 DAT	Genotype	14	1109.35	79.24	4.66**
		Error	28	476.06	17.00	
19.	Tv PDI at 15 DAT	Genotype	14	1053.37	75.24	2.53**
		Error	28	831.69	29.70	
20.	Total PDI at 15 DAT	Genotype	14	953.56	68.33	2.38**
		Error	28	804.01	28.71	
21.	TLCV PDI at 30 DAT	Genotype	14	1633.30	116.66	7.05**
		Error	28	463.41	16.55	
22.	Tv PDI at 30 DAT	Genotype	14	322.91	23.07	3.19**
		Error	28	202.34	7.23	
23.	Total PDI at 30 DAT	Genotype	14	812.27	58.02	7.31**
		Error	28	222.27	7.94	
24.	TLCV PDI at 45 DAT	Genotype	14	1217.19	86.94	4.03**
		Error	28	603.39	21.55	
25.	Tv PDI at 45 DAT	Genotype	14	1173.15	83.80	12.14**
		Error	28	193.25	6.90	
26.	Total PDI at 45 DAT	Genotype	14	1427.96	101.99	10.16**
		Error	28	281.18	10.04	
27.	TLCV PDI at 60 DAT	Genotype	14	1209.30	86.38	3.89**
		Error	28	620.32	22.15	
28.	Tv PDI at 60 DAT	Genotype	14	1145.79	81.84	6.80**
		Error	28	336.88	12.03	
29.	Total PDI at 60 DAT	Genotype	14	1355.44	96.82	9.39**
		Error	28	288.74	10.31	
30.	TLCV PDI at 75 DAT	Genotype	14	377.02	26.93	4.29**
		Error	28	175.84	6.28	
31.	Tv PDI at 75 DAT	Genotype	14	1080.14	77.15	10.82**
		Error	28	199.63	7.13	
32.	Total PDI at 75 DAT	Genotype	14	1611.63	115.17	14.89**

		Error	28	216.53	7.73	
33.	TLCV PDI at 90 DAT	Genotype	14	426.54	30.47	4.82**
		Error	28	176.86	6.31	
34.	Tv PDI at 90 DAT	Genotype	14	1018.13	72.72	10.54**
		Error	28	193.10	6.90	
35	Total PDI at 90 DAT	Genotype	14	1373.20	98.09	13.98***
		Error	28	196.51	7.02	
36	CI for TLCV at 75 DAT	Genotype	14	95.11	6.79	35.68**
		Error	28	5.33	0.19	

* Significance at 5 per cent level

** Significance at 1 per cent level

Table 34: Effect of different physical manipulations on various characters of the hybrid H 24 x CLN 2123A

Treat ment	Parti culars	Plant height (cm)	Branches/plant	Fruit No.	Av. fr. wt. (g)	Yield/ plant (kg)	Polar diameter (cm)	Equatorial diameter (cm)	Pericarp thickness		TSS (°Brix)	Total acidity (%)
									Outer wall thickness (cm)	Inner wall (Septa) thickness (cm)		
T ₁	Nipping	65.47	7.67	51.67	44.00	2.27	4.34	5.01	0.48	0.50	5.38	0.69
T ₂	Flower Thinning	93.73	8.92	47.62	53.55	2.55	4.03	4.61	0.46	0.45	5.48	0.50
T ₃	Truss Thinning	86.13	9.18	33.78	57.23	1.93	4.57	4.92	0.44	0.51	5.32	0.67
T ₄	Alternate Truss Thinning	90.04	9.00	35.08	54.62	1.92	4.38	4.90	0.58	0.62	5.25	0.65
T ₅	Control	90.67	8.89	76.58	40.67	3.11	3.85	4.19	0.42	0.41	5.97	0.69

Table 76: Genotypic correlation between yield and yield components in the season I

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	1.000	-0.372*	0.407*	- 0.433**	-0.315	0.042	-0.285	0.051	-0.025	-0.267	-0.009	-0.294	-0.374*	-0.348*	-0.316
2		1.000	0.704**	0.045	0.001	0.324	0.000	-0.127	-0.090	0.157	-0.034	-0.048	-0.328*	-0.231	-0.032
3			1.000	-0.306	-0.245	0.362*	-0.248	-0.109	-0.136	-0.062	-0.071	-0.316	- 0.666**	- 0.537**	-0.317
4				1.000	0.618	0.149	0.191	0.258	0.087	-0.113	0.015	0.249	0.180	0.029	0.561**
5					1.000	0.303	0.135	0.035	0.048	-0.253	-0.151	0.150	0.029	0.048	0.281
6						1.000	- 0.447**	0.388*	-0.201	-0.360*	-0.059	-0.146	-0.187	-0.081	-0.045
7							1.000	- 0.553**	0.010	0.277	-0.036	0.181	0.187	0.062	0.548**
8								1.000	-0.110	- 0.479**	0.143	-0.164	0.111	-0.043	0.326*
9									1.000	0.011	-0.118	0.287	0.069	0.249	-0.048
10										1.000	0.246	0.082	-0.083	-0.042	-0.155
11											1.000	-0.085	-0.041	0.071	0.082
12												1.000	0.227	0.288	0.084
13													1.000	0.793**	0.345
14														1.000	0.038
15															1.000

* Significance at 5 per cent level

** Significance at 1 per cent level

1. TLCV at 75 DAT

2. Tv at 75 DAT

3. Total disease at 75 DAT

4. Plant height

5. Number of branches per plant

6. Days to 50 per cent flowering

7. Number of fruits per plant

8. Fruit weight

9. TSS

10. Acidity

11. Ascorbic acid

12. Lycopene

13. Total phenol

14. OD phenol

15. Yield per plant

Table 78: Path coefficients of component traits on yield in the season I

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Correlation with yield
1	0.477	-0.252	-0.244	-0.050	-0.015	0.002	-0.260	0.037	-0.001	0.008	0.000	0.005	-0.083	0.061	-0.316
2	-0.177	0.679	-0.422	0.005	0.000	0.017	0.000	-0.092	-0.005	-0.005	-0.001	0.001	-0.073	0.040	-0.032
3	0.194	0.478	-0.599	-0.035	-0.012	0.019	-0.227	-0.079	-0.007	0.002	-0.003	0.005	-0.148	0.094	-0.317
4	-0.207	0.031	0.183	0.114	0.030	0.008	0.175	0.187	0.004	0.003	0.001	-0.004	0.040	-0.005	0.561**
5	-0.150	0.000	0.147	0.071	0.048	0.016	0.123	0.026	0.002	0.008	-0.006	-0.003	0.007	-0.008	0.281
6	0.020	0.220	-0.217	0.017	0.015	0.053	-0.409	0.282	-0.010	0.011	-0.002	0.002	-0.042	0.014	-0.045
7	-0.136	0.000	0.148	0.022	0.007	-0.024	0.915	-0.402	0.001	-0.008	-0.001	-0.003	0.042	-0.011	0.548**
8	0.024	-0.086	0.065	0.029	0.002	0.021	-0.506	0.728	-0.006	0.014	0.005	0.003	0.025	0.007	0.326*
9	-0.012	-0.061	0.082	0.010	0.002	-0.011	0.009	-0.080	0.051	0.000	-0.005	-0.005	0.015	-0.044	-0.048
10	-0.127	0.107	0.037	-0.013	-0.012	-0.019	0.254	-0.349	0.001	-0.030	0.009	-0.001	-0.019	0.007	-0.155
11	-0.004	-0.023	0.042	0.002	-0.007	-0.003	-0.033	0.104	-0.006	-0.007	0.038	0.001	-0.009	-0.012	0.082
12	-0.140	-0.033	0.190	0.028	0.007	-0.008	0.166	-0.119	0.015	-0.002	-0.003	-0.017	0.051	-0.051	0.084
13	-0.179	-0.223	0.399	0.021	0.001	-0.010	0.170	0.081	0.004	0.003	-0.002	-0.004	0.223	-0.139	0.345
14	-0.166	-0.157	0.321	0.003	0.002	-0.004	0.057	-0.031	0.013	0.001	0.003	-0.005	0.176	-0.176	0.038

Residual effect = 0.132

* Significance at 5 per cent level

1. TLCV at 75 DAT
2. Tv at 75 DAT
3. Total disease at 75 DAT
4. Plant height
5. Number of branches per plant

** Significance at 1 per cent level

6. Days to 50 per cent flowering
7. Number of fruits per plant
8. Fruit weight
9. TSS
10. Acidity
11. Acidity
12. Ascorbic acid
13. Lycopene
14. Total phenol

Table 77: Genotypic correlation between yield and yield components in the season III

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1	1.000	0.342	0.484**	- 0.373*	-0.248	0.561**	- 0.559**	-0.080	0.061	-0.295	0.247	0.322	-0.050	-0.267	-0.310	-0.074	-0.012	-0.347	-0.22
2		1.000	0.940**	-0.277	-0.233	-0.349	0.011	-0.337	-0.123	0.265	0.083	-0.276	0.217	0.494**	0.621**	0.253	0.293	- 0.408*	-0.16
3			1.000	-0.247	-0.225	-0.149	-0.066	-0.357	-0.143	0.222	0.060	0.079	0.077	0.439*	0.506**	0.240	0.193	-0.274	-0.06
4				1.000	0.589**	0.119	0.367*	0.121 0.636**	-	0.078	0.209	0.092	-0.461*	0.219	0.104	0.129	0.207	-0.095	-0.17
5					1.000	-0.082	0.426*	-0.260	-0.356	0.193 0.297	-	0.208	-0.144	-0.084	-0.069	-0.181	-0.253	0.213	0.17
6						1.000	- 0.604**	0.441* 0.675**	-0.223	- 0.085	-	0.409* 0.468**	-0.314	-0.337	-	0.064	-0.067	-0.021	-0.19
7							1.000 0.437*	- 0.437*	-0.071	0.379*	0.056	0.071	0.093	0.363*	0.471**	0.115	-0.357	0.191	0.19
8								1.000	0.173	- 0.485**	- 0.124	-0.046	-0.273	0.006	-0.246	0.246	0.066	-0.275	- 0.515
9									1.000	- 0.518**	- 0.073	-0.224	0.243	-0.258	-0.202	0.068	-0.404*	-0.243	-0.373
10										1.000	0.110	0.225	-0.119	-0.078	-0.022	- 0.579**	0.158	0.232	0.556

11											1.000	-0.253	-0.080	-0.030	-0.026	-0.067	0.565**	-0.109	0.000
12												1.000	-	-0.371*	-0.402*	-0.409*	-	0.412*	0.442
													0.766**				0.493**		
13													1.000	0.059	0.216	0.102	-0.073	0.044	-0.05
14														1.000	0.921**	0.858**	0.150	-0.216	-0.27
15															1.000	0.758**	0.099	-0.248	-0.21
16																1.000	0.035	-0.266	-
																		0.491	
17																	1.000	-0.069	-0.00
18																		1.000	0.901
19																			1.00
20																			

* Significance at 5 per cent level

** Significance at 1 per cent level

1. TLCV at 75 DAT

2. Tv at 75 DAT

3. Total disease at 75 DAT

4. Plant height

5. Number of branches per plant

6. Days to 50 per cent flowering

7. Number of fruits per plant

8. Fruit weight

9. pH

10. Acidity

11. TSS

12. Ascorbic acid

13. Lycopene

14. Total sugar

15. Reducing sugar

16. Sugar to acid ratio

17. Pectin

18. Total phenol

19. OD phenol

20. Yield per plant

Table 79: Path coefficients of component traits on yield in the season III

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	Cor with yield
1	-0.363	0.010	0.143	-	0.082	0.009	-	-	0.007	-	-	-	0.000	0.142	0.030	-	0.002	0.094	-	-0.741
				0.175			0.446	0.058		0.010	0.052	0.052				0.028			0.073	
2	-0.124	0.030	0.277	-	0.077	-	0.009	-	-	0.009	-	0.044	0.002	-	-	0.097	-	0.111	-	-0.274
				0.107		0.006		0.245	0.013		0.018			0.262	0.060		0.041		0.053	
3	-0.175	0.028	0.294	-	0.074	-	-	-	-	0.007	-	-	0.001	-	-	0.092	-	0.074	-	-0.406*
				0.116		0.002	0.053	0.260	0.016		0.013	0.013		0.233	0.049		0.027		0.021	
4	0.135	-	-	0.470	0.194	0.002	0.293	0.088	-	0.003	-	-	-	-	-	0.050	-	0.026	-	0.451*
		0.007	0.073						0.069		0.045	0.015	0.004	0.116	0.010		0.029		0.054	
5	0.090	-	-	0.277	-	-	0.340	-	-	0.006	0.063	-	-	0.045	0.007	-	0.035	-	0.057	0.127
		0.007	0.066		0.329	0.001		0.189	0.039			0.033	0.012			0.069		0.058		
6	-0.203	-	-	0.056	0.027	0.016	-	0.321	-	-	0.018	-	-	0.179	0.045	0.025	0.009	0.006	-	-0.215
		0.010	0.044				0.482		0.024	0.023		0.066	0.003						0.062	
7	0.203	0.000	-	0.173	-	-	0.798	-	-	0.013	-	-	0.001	-	-	0.044	0.050	-	0.061	0.532**
			0.019		0.140	0.010		0.319	0.008		0.012	0.011		0.193	0.045			0.052		
8	0.029	-	-	0.057	0.085	0.007	-	0.728	0.019	-	0.026	0.007	-	-	0.024	0.	-	0.075	-	0.492**
		0.010	0.105				0.349			0.016			0.002	0.003		0.094	0.009		0.165	
9	-0.022	-	-	-	0.117	-	-	0.126	0.108	-	0.016	0.036	0.002	0.137	0.019	0.026	0.056	0.066	-	0.146
		0.004	0.042	0.299		0.004	0.057			0.017									0.119	

10	0.107	0.008	0.065	0.037	-	-	0.302	-	-	0.033	-	-	-	0.042	0.002	-	-	-	0.178	-0.077
					0.064	0.011		0.353	0.056		0.023	0.036	0.001			0.222	0.022	0.063		
11	-0.089	0.002	0.018	0.098	0.098	-	0.045	-	-	0.004	-	0.041	-	0.016	0.002	-	-	0.030	0.000	-0.153
						0.001		0.090	0.008		0.213		0.001			0.026	0.079			
12	-0.117	-	0.023	0.043	-	0.007	0.057	-	-	0.008	0.054	-	-	0.197	0.039	-	0.069	-	0.141	-0.049
		0.008			0.068			0.033	0.024		0.160	0.006				0.157		0.112		
13	0.018	0.007	0.023	-	0.047	-	0.074	-	0.026	-	0.017	0.123	0.008	-	-	0.039	0.010	-	-	-0.115
				0.217		0.005		0.199		0.004				0.031	0.021			0.012	0.019	
14	0.097	0.015	0.129	0.103	0.028	-	0.290	0.004	-	-	0.006	0.059	0.000	-	-	0.329	-	0.059	-	0.354
						0.005			0.028	0.003				0.531	0.089		0.021		0.089	
15	0.113	0.019	0.149	0.049	0.023	-	0.376	-	-	-	0.005	0.065	0.002	-	-	0.290	-	0.067	-	0.280
						0.007		0.179	0.022	0.001				0.489	0.096		0.014		0.070	
16	0.027	0.008	0.071	0.061	0.059	0.001	0.091	0.179	0.007	-	0.014	0.066	0.001	-	-	0.383	-	0.073	-	0.331
										0.019				0.456	0.073		0.005		0.157	
17	0.004	0.009	0.057	0.097	0.083	-	-	0.048	-	0.005	-	0.079	-	-	-	0.013	-	0.019	-	-0.265
						0.001	0.285		0.044		0.120		0.001	0.080	0.010		0.140		0.001	
18	0.126	-	-	-	-	0.000	0.153	-	-	0.008	0.023	-	0.000	0.115	0.024	-	0.010	-	0.288	-0.129
		0.012	0.081	0.045	0.070			0.200	0.026			0.066				0.102		0.272		
19	0.083	-	-	-	-	-	0.151	-	-	0.019	0.000	-	0.000	0.148	0.021	-	0.000	-	0.320	-0.344
		0.005	0.019	0.080	0.058	0.003		0.375	0.040			0.071				0.188		0.245		

* Significance at 5 per cent level

** Significance at 1 per cent level

Residual effect = -0.307

1. TLCV at 75 DAT
2. Tv at 75 DAT
3. Total disease at 75 DAT
4. Plant height
5. Number of branches per plant
6. Days to 50 per cent flowering
7. Number of fruits per plant
8. Fruit weight
9. pH
10. Acidity
11. TSS
12. Ascorbic acid
13. Lycopene
14. Total sugar
15. Reducing sugar
16. Sugar to acid ratio
17. Pectin
18. Total phenol
19. OD phenol

Table 83: Best parents with significantly high *per se* and *gca* effects for selected characters

Characters	<i>Per se</i>	<i>gca</i>	<i>Per se and gca</i>
Fruit weight	LCR 1, LCR 9	LCR 1, LCR 9	LCR 1, LCR 9
Yield	LCR 3, LCR 1	LCR 1, H 24	LCR 1
Total phenol	LE 415, H 24	H 24	H 24
OD phenol	LE 415	H 24, LE 415	LE 415
TLCV incidence ^b	CLN 2123A, LCR 3, H 24	H 24, CLN 2123A	H 24, CLN 2123A
Tv incidence ^b	LCR 1, LCR 9 LE 415, H 24	LCR 9, LCR 1, LCR 3, H 24	LCR 1, LCR 9, H 24
Total disease incidence ^{b,c}	LCR 3 LCR 9, LE 415	-	-

^b – low values were considered as best

^c – not *gca* since mostly arithmetic mean of disease incidence

Table 84: Best hybrids with significantly high *per se*, *sca* effects and heterosis over best parent for selected characters

Characters	<i>Per se</i> and heterosis	<i>Per se</i> , heterosis and <i>sca</i>
Fruit weight	CLN 2123A x LCR 9	CLN 2123A x LCR 9
Yield	H 24 x LE 415, H 24 x CLN 2123A, H 24 x LCR 9, LE 415 x LCR 1	H 24 x LE 415, H 24 x CLN 2123A, H 24 x LCR 9
Total phenol	H 24 x CLN 2123A	H 24 x CLN 2123A
OD phenol	H 24 x LCR 1, LCR 1 x LCR 9	H 24 x LCR 1, LCR 1 x LCR 9
TLCV incidence ^b	H 24 x LCR 1, LCR 9 x CLN 2123A, LCR 1 x CLN 2123A, H 24 x CLN 2123, CLN 2123A x LCR 9, LCR 9 x H 24, LCR 9 x LE 415, LCR 1 x CLN 2123A,	H 24 x LCR 1, LCR 9 x CLN 2123A, H 24 x CLN 2123, CLN 2123A x LCR 9, LCR 9 x LE 415
Tv incidence ^b	LE 415 x LCR 3, LCR 9 x H 24, LCR 3 x LCR 1, CLN 2123A x LCR 3, H 24 x CLN 2123A, H 24 x LCR 3, CLN 2123A x LCR 1	LE 415 x LCR 3, LCR 9 x H 24, CLN 2123A x LCR 3, H 24 x CLN 2123A
Total disease incidence ^{b,c}	H 24 x LCR 1, LE 415 x LCR 3, LCR 9 x H 24, H 24 x LCR 9, H 24 x CLN 2123A, LCR 1 x CLN 2123A, CLN 2123A x LCR 1, CLN 2123A x LCR 9	-

^a – if not over diii, then dii/di

^b – low values were considered as best

^c not *sca* since arithmetic mean of disease incidence

Table 85: Best hybrids with mean, heterosis over best parent and *gca* of their parents for selected characters

Characters	Hybrids	Mean	diii (%)	<i>gca</i> ^d of the parents
Fruit weight	CLN 2123A x LCR 9	73.00 kg	-34.36**	Low x High
Yield	H 24 x LE 415	3.25 kg	22.18**	High x Low
	H 24 x CLN 2123A	3.12 kg	17.11**	High x Medium (+)
	H 24 x LCR 9	2.85 kg	6.95**	High x Low
Total phenol	H 24 x CLN 2123A	307.60 mg/100g	0.56	High x Low
OD phenol	H 24 x LCR 1	247.35 mg/100g	-8.88**	High x Low
	LCR 1 x LCR 9	190.45 mg/100g	-29.84**	Low x Low
TLCV-incidence - season I ^b	H 24 x LCR 1	0.00 %	-	High x Medium (-)
	LCR 9 x CLN 2123A	0.00 %	-	Low x High
	H 24 x CLN 2123A	0.00 %	-	High x High
TLCV incidence - season II ^b	CLN 2123A x LCR 9	0.00 %	-	High x Medium (+)
	LCR 9 x LE 415	0.00 %	-	Medium (+) x Medium (-)
Tv incidence - season I ^b	LE 415 x LCR 3	5.50%	-38.95*	Medium (-) x High
	LCR 9 x H 24	9.14%	-20.76	High x Medium (-)
	CLN 2123A x LCR 3	13.57%	-2.66	Low x High
Tv incidence - season II ^b	H 24 x CLN 2123A	14.65%	34.41	High x Low
Total disease incidence - season I ^{b,c}	H 24 x LCR 1	15.15%	-17.68	-
	LE 415 x LCR 3	18.54%	-8.37	-
	LCR 9 x H 24	21.76%	-2.26	-

	H 24 x LCR 9	20.90%	-0.07	-
Total disease incidence - season II ^{b,c}	H 24 x CLN 2123A	19.19%	-17.81**	-
	LCR 1 x CLN 2123A	22.14%	-11.20**	-
	CLN 2123A x LCR 1	22.96%	-9.43*	-
	LCR 9 x H 24	23.34%	-8.60*	-
	CLN 2123A x LCR 9	23.37%	-8.54*	-
	H 24 x LCR 9	26.93%	-1.11	-
	H 24 x LCR 1	27.38%	-0.19	-

^b – Low values were considered as best ^c not *sca* since arithmetic mean of disease incidence

^d – High and low represents those having significant values with positive and negative signs respectively whereas medium represents non significant values

APPENDIX I

Screening of genotypes for selection of parents in the study

A. TLCV

Genotypes	Field condition			Controlled condition			Fruit weight (g)
	PDI ^a	CI	Reaction	PDI ^b	CI	Reaction	
LCR 2	30.00	9.98	MR	50.00	23.75	MS	52.50
LCR 4	35.00	11.25	MR	45.33	24.00	MS	58.00
LCR 9	20.00	6.25	R	38.88	9.72	MR	71.00
LCR 1	10.00	5.00	R	33.33	11.90	MR	105.50
LCR 3	15.00	4.68	R	20.00	6.00	R	39.00
CLN 2123A	0.00	0.00	HR	15.00	1.88	HR	52.50
H 24	10.00	5.00	R	25.00	10.00	MR	43.00

PDI – Percentage Disease Incidence; CI – Coefficient of Infection; MS–Moderately Susceptible; R– Resistant; MR–Moderately Resistant; HR – Highly Resistant

^a after 75 days of transplanting

^b 60 days after inoculation

B. Tv (Field condition)

Genotype	PDI at 75 DAT	Average fruit weight
LE 415	10.00	33.00
Stevens	60.00	74.00

APPENDIX III**Weather data during the cropping period (December 2000 to May 2003)**

Month & Date	Temperature °C		Relative humidity (hrs)		Rainfall (mm)	Sunshine (hrs. /day)	Evaporation (mm)
	Min.	Max.	7.22	14.22			
2000 December	18.0	31.0	88	46	22.6	3.8	7.9
2001							
January	19.7	30.3	87	44	-	4.3	6.2
February	20.2	33.5	87	37	-	5.5	8.2
March	22.1	35.3	80	35	-	6.8	8.8
April	23.6	34.7	87	42	96.0	5.6	7.0
May	23.5	35.0	82	44	6.5	6.8	8.6
June	22.1	31.2	78	52	52.6	6.9	4.9

July	22.7	31.2	80	54	19.9	6.8	5.6
August	22.1	31.4	85	53	22.8	6.0	5.7
September	22.3	32.7	89	50	96.6	5.1	6.8
October	22.1	31.0	90	61	286.7	4.3	5.3
November	21.6	29.5	92	63	136.6	3.8	6.4
December	18.7	28.2	90	55	16.1	3.6	6.2
2002							
January	19.2	30.3	88	49	-	3.9	7.5
February	19.8	31.6	86	40	-	4.9	7.1
March	21.3	35.0	84	32	69.2	7.2	9.4
April	24.1	36.3	81	36	3.0	6.8	9.3
May	24.0	35.1	82	46	29.1	6.1	7.2
June	23.7	32.9	78	49	6.8	7.0	6.4
July	22.6	32.8	84	47	6.5	6.2	6.7
August	22.7	31.1	81	54	59.8	5.9	5.1
September	21.9	33.2	87	43	19.9	6.8	8.5
October	22.6	30.9	90	61	229.9	3.8	4.7
November	21.2	29.7	92	57	99.7	3.2	5.6
December	17.3	30.3	87	37	1.7	4.1	7.1
2003							
January	18.5	30.8	85	38	-	4.6	8.3
February	21.7	33.0	85	37	25.0	5.0	8.7
March	21.8	34.4	87	35	119.1	5.4	9.2
April	23.6	35.5	86	42	63.7	5.4	7.9
May	24.4	35.5	86	43	19.5	5.9	7.5

Source: Department of Agricultural Meteorology, Tamil Nadu Agricultural University, Coimbatore – 641 003.

Appendix II

Brief characterization of parents under study

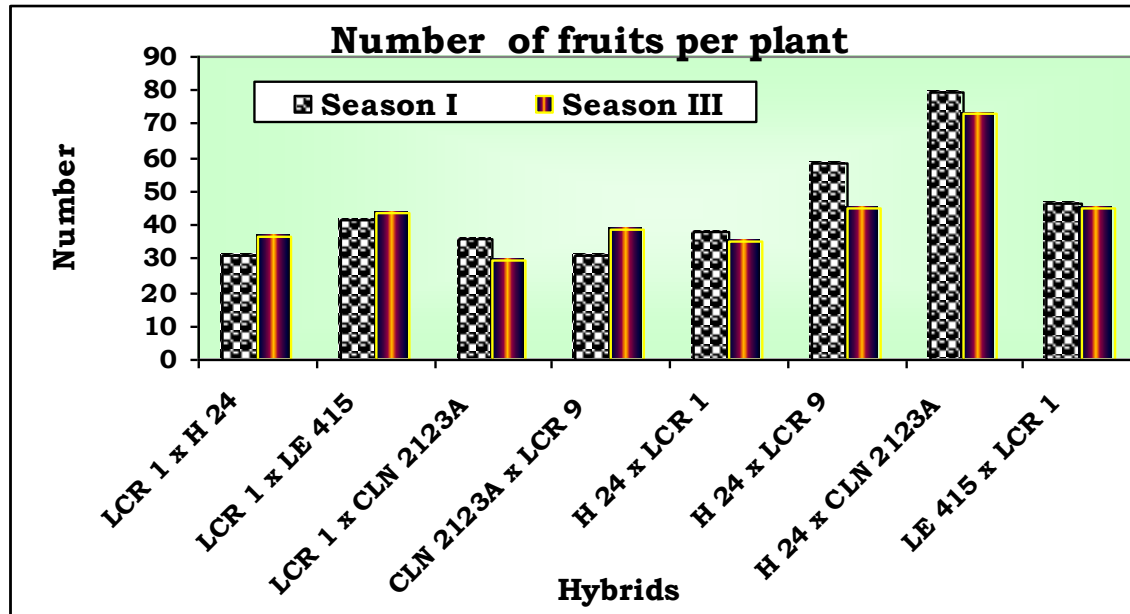
S.No.	Characters	LCR 9	LCR 1	LCR 3	H 24	CLN 2123A	LE 415
1.	Anthocyanin colouration of hypocotyl	Present	Present	Present	Present	Absent	Present
2.	Growth	Determinate-Intermediate	Determinate-Intermediate	Determinate-Compact	Determinate-Intermediate	Determinate-large	Determinate-Intermediate
3.	Number of leaves under first inflorescence ^a	Many	Medium	Medium	Medium	Many	Medium
4.	Internode length	Short	Short	Short	Medium	Medium	Medium
5.	Foliage cover ^b	Good	Good	Fair	Fair	Fair	Fair
6.	Fruit size ^c	Medium	Large	Small	Small	Small	Small
7.	Fruit shape	Slightly flattened	flattened	Pear shaped	Slightly flattened	High - Round	High - Round
8.	Exterior colour of immature fruit	Dark, green back present	Light, green back present	Light, green back present	Light, green back present	Light, green back present	Light, green back present
9.	Interior flesh colour	Tangerine and Red	Tangerine and Red	Red	Tangerine	Tangerine and Red	Tangerine and Red
10.	Transverse section of fruit	Irregular	Irregular	Round	Irregular	Round	Round
11.	Jointless pedicel	Absent	Absent	Absent	Absent	Present	Absent
12.	Ribbing at calyx end	Medium	Medium	Absent	Slight	Absent	Slight
13.	Pedicel area	Moderately depressed	Moderately depressed	Flat	Slightly depressed	Flat	Slightly depressed
14.	Shape of pistil scar	Irregular	Irregular	Dot	Stellate	Dot	Stellate
15.	Blossom end shape	Indented	Indented	Flat	Flat	Pointed	Flat

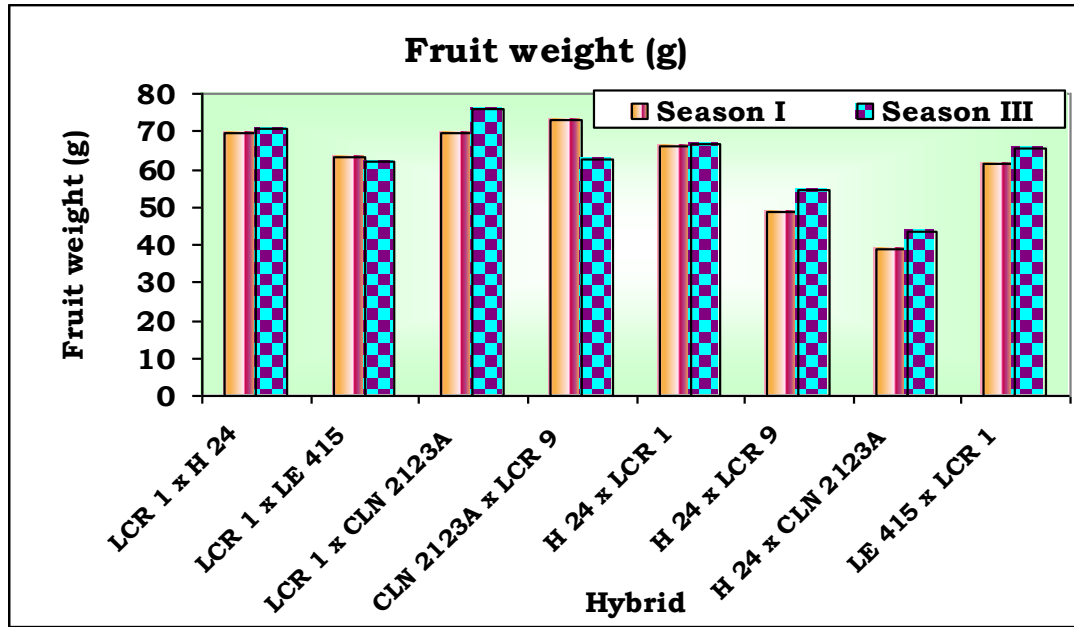
^a <b Few; 6-8 Medium; > 8 many

^b compared with CO 3;

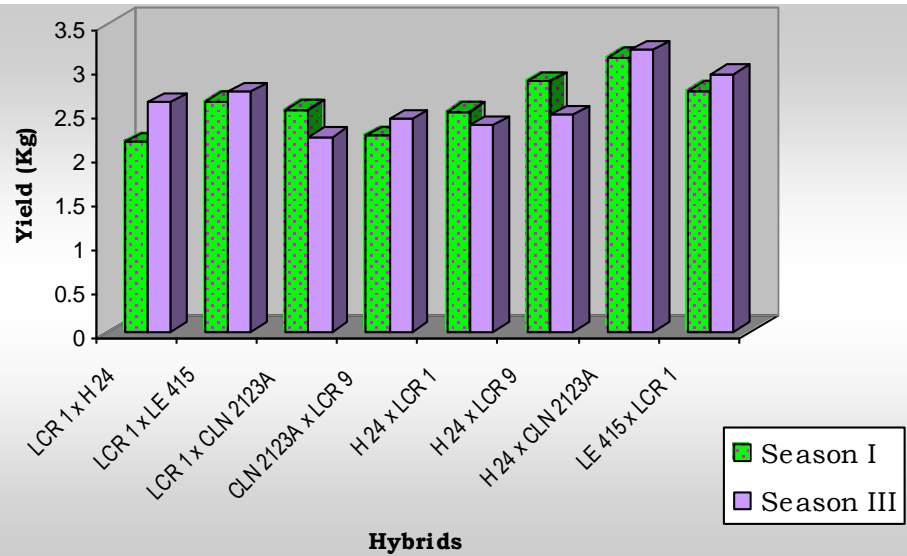
^c 3.0-4.0 cm - small; 4.0-5.0 cm - medium; 5.0-6.0 - large

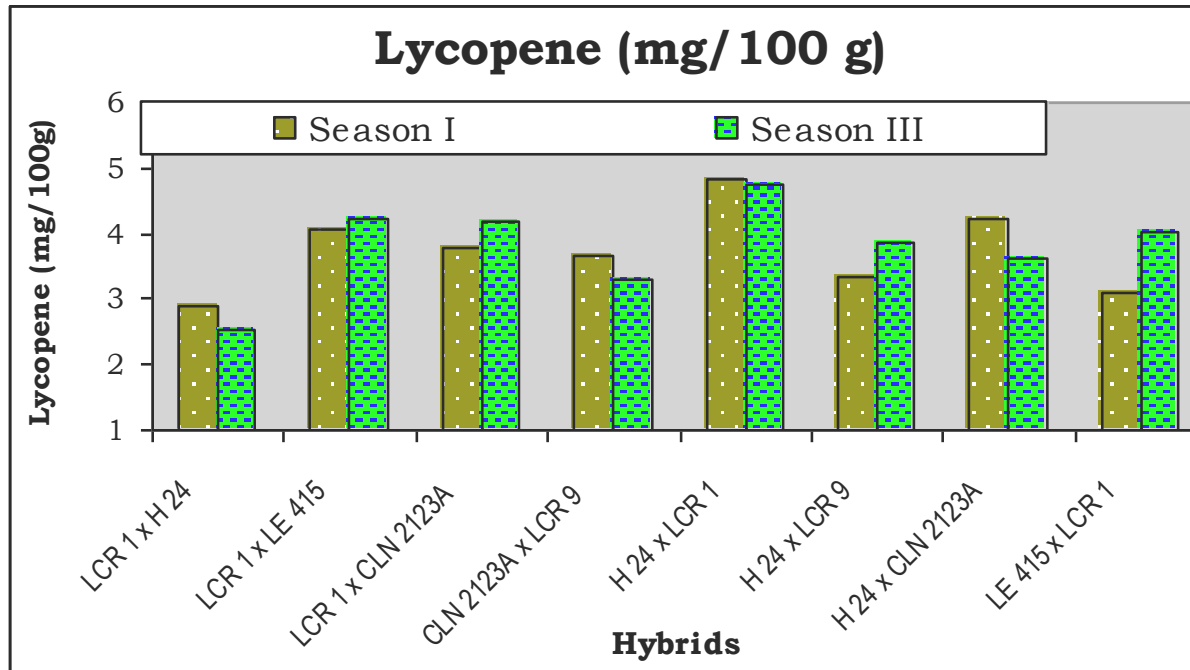
Fig . 21: Performance of selected F₁ hybrids for biometrical characters in different seasons

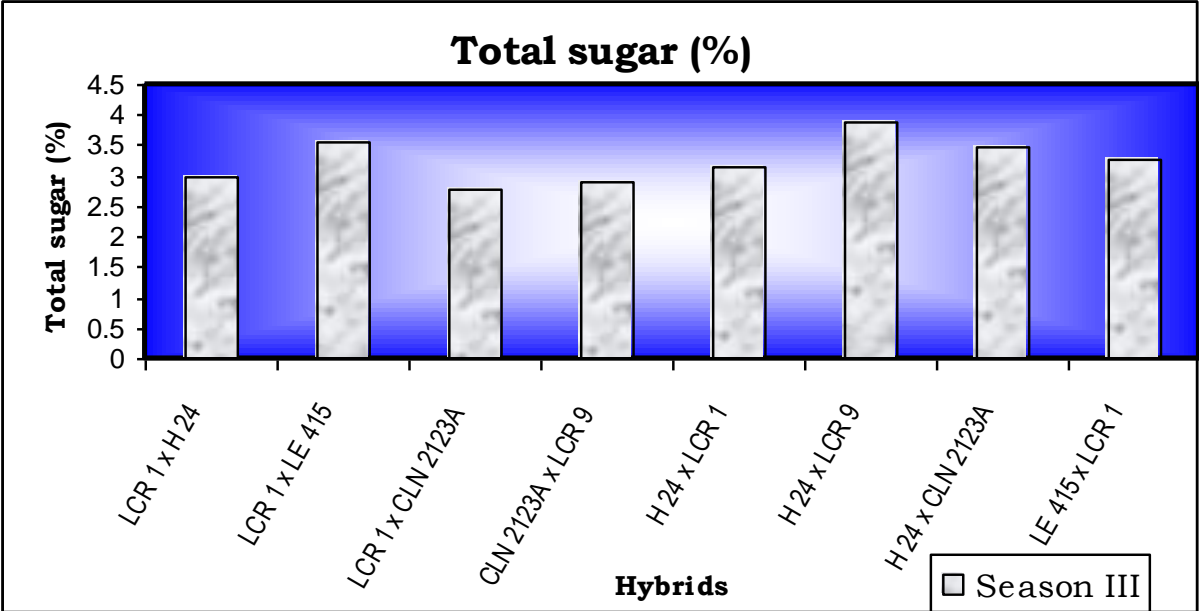


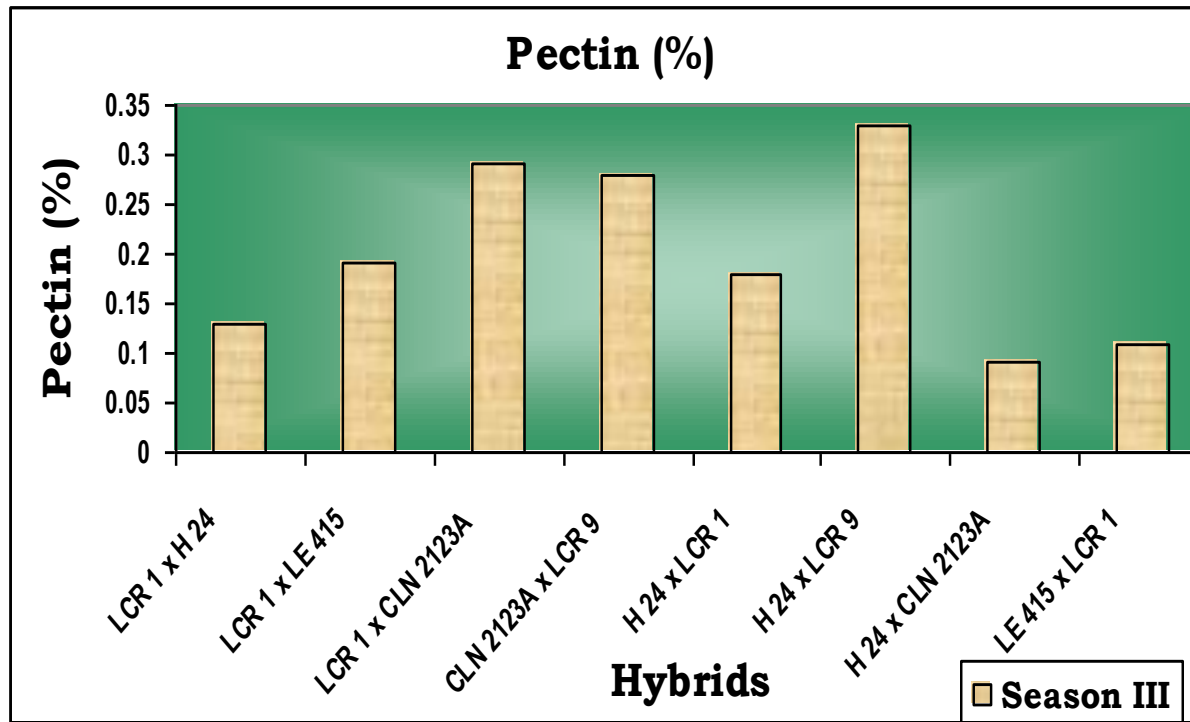


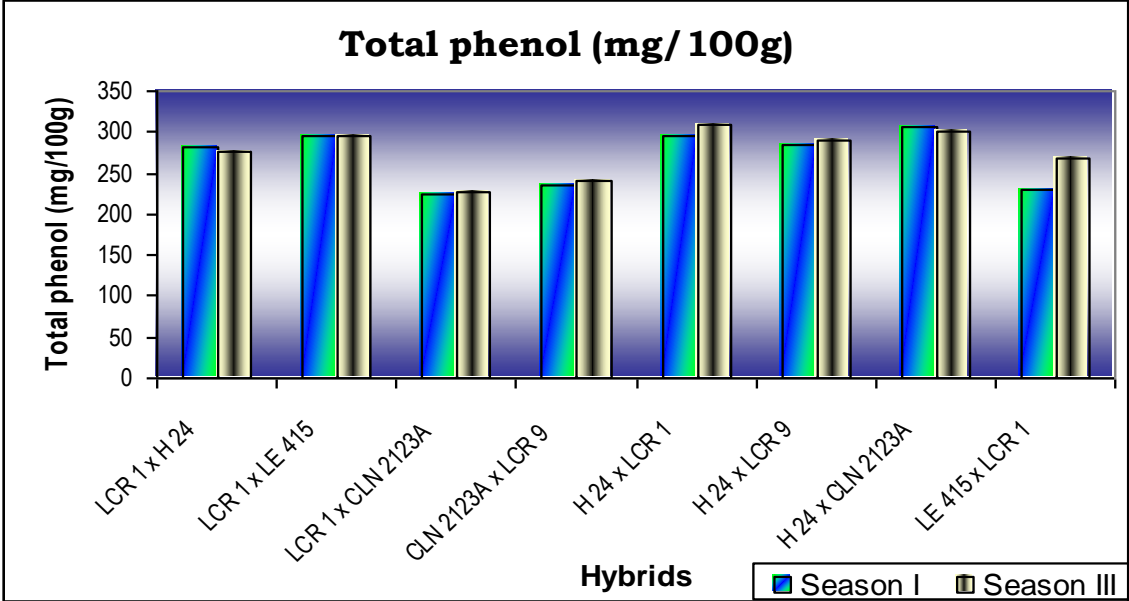
Yield per plant (Kg)











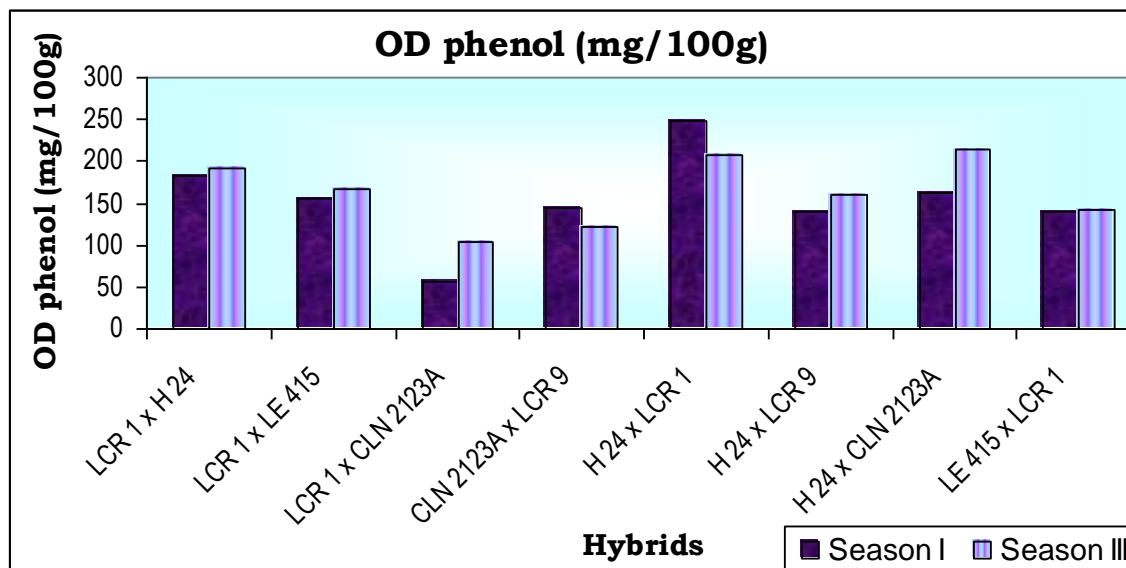
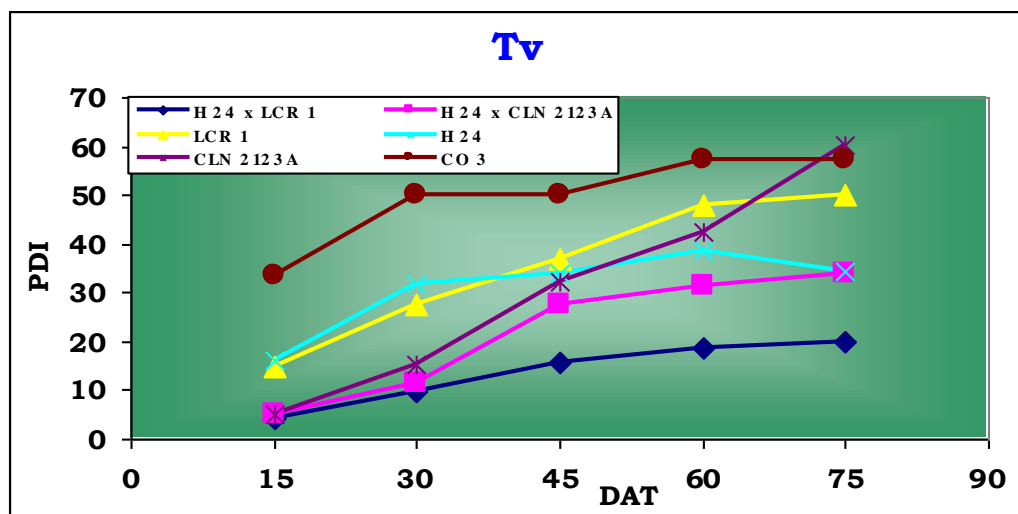
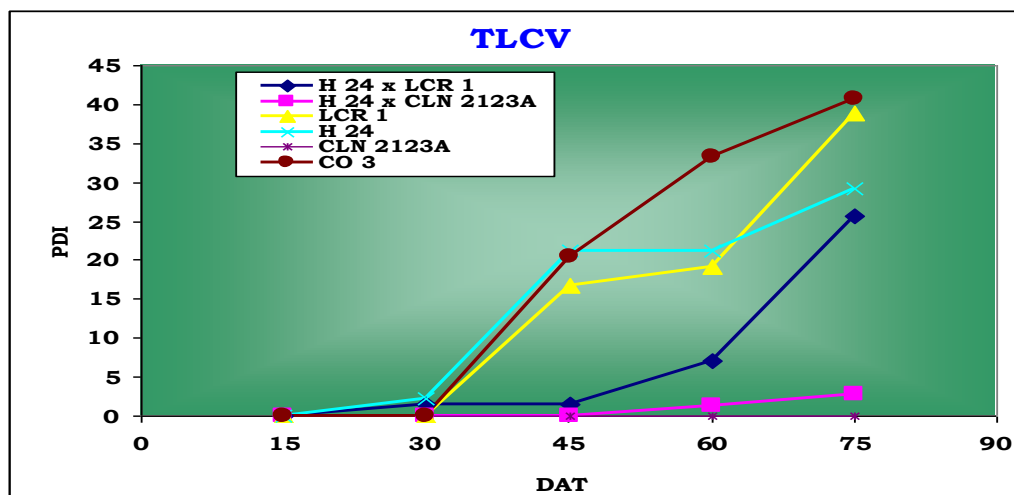


Fig . 20. Percentage Disease Infection of best-performed F₁ hybrids along with parents and check (December 2002 – March 2003)



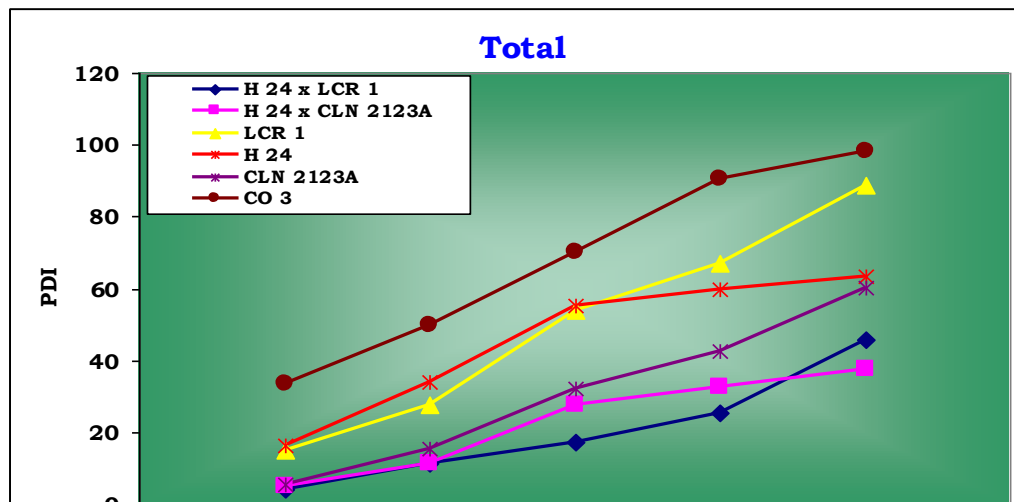
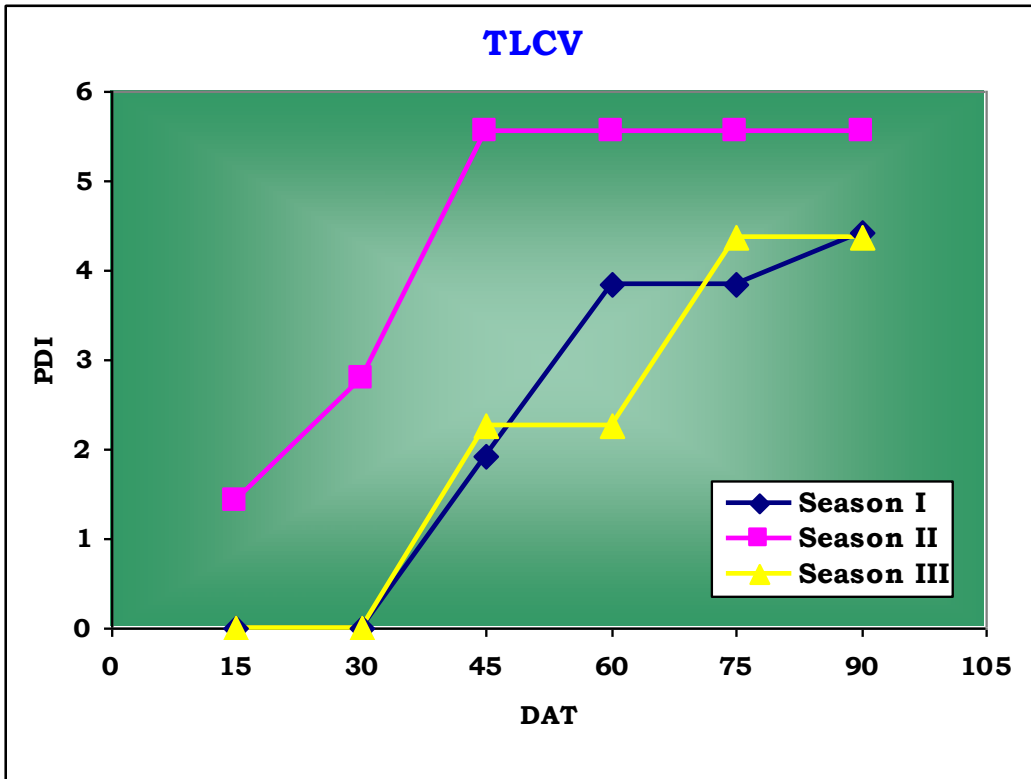
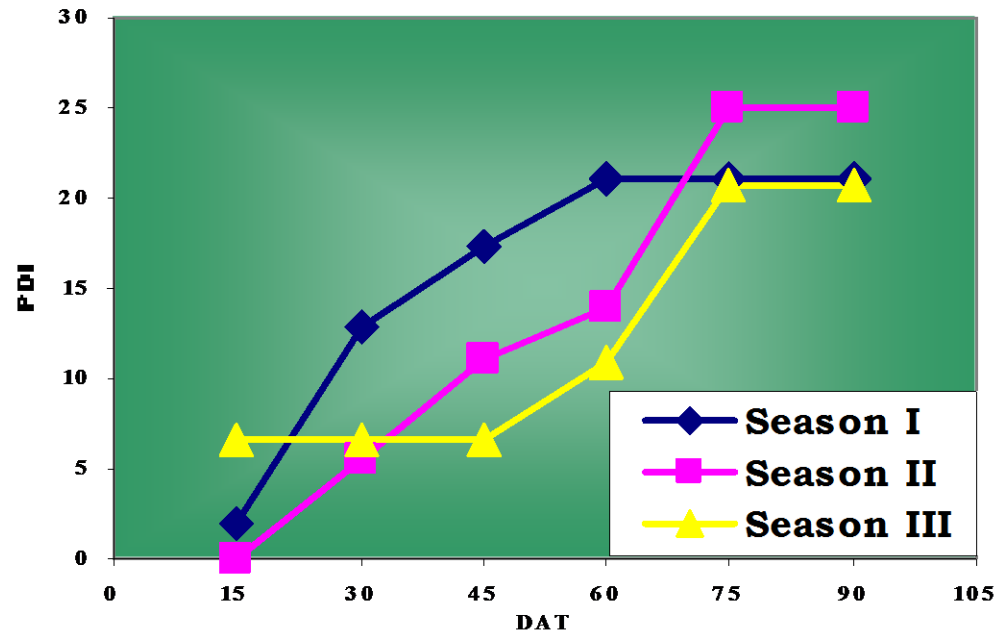
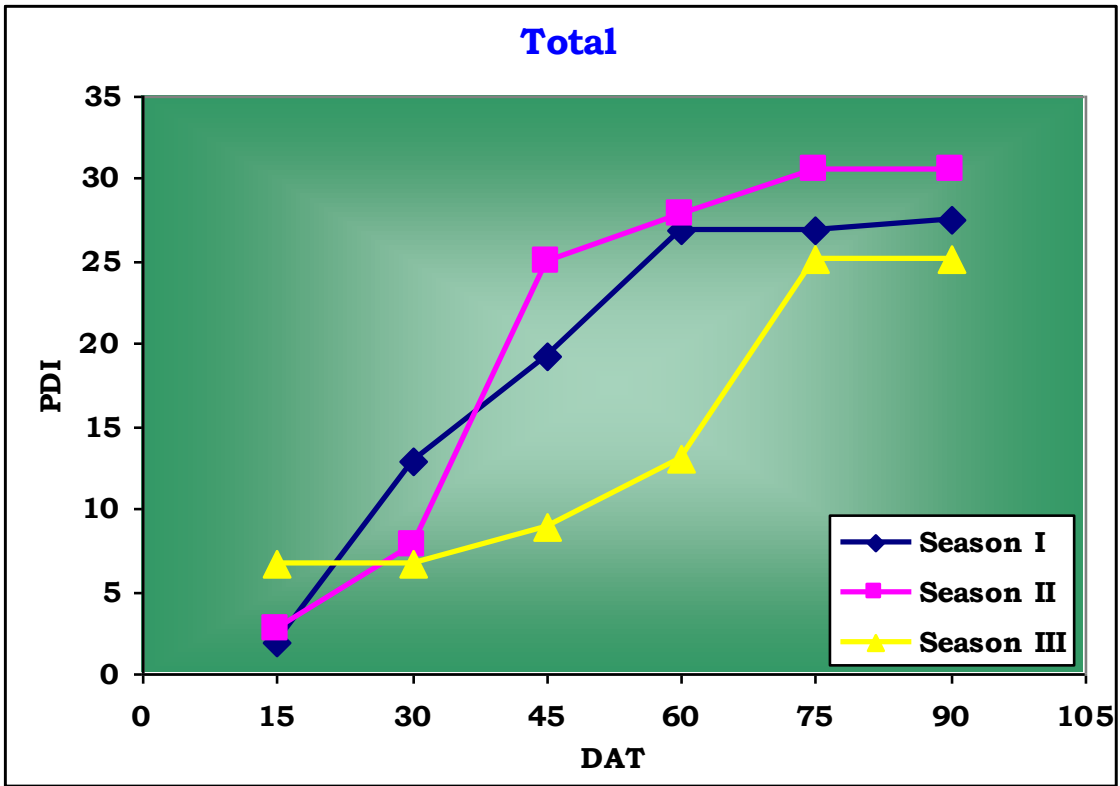


Fig . 19. Percentage Disease Infection of selected F₁ hybrids in different seasons

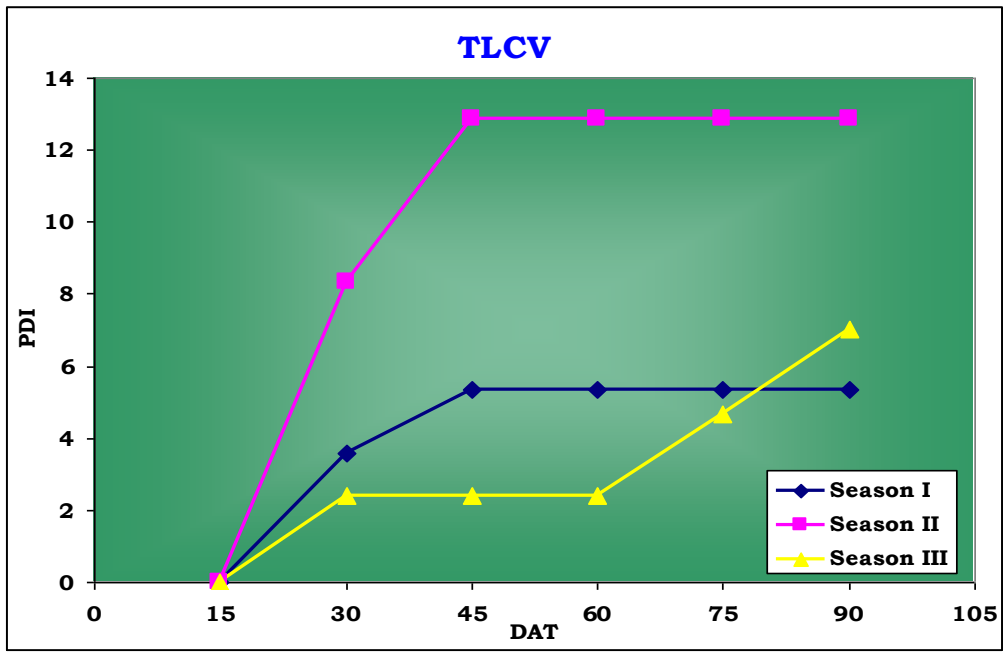


Tv

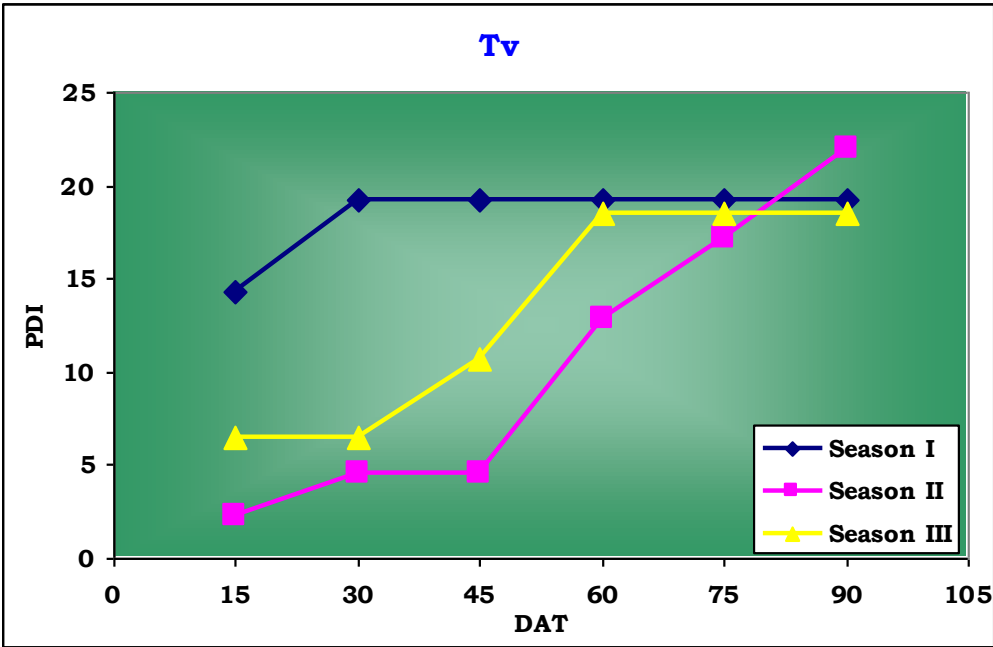


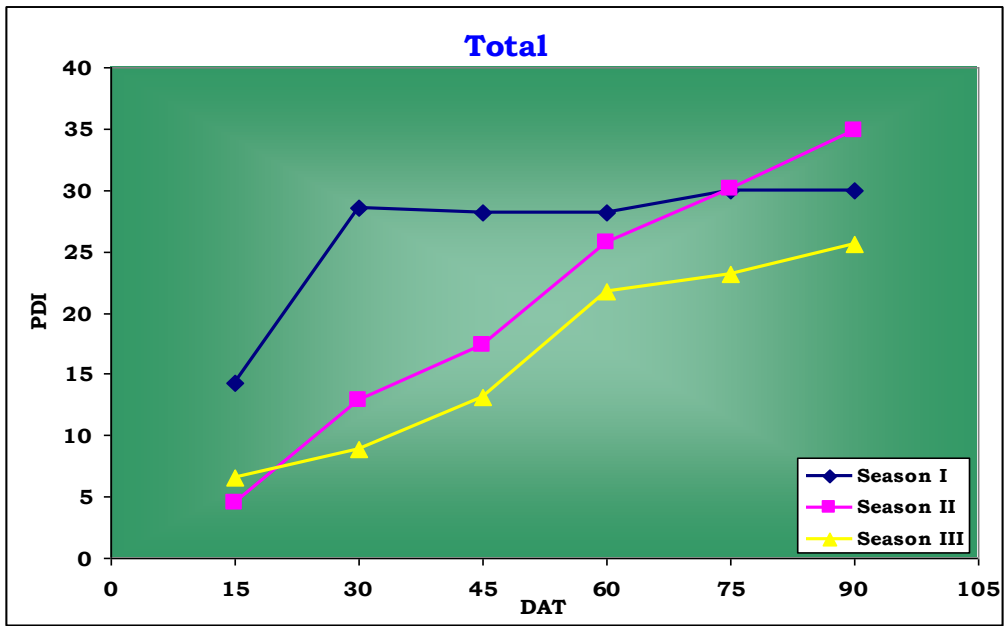


LCR 1 x H 24



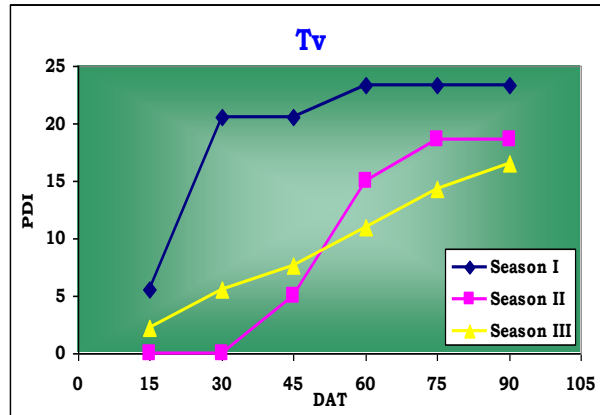
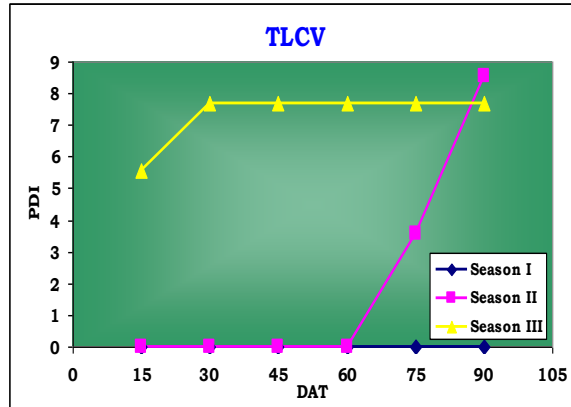
Tv

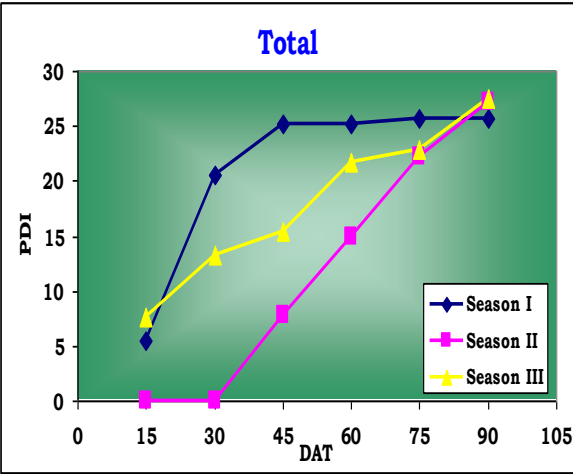




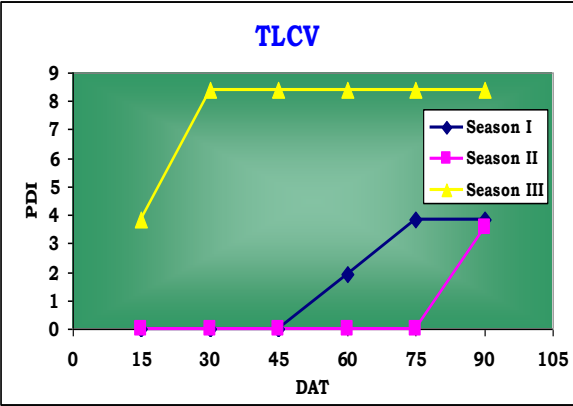
LCR 1 x LE 415

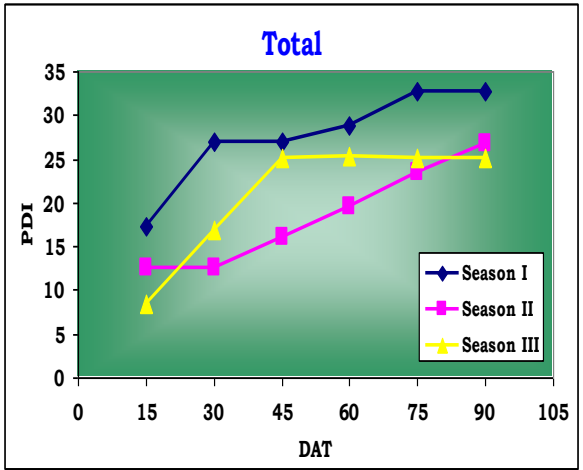
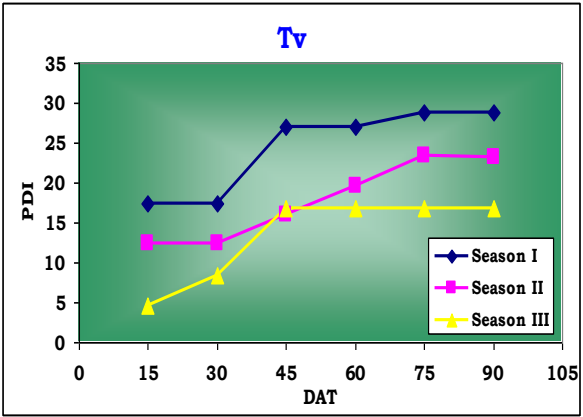
Fig . 19. Percentage Disease Infection of selected F₁ hybrids in different seasons –Contd.





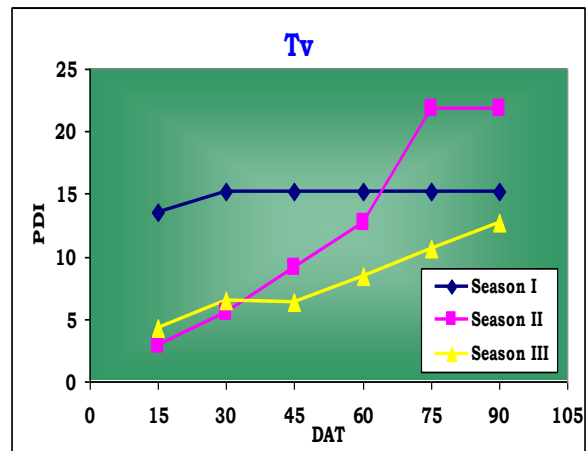
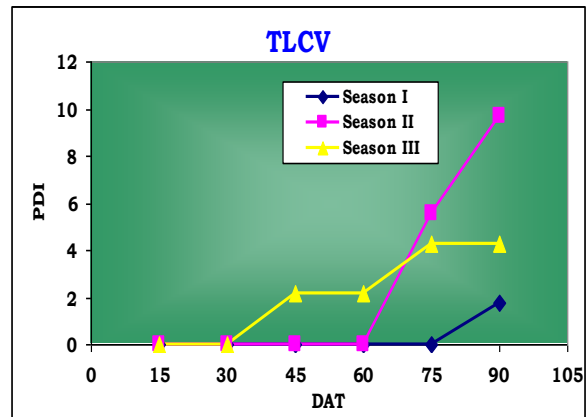
LCR 1 x CLN 2123A

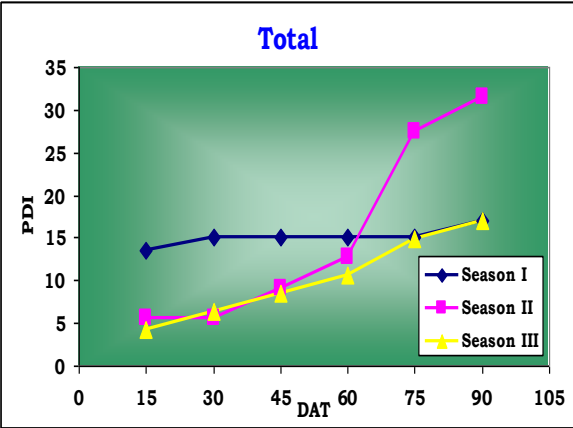




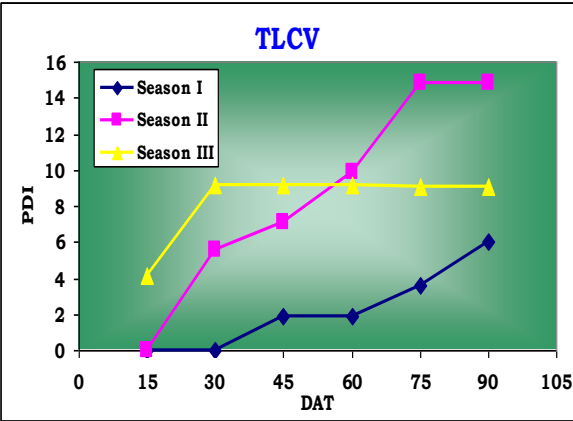
CLN 2123A x LCR 9

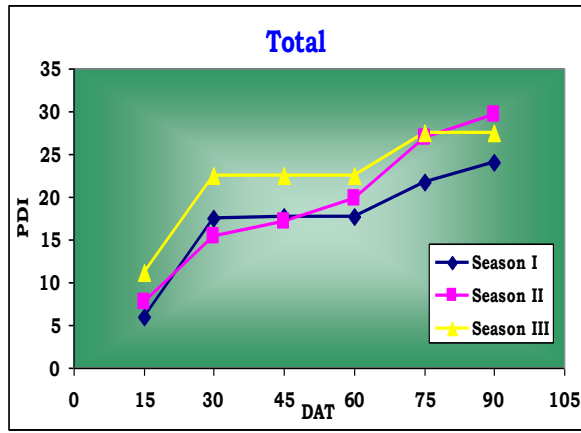
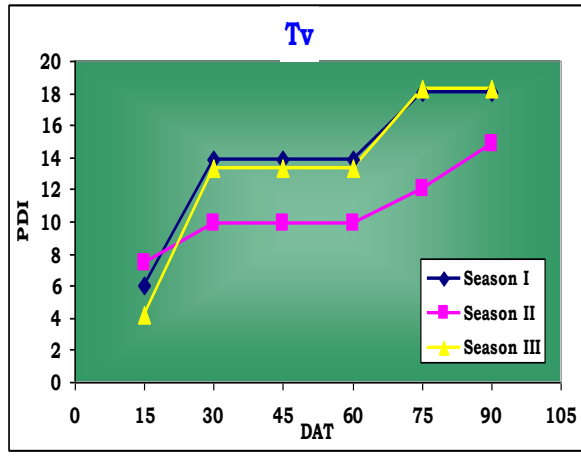
Fig . 19. Percentage Disease Infection of selected F₁ hybrids in different seasons –Contd.





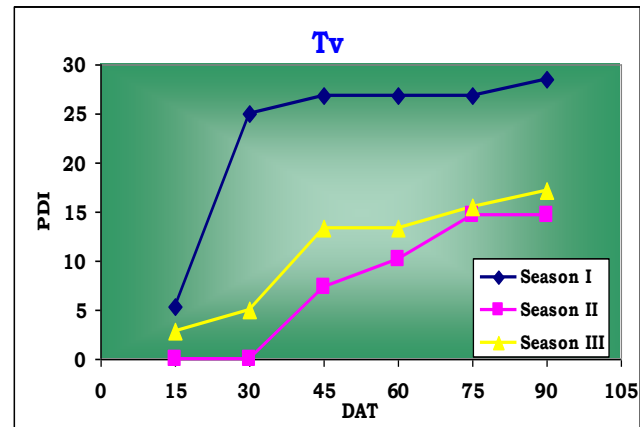
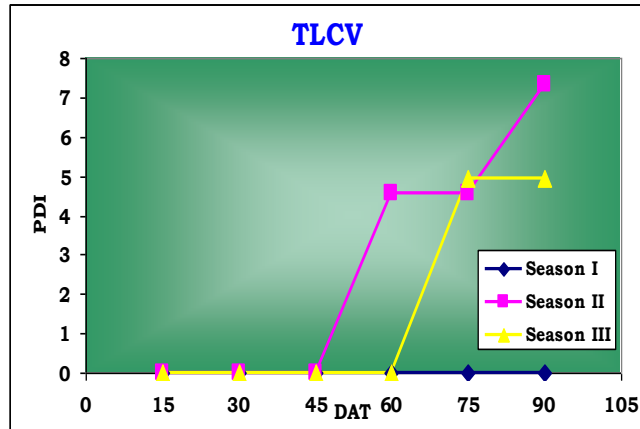
H 24 x LCR 1

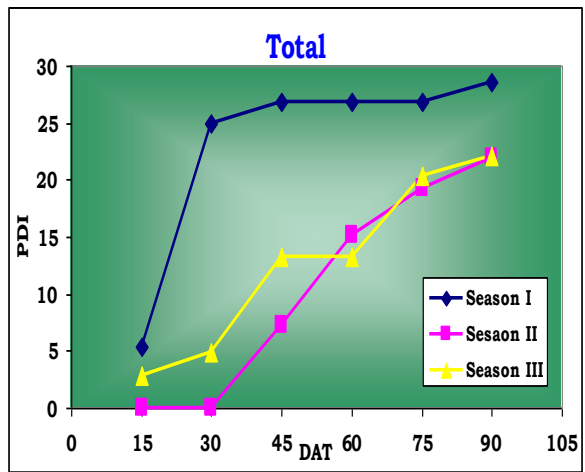




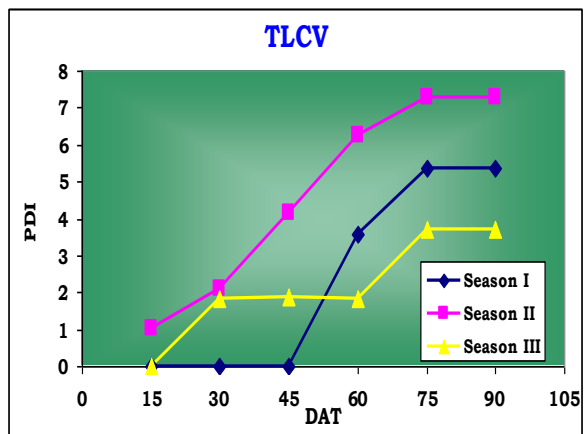
H 24 x LCR 9

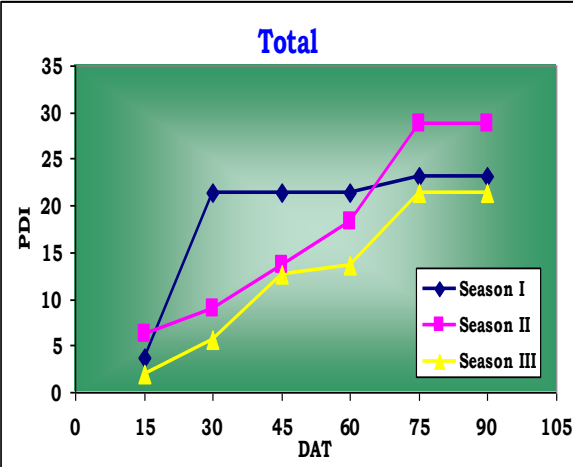
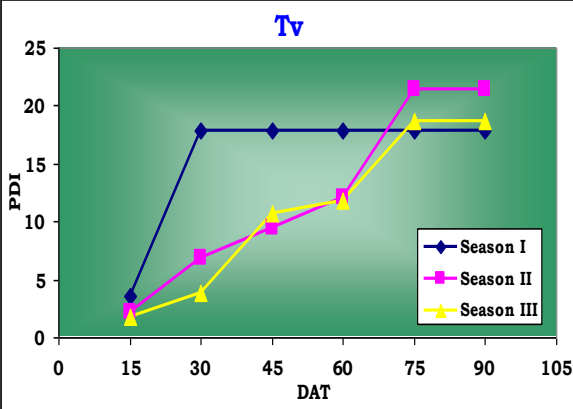
Fig . 19. Percentage Disease Infection of selected F₁ hybrids in different seasons –Contd.



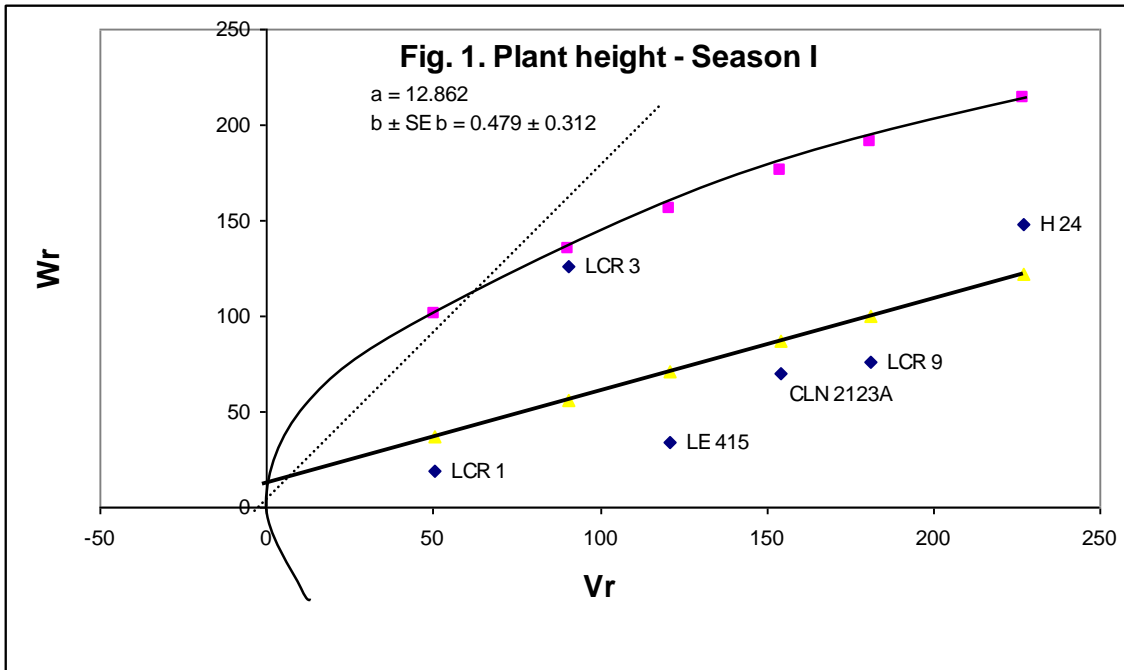


H 24 x CLN 2123A





LE 415 x LCR 1



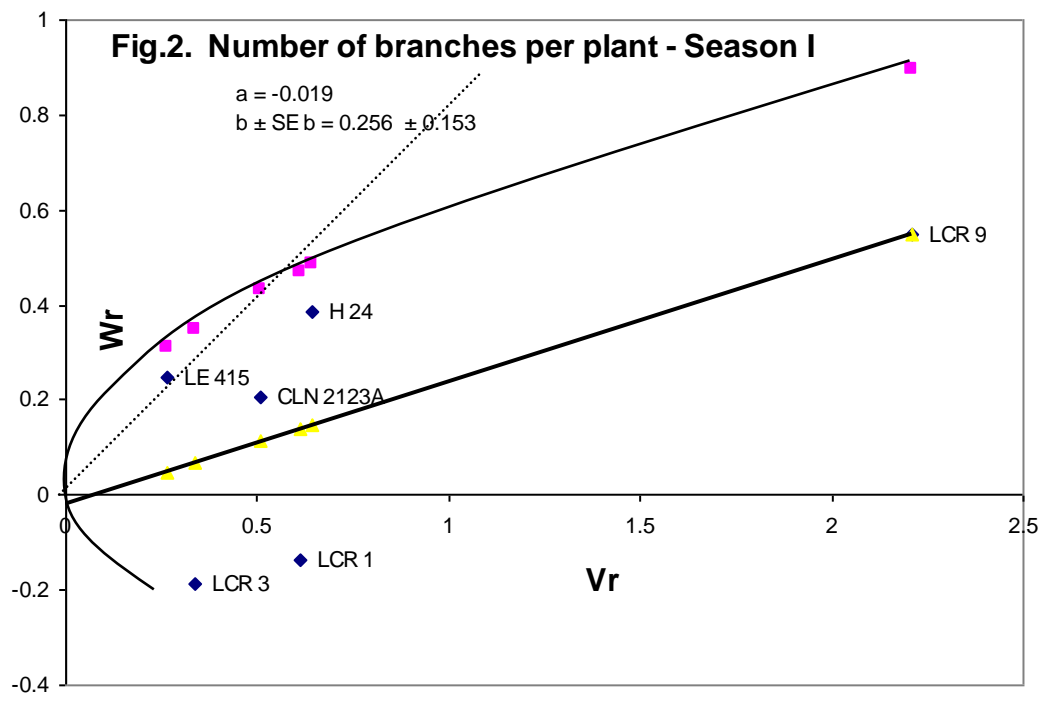


Fig. 3. Days to 50 per cent flowering - Season I

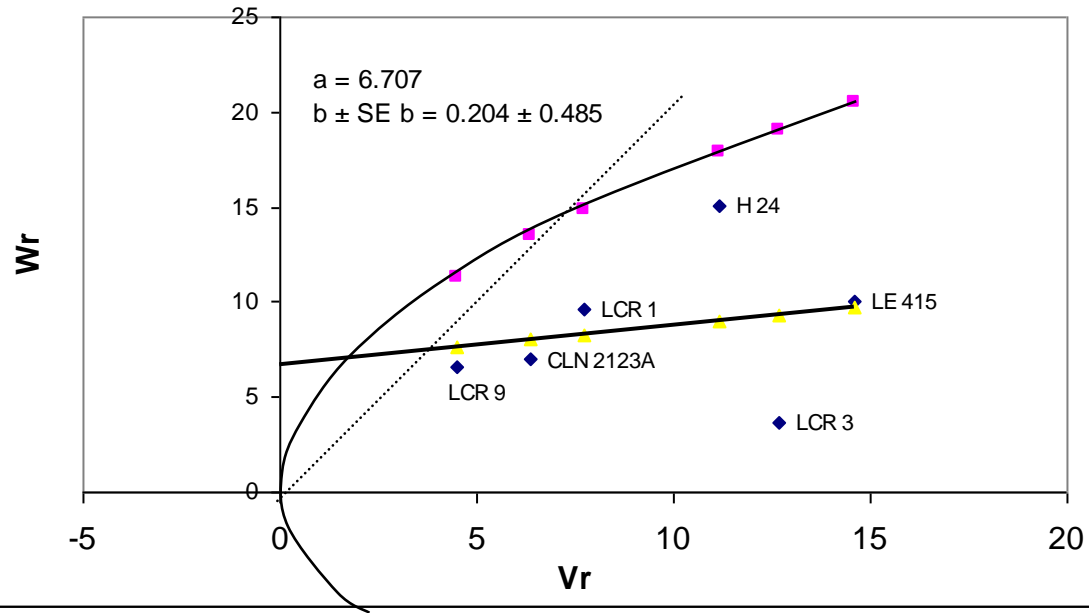


Fig. 4. Number of fruits per plant - Season I

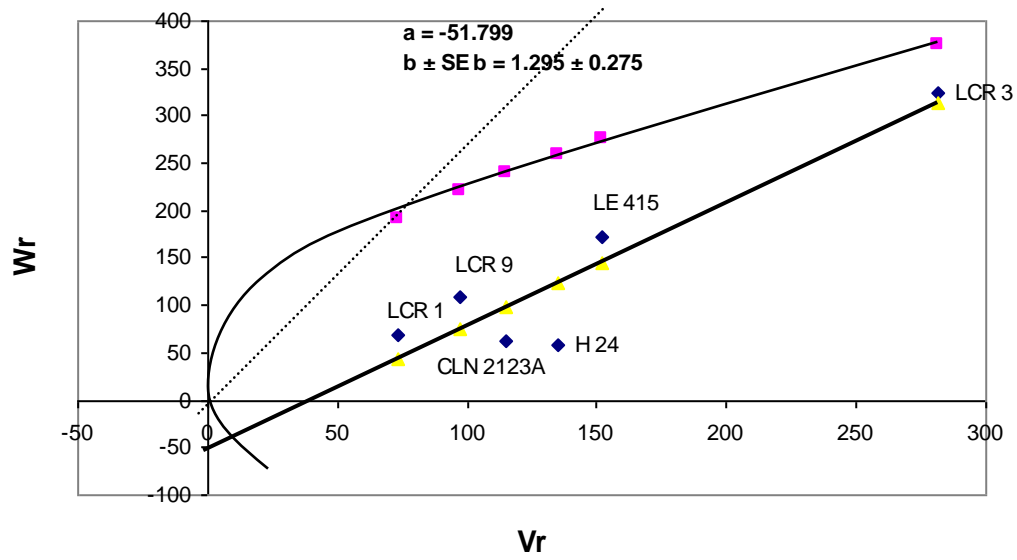


Fig. 5 Fruit weight - Season I

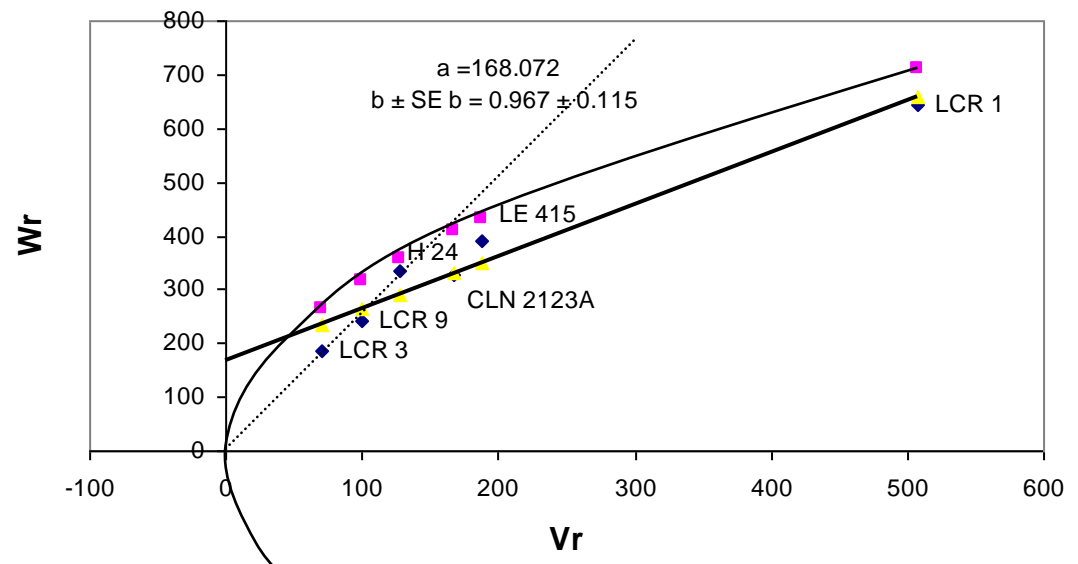


Fig. 6. Yield per plant - Season I

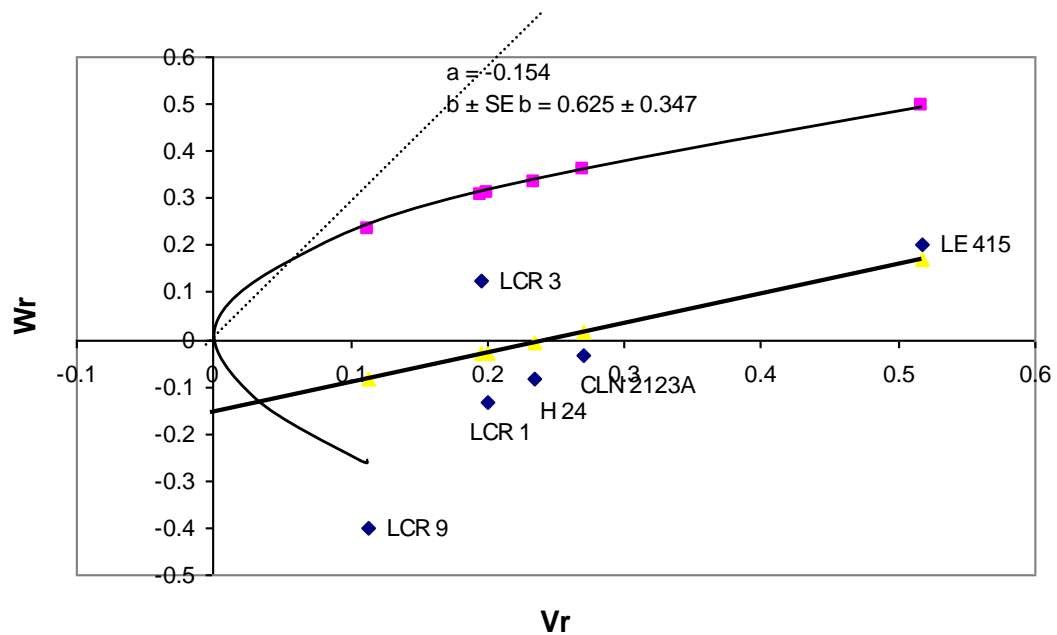


Fig. 7. TSS - Season I

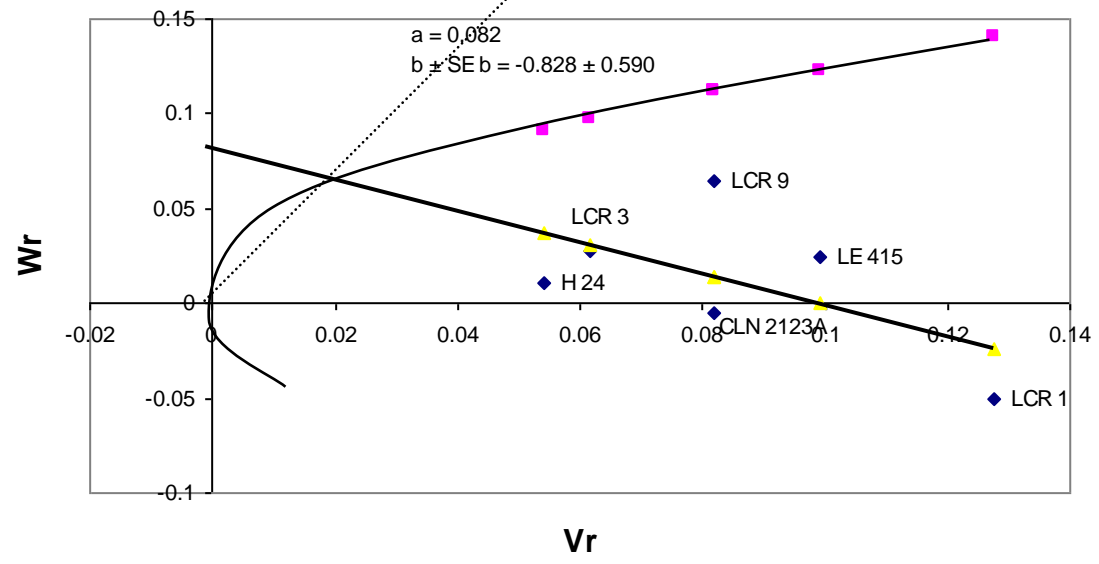


Fig. 8. Acidity - Season I

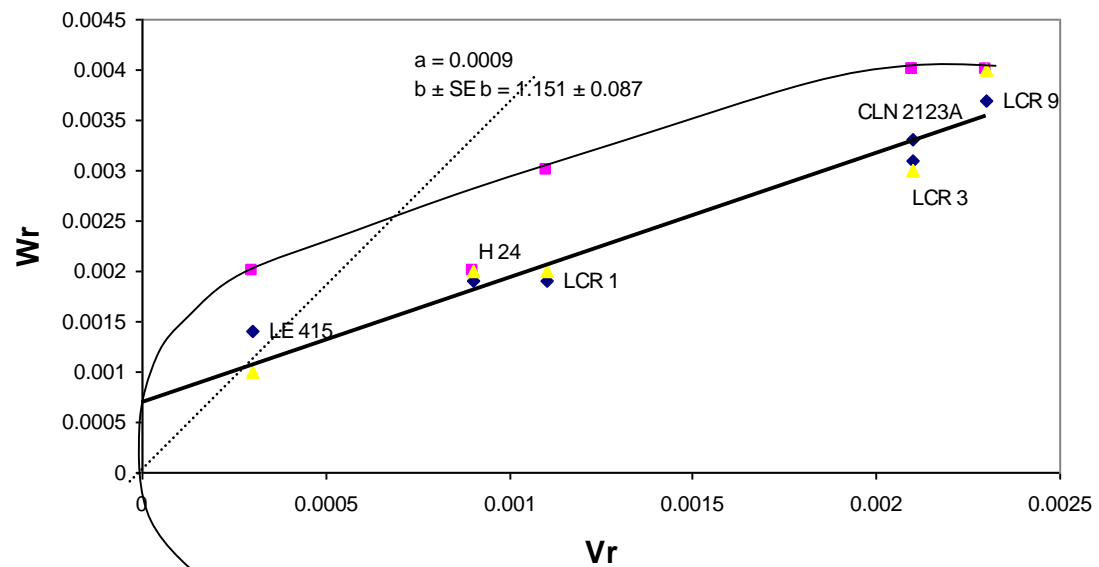


Fig. 9. Ascorbic acid - Season I

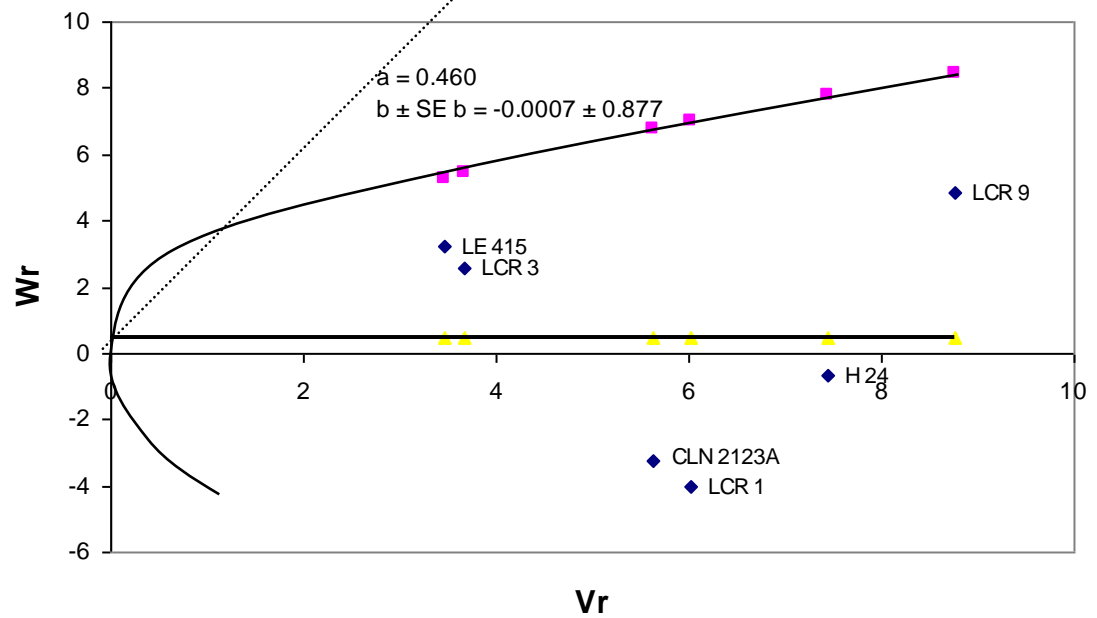


Fig. 10. Lycopene - Season I

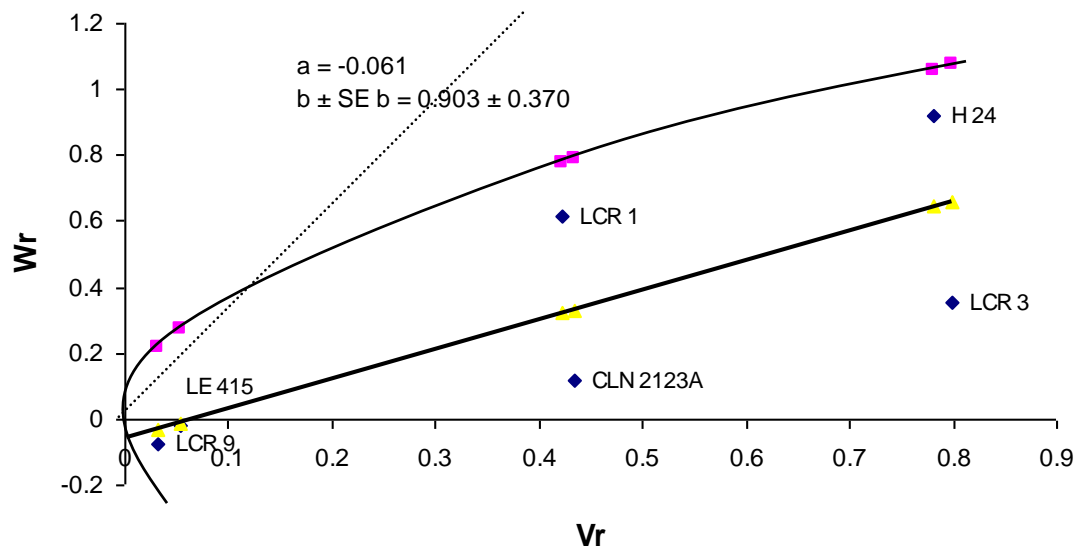


Fig.11. Total phenol - Season I

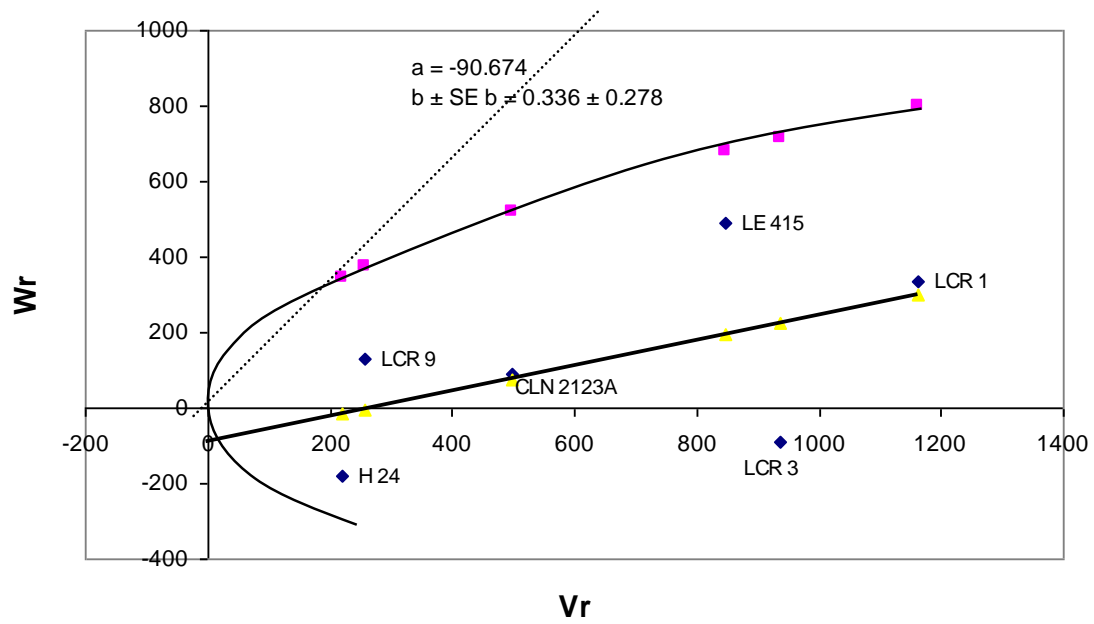


Fig. 12. OD phenol - Season I

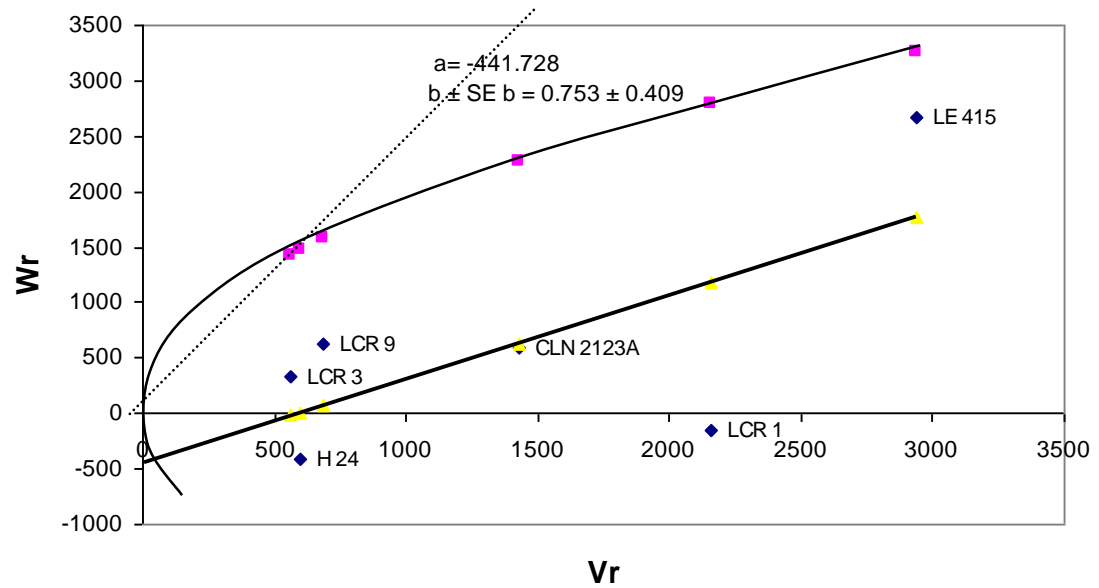


Fig. 13. TLCV incidence at 75 DAT - Season I

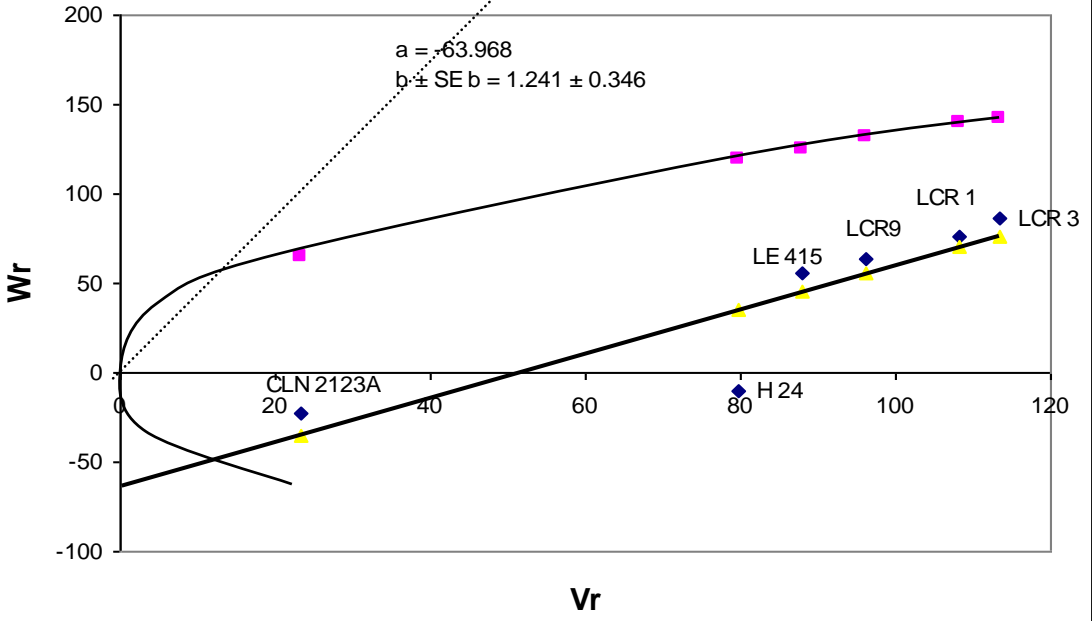


Fig. 14. Tv incidence at 75 DAT - Season I

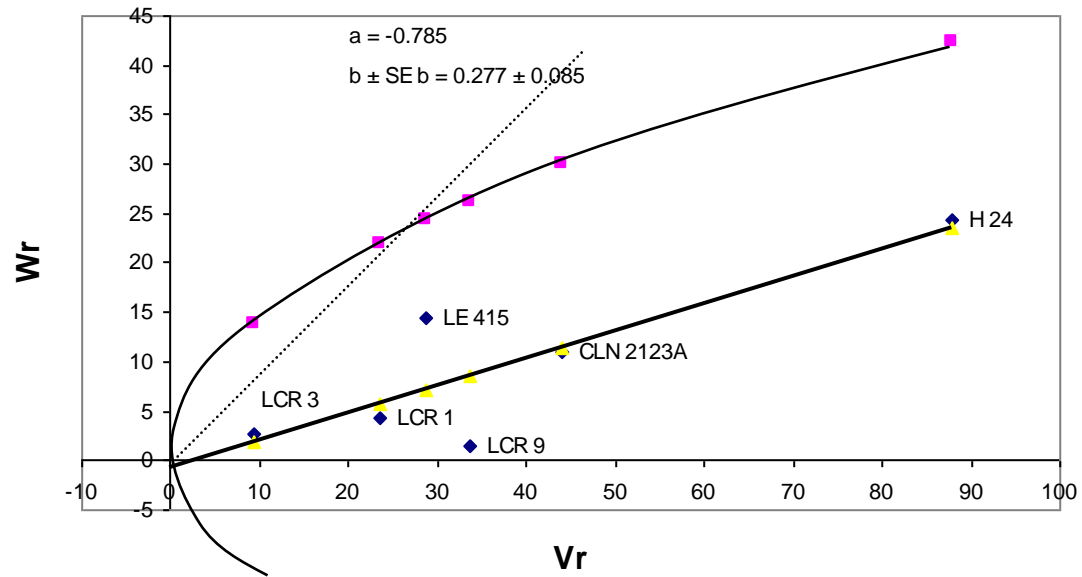


Fig. 15. Total disease incidence at 75 DAT - Season I

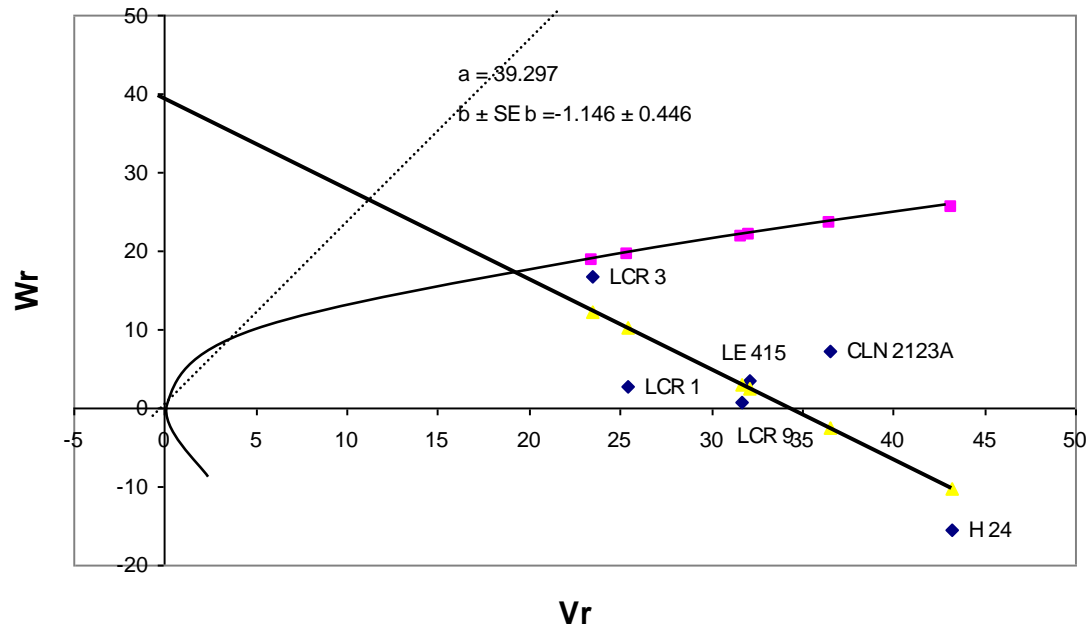


Fig. 16. TLCV incidence at 75 DAT - Season II

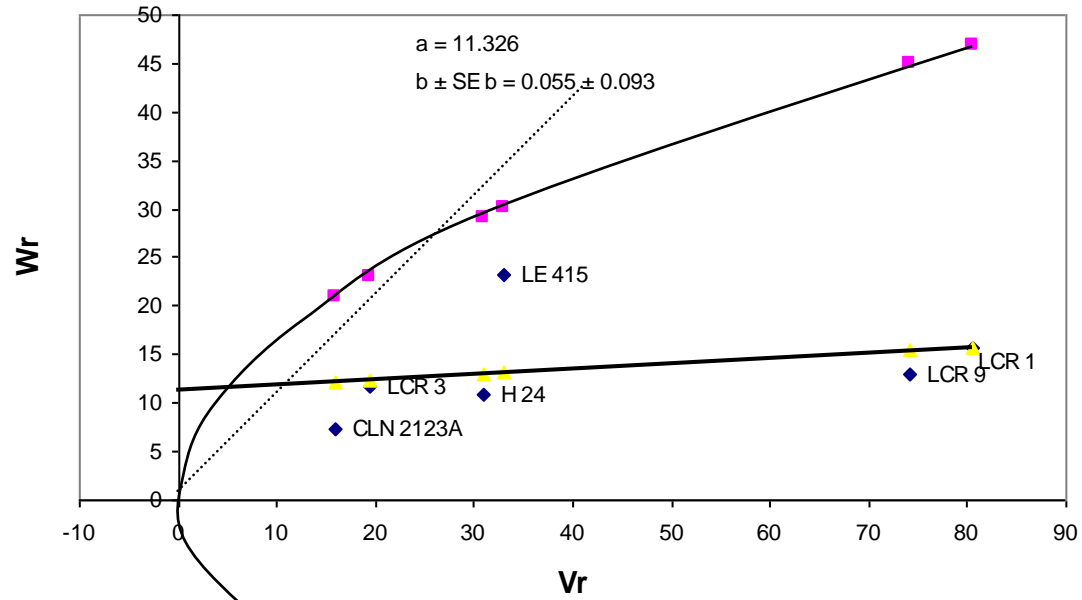


Fig. 17. Tv incidence at 75 DAT - Season II

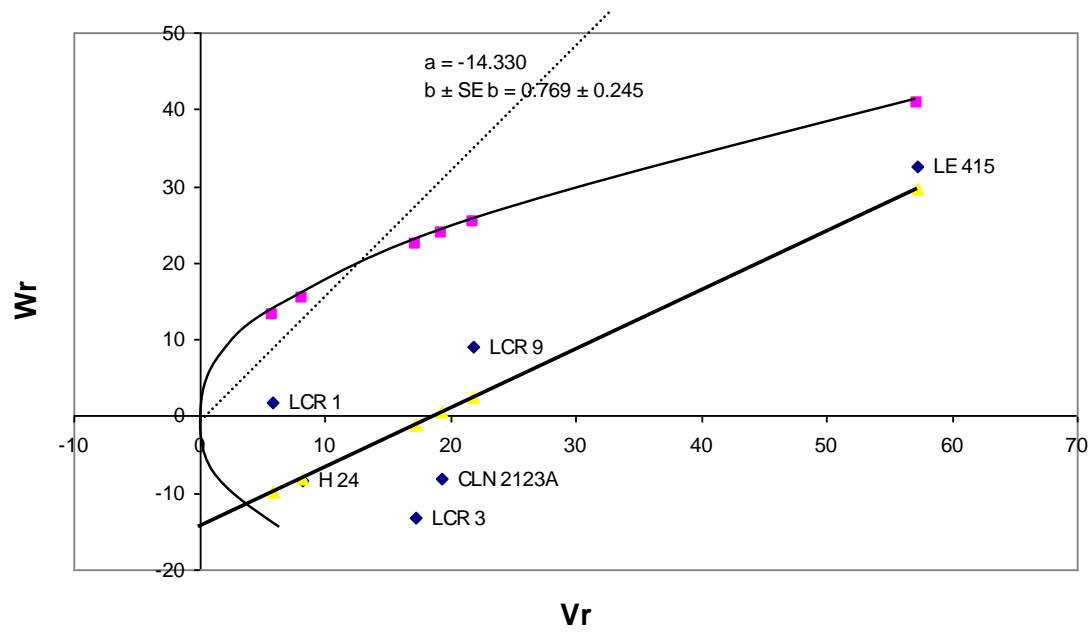


Fig. 18. Total disease incidence at 75 DAT - Season II

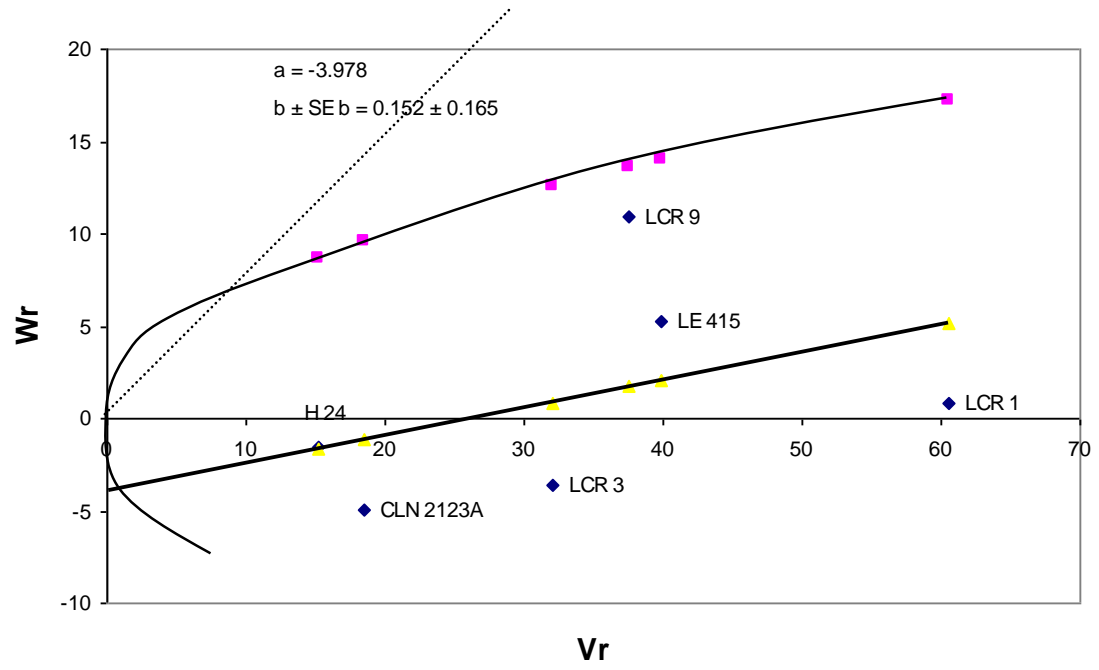
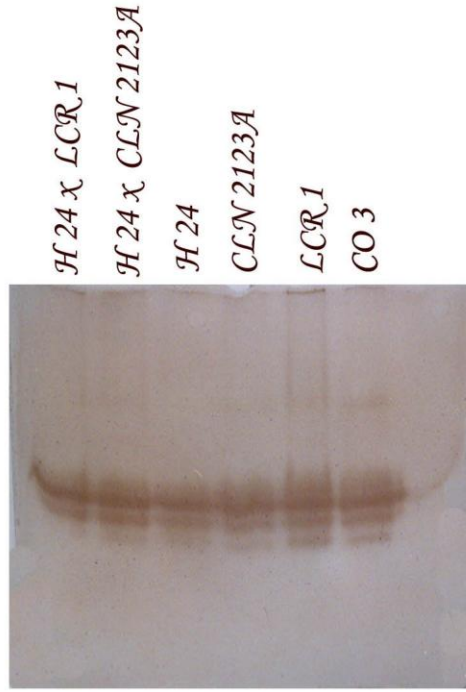
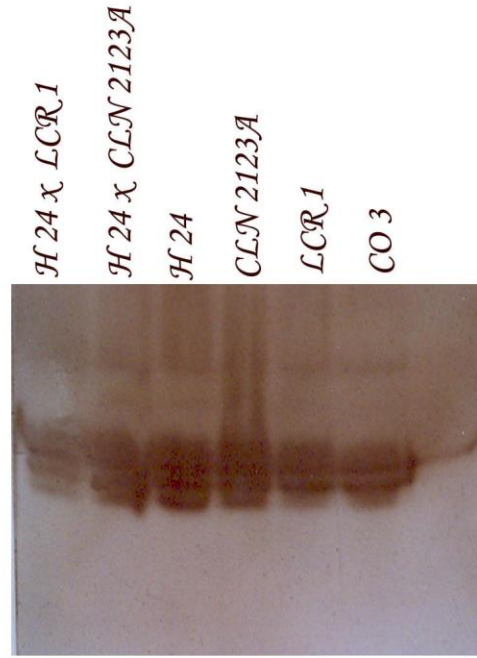


Plate 10. Isozyme banding pattern of polyphenol oxidase



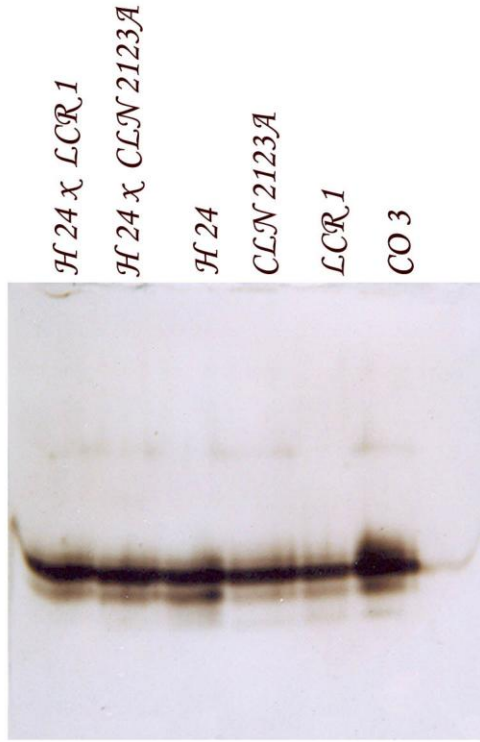
Before graft inoculation



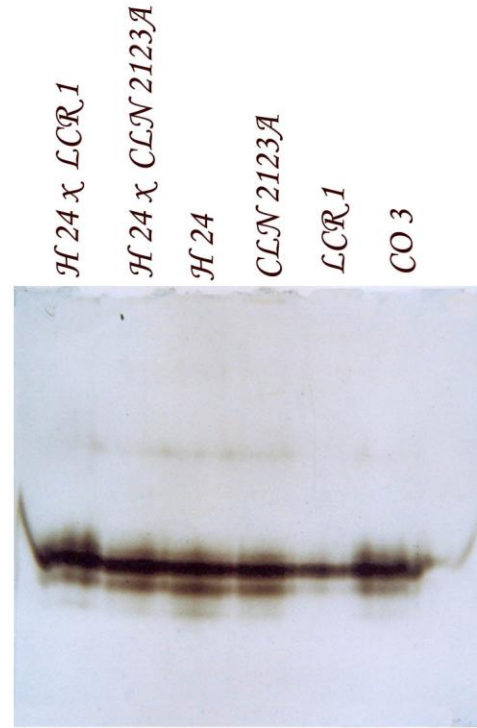
R 1
N 2123A

A

Plate 10. Isozyme banding pattern of peroxidase



Before graft inoculation



R 1
N 2123A
A

*Plate 9. Physical manipulation of source- sink relationship
in the hybrid H 24 x CLN 2123A*



Nipping



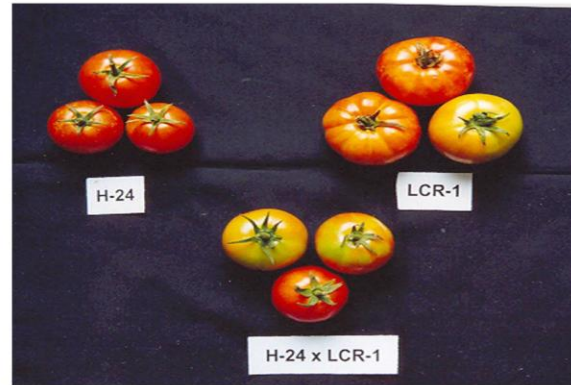
Flower thinning



Plate 8. Promising F₁ hybrids for combined resistance to TLCV and Tv under unprotected condition



H 24 x LCR 1

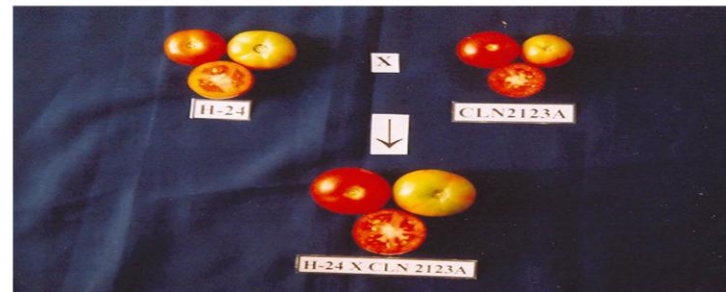


PDI : 19.13

Yield : 2.43kg / plant



H 24 x CLN 2123A



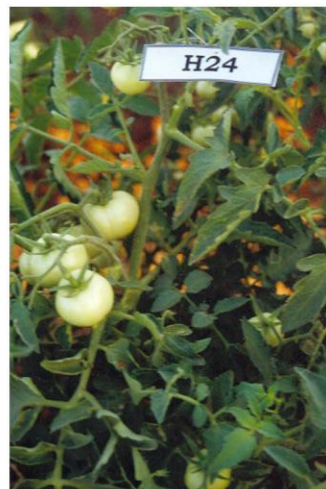
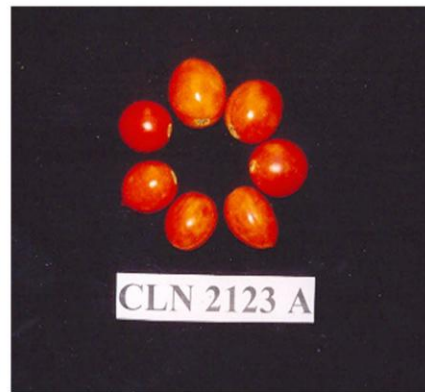
PDI : 22.13

Yield : 3.16 Kg/ plant

Plate 7. Parents selected for hybridisation (Cont...)



CLN 2123A



H 24

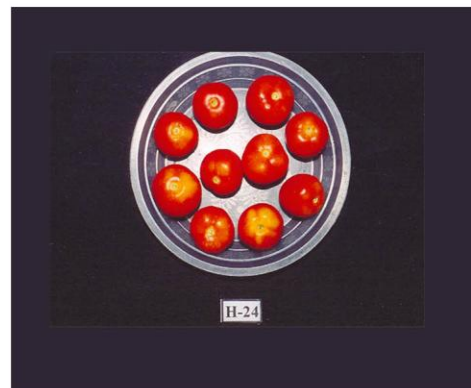


Plate 7. Parents selected for hybridisation



LCR 9



LCR 1



Plate 6. Steps in graft - inoculation of TLCV



A slanting cut is made in the stem



Insertion of TLCV infected tissue



Inserted tissue tied with polythene strip



Graft - inoculated plant



Grafted plants covered with polyethene bag

Plate 5. Characteristics of Tv-Necrotic symptom



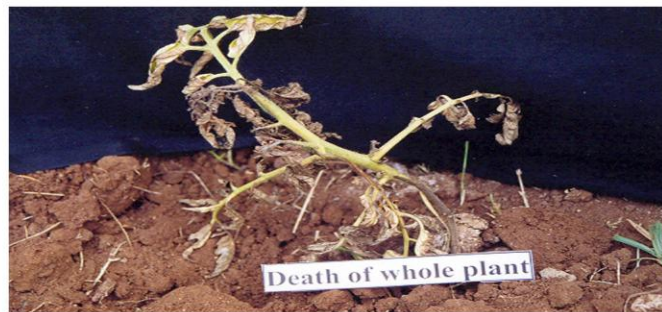
Necrotic lesion



*Stem necrosis -
initial stage*



*Stem necrosis -
advanced stage*



Death of whole plant

Plate 3. Characteristic symptom of TLCV

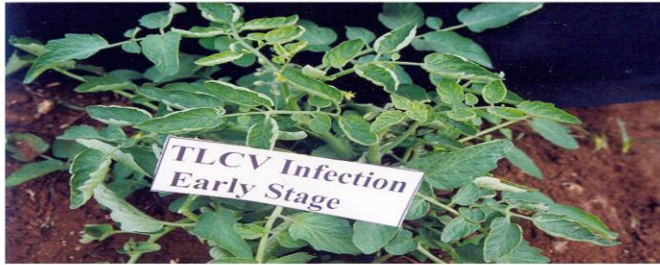


Plate 4. Characteristics of T_{sw}- bronzing symptom

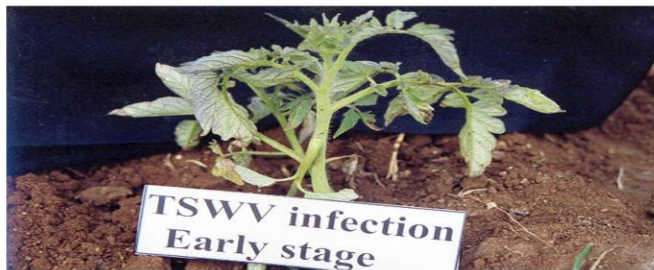


Plate 1. Field severely infected with TLCV



Plate 2. Field view of the experimental plot



Fig. SDS - PAGE analysis for crude proteins in best performed hybrids

