Madras Agric. J., 93 (7-12) : 239-247 July-December 2006

https://doi.org/10.29321/MAJ.10.100758

Heterosis and combining ability studies in tomato *(Lycopersicon esculentum* Mill.) with an emphasis to virus resistance

K. PRADHEEP, D. VEERARAGAVATHATHAM AND J. AUXCILIA
Horticultural College and Research Institute, Tamil Nadu Agrl. University, Coimbatore - 641 003.

Abstract : Heterosis and combining ability analysis were made on 30 F_1 hybrids, obtained through direct and reciprocal crosses of six parents in two seasons during 2002. Majority of the hybrids exhibited unfavorable heterotic response and reciprocal differences for all the traits studied, were observed. Eleven out of 30 hybrids recorded heterobeltiosis for yield per plant. The hybrids LCR 9 x H 24, H 24 x LCR 9 and H 24 x LCR 1 registered heterosis over the best parent in respect of total disease resistance. Additive gene action played an overall role in the inheritance of fruit weight, total and ortho-dihydroxy phenol (OD) and tomato leaf curl virus (TLCV) resistance. The parent H 24 possessed high favorable *gca* for five out of six characters *viz.,* yield, total and OD phenol, TLCV and Tospovirus (Tv) resistance followed by CLN 2123A for total and OD phenol and TLCV resistance. The hybrid H 24 x CLN 2123A exhibited high heterosis and *sca* for yield, total phenol, TLCV and Tv resistance. Influence of modifiers and environment on the expression of resistance to viral diseases by the hybrids was evident. The hybrids showing favorable *sca* for the studied traits seldom had high x high *gca* parental combinations.

Key words: Tomato, heterosis, combining ability, tomato leaf curl virus, tospovirus

Introduction

In southern India, tomato *(Lycopersicon esculentum* Mill.) is frequently challenged by two serologically different viruses namely Tomato Leaf Curl Virus (TLCV) and a Tospovirus (Tv) often mistaken for Tomato Spotted Wilt Virus (TSWV) as the symptoms are similar. It was reported that most of the resistant traits in tomato are controlled by single dominant gene and it is possible to develop F_1 hybrids accumulating several such genes (Rick and Butler, 1956; Kalloo, 1986). In the F_1 hybrids, the effect of heterosis is expected to show increased vigor, size, early and total yield, resistance to diseases and unfavorable conditions *etc.* An appreciable improvement in these aspects can be achieved when donor for resistance with good combining ability for yield is identified. The ability of the hybrids to resist diseases depends on the degree of resistance found in either one or both the parents. Combining ability analysis following the diallel technique is used for testing the performance of the lines in a hybrid combination and also for characterizing the nature and magnitude of gene action involved in controlling a quantitative trait. Superior cross combinations can also be identified by this technique. In the light of above context, an attempt was made.

Table 1. Heterosis (%) for various traits over better and best parents under study

Cross	Fruit weight[a]		Yield per plant[a]		Total phenol[a]		OD phenol[a]		TLCV incidence (season I)[w]	
	dii	diii	dii	diii	dii	diii	dii	diii	dii	diii
LCR 9 x LCR1	-46.95**	-46.95**	-35.98**	-40.79**	-4.21	-12.88**	0.56	-46.77**	18.46	4436.21**
LCR9\LCR3	-34.04**	-58.19**	-33.08**	-33.08**	-4.14	-18.24**	-19.38**	-57.10**	3424.13**	3424.13**
LCR9xCLN2123A	-9.22**	-42.45**	12.98**	-16.54**	-7.49**	-16.46**	-24.99**	-44.70**	0.00	0.00
LCR 9 x H 24	-23.40**	-51.45**	18.51**	-31.39**	-4.21	-10.26**	-7.75*	-36.38**	2687.93**	2687.93**
LCR9xLE415	-20.57**	-49.65**	56.60**	-15.23**	-15.89**	-15.89**	-55.98**	-55.98**	3943.10**	3943.10**
LCR1 XLCR9	-26.94**	-26.94**	-22.56**	-28.38**	-4.69	-13.32**	32.53**	-29.84**	0.00	3729.31**
LCR 1 x LCR 3	-69.16**	-69.16**	-59.02**	-59.02**	-30.79**	-37.05**	-26.41**	-60.84**	5663.79**	5663.79**
LCR1 xCLN2123A	-37.51**	-37.51**	2.24	-5.45	-19.8!**	-27.07**	-71.81**	-79 22**	0.00	0.00
LCR 1 x H 24	-37.51**	-37.51**	-12.60**	-19.17**	-1.50	-7.73**	-2.54	-32.79**	1851.72**	1851.72**
LCR1 xLE415	-43.35**	-43.35**	6.50	-1.50	-3.33	-3.33	-42.46**	-42.46**	2208.62**	2208.62**
LCR 3 x LCR 9	-21.28**	-50.10**	-19.17**	-19.17**	-1.55	-16.63**	-19.25**	-57.03**	5615.52**	5615.52**
LCR 3 x LCR 1	-53.03**	-53.03**	-22.93**	-22.93**	-17.56**	-25.02**	-30.36**	-62.94**	4144.83**	4144.83**
LCR3xCLN2123A	-42.80**	-76.08**	-48.31**	-48.31**	-26.77**	-33.87**	-58.75**	-69.59**	2127.59**	2127.59**
LCR 3 x H 24	1.33	-65.83**	-43.98**	-43.98**	4.69	-1.93	-17.20**	-42.90**	4655.17**	4655.17**
LCR3xLE415	-19.39**	-76.08**	-55.83**	-55.83**	-30.55**	-30.55**	-69.35**	-69.35**	3468.97**	3468.97**
CLN2123AxLCR9	3.55**	-34.36**	13.49**	-16.17**	-15.37**	-23.57**	-28.19**	-47.06**	1851.72**	1851.72**
CLN2123AxLCRl	-50.55**	-50.55**	0.00	-7.52*	-20.56**	-27.75**	-43.80**	-58.57**	2574.13**	2574.13**
CLN2123AxLCR3	-9.68**	-62.24**	-43.80**	-43.80**	-5.76*	-14.89**	-16.32**	-38.31**	2834.48**	2834.48**
CLN2123AxH24	11.83**	-53.24**	30.79**	-3.38	-15.74**	-21.07**	-28.36**	-47.19**	2358.62**	2358.62**
CLN2I23AxLE415	-8.60**	-61.79**	-34.61**	-51.69**	-27 26**	-27.26**	-57.69**	-57.69**	1301.72*	1301.72*
H 24 x LCR 9	-31.21**	-56.39**	84.74**	6.95	-0.54	-6.83**	-25.24**	-48.44**	1777.58**	1777.58**
H 24 x LCR 1	-40.43**	-40.43**	1.63	-6.02	3.30	-3.24	32.13**	-8.88**	0.00	0.00
H 24 x LCR 3	-1.33	-66.73**	-12.78**	-12.78**	3.87	-2.70	0.32	-30.82**	3724.13**	3724.13**
H24xCLN2123A	-16.02**	-64.89**	58.52**	17.11**	7.35**	0.56	-19.19**	-40.43**	0.00	0.00
H24xLE415	12.00**	-62.24**	111.04**	22.18*	-8.60**	-8.60**	-34.63**	-34.63**	1777.59**	1777.59**
LE415xLCR9	-30.50**	-55.94**	42.36**	-22.93**	-24.68**	-24.68**	-43.99**	-43.99**	5463.79**	5463.79**
LE415xLCRl	-44.70**	-44.70**	11.38**	3.01	-8.61**	-8.61**	-48.17**	-48.17**	2208.62**	2208.62**
LE415x LCR 3	-15.76**	-75.00**	-22.56**	-22.56**	-17.85**	-17.85**	-36.07**	-36.07**	3551.72**	3551.72**
LE415xCLN2123A	-10.97**	-62.77**	6.11	-21.62**	-14.25**	-14.25**	-41.87**	-41.87**	3193.10**	3193.10**
LE415xH24	28.00**	-56.84**	37.34**	-20.49**	-24.24**	-24.24**	-51.32** ‘	-51.32**	4208 62**	4208.62**

Table 1. Contd...

Cross	Fruit weight[a]		Yield per plant[a]		Total phenol[a]		OD phenol[a]		TLCV incidence (season I)[w]	
	dii	diii	dii	diii	dii	diii	dii	diii	dii	diii
LCR9xLCR1	26.74	26.74	29.74**	50.63**	62.25*	123.53**	22.49	85.72**	41.64**	42.36**
LCR9 x LCR 3	35.97*	35.97**	35.57**	35.57**	75.11**	141.25**	11.82	69.53**	40.57**	41.28**
LCR9xCLN2123A	80.82**	80.82**	37.59**	44.41**	19.97	19.97	80.54**	173.72**	57.16**	57.96**
LCR9 x H 24	-20.76	-20.76	-12.74	-2.26	-96.74**	-95.36**	13.83	72.58**	-9.07*	-8.60*
LCR9x LE415	44.98**	44.98**	28.54**	49.23**	-96.74**	-95.36**	125.57**	125.57**	19.46**	19.46**
LCR1 x LCR 9	29.67*	29.67*	16.87	35.68**	68.54**	132.20**	18.20	79.21**	41.64**	42.36**
LCR1 x LCR 3	0.00	0.00	61.70**	61.70**	52.98*	132.20**	-13.31	50.24**	13.38**	29.74**
LCRl x CLN2123A	30.17*	30.17*	4.18	9.34	-15.71	-15.71	-13.37	52.51**	-20.97**	-11.20**
LCR1 x H 24	23.32	23.32	2.79	19.33	-30.27	5.57	15.79	79.21**	0.48	6.17
LCR1 xLE415	17.29	17.29	2.79	2.79	-7.24	62.77	46.24*	46.24*	5.16	5.16
LCR3 x LCR 9	21.93	21.93	62.99**	62.99**	24.72	71 83*	35.89**	106.03**	35.06**	35.75**
LCR3 x LCR 1	-8.73	-8.73	17.89	17.89	52.98**	132.00	30.16**	125.57**	52.72**	74.75**
LCR3xCLN2123A	31.24*	64.39**	41.79**	41.79**	0.00	0.00	7.17	85.72**	-3.58	8.35*
LCR 3 x H 24	-14.05	6.39	35.68**	35.68**	14.78	73.76*	15.79	79.21**	16.74**	23.35**
LCR3xLE415	43.31**	79.51**	76.50**	76.50**	13.03	71.90*	181.48**	181.48**	75.70	75.70**
CLN2123AxLCR9	46.29**	46.29**	19.38	25.30*	-95.51**	-95.51**	13.91	72.70**	-9.00*	-8.54*
CLN2123AxLCRl	79.47**	79.47**	50.56**	58.03**	5.57	5.57	-16.32	47.31**	-19.40**	-9.43*
CLN2123AxLCR3	-22.29	-2.66	0.86	0.86	105.65**	105.65**	14.4S	100.0**	26.69**	42.36**
CLN2123AxH24	80.99**	138.00**	93.63**	103.23**	68.89*	68.89*	31.61**	103.70**	26.53**	33.69**
CLN2123AxLE415	77.82**	133.86**	81.89**	90.20**	0.00	0.00	122.64**	122.64**	27.17**	27.17**
H 24 x LCR 9	13.24	13.24	-10.78	-0.07	27.19	75.23*	-19.86	21.51	-3.61	-1.11
H 24 x LCR 1	3.15	3.15	-26.50*	-17.68	-30.27	5.57	7.49	66.37**	-5.54	-0.19
H 24 x LCR 3	-15.24	6.17	19.87	19.87	27.97	93.73**	-18.22	26.58	1.23	6.96
H24xCLN2123A	6.68	40.30**	6.68	11.96	-4.64	-4.64	-13.16	34.41	-22.22**	-17.81**
H24xLE415	-0.03	40.30**	7.28	20.16	-10.28	17.63	67.68**	67.68**	7.56	7.56
LE415xLCR9	7.02	7.02	40.76**	63.42**	38.15	90.33	46.89**	46.89**	14.01**	14.01**
LE415xLCRl	12.56	12.56	-10.83	3.52	-20.35	21.13	64.99**	64.99**	2.63	2.63
LE415x LCR 3	-51.26**	-38.95*	-8.37	-8.37	38.78	111.07**	76.52**	76.52**	33.88**	33.88**
LE415xCLN2123A	2.70	35.07*	25.44*	31.66**	26.24	26.24	87.75**	87.75**	14.90**	14.90**
LE415xH 24	3.66	45.48**	38.72**	55.37**	36.09	106.04**	120.73**	120.73**	53.62**	53.62**

[a]based on one year data ** significance at 1 % level * significance at 5% level [w]worked out for arc sine transformed values

[dii]heterosis over better parent [diii]heterosis over best parent

Materials and Methods

Six varieties/lines of tomato LCR 9, LCR 1, LCR 3, CLN 2123A, H 24 and LE 415 were crossed in a diallel fashion (including reciprocals) and their 30 F_1 hybrids were evaluated along with the parents. Field experiment was conducted at the orchard of Horticultural College and Research Institute, TNAU, Coimbatore during 2002. In the season I (January - April 2002), thirty day old seedlings were raised in randomized block design (RBD) with two replications. The size of the plot was 3.0 x 2.4 m so as to accommodate 25 plants in each replication. Seedlings were planted at a spacing of 60 x 45cm. All the thirty six entities were inter-planted with susceptible variety CO 3 in rows over four sides of the test plants. Recommended package of practices were followed except spraying any pesticides.

Percentage Disease Infection (PDI) of respective viral diseases *viz.,* TLCV and Tv was recorded on 75th day after transplanting (DAT), since further infection may have little influence on yield loss. Total PDI was calculated by adding the PDI values of individual and combined virus infected plants. In respective plots, five randomly selected representative plants were taken for observation and analysis. Parameters such as fruit weight, yield per plant, total phenol (Bray and Thorpe, 1954) and ortho-dihydroxy (OD) phenol (Johnson and Schaal, 1957) were also recorded.

Second season crop was also evaluated similarly during summer *i.e.* March - June 2002. Due to scorching summer, fruit set and fruit size were adversely affected. As they might not represent hybrid yield truly, only PDI was recorded. The PDI values were transformed (arc-sine) before being subjected to analysis.

Heterosis was calculated as the percentage of F_1's performance in the favorable direction over the better and the best parent (dii and diii respectively) for each trait. Significance of heterosis was calculated as suggested by Wynne, Emery and Rice (1970). 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) after validating needed assumptions. For the trait total PDI, combining ability estimation was not worked out as it frequently involved mere arithmetic mean of TLCV and Tv.

Results and Discussion

Most of the crosses, although not all, significantly differed from their better or the best parental values (Table 1). Majority of the hybrids exhibited unfavorable heterotic response and only a few hybrids could be considered for selection. The analysis of variance for combining ability (Table 2 & 3) revealed significant mean squares for general combining ability (GCA), specific combining ability (SCA) and reciprocal combining ability (RCA) for all the traits studied in both the seasons. The mean square value for GCA was higher than SCA for fruit weight, total and OD phenol, TLCV resistance (both seasons) and Tv resistance in season I, whereas that of GCA was higher than RCA for all the traits except Tv incidence in season II, indicating an overall role of additive gene action in the inheritance of these characters, and the possibility of effective simple selection for these traits in the later generations to fix up the trait. A significant reciprocal difference was noticed in majority of the crosses for all the characters studied. As both heterosis and specific combining ability effects *(sca)* show clear picture about

Table 2. Estimates of *gca* effects of parents for various traits under study

Parent	Fruit weight[a]	Yield per plant[a]	Total penol[a]	OD phenol[a]	TLCV incidence (season I)	TLCV incidence (season II)	Tv incidence (season I)	Tv incidence (season II)
LCR9	9.32**	-0.08**	-4.47*	-11.37**	6.33**	-0.54	-2.10*	0.27
LCR1	18.17**	0.15**	0.39	-4.30*	0.94	1.13	-3.15**	-1.36
LCR3	-13.61**	-0.20**	-10.68**	-16.50**	4.22**	5.54**	-3.12**	1.46
CLN 2123A	-1.49**	0.05	-10.04**	-7.15**	-7.62**	-6.05**	6.56**	1.79*
H24	-3.88**	0.21**	22.17**	24.82**	-3.56**	-1.18	0.31	-2.20*
LE415	-8.50**	-0.12**	2.64	14.50**	-0.31	1.10	1.51	0.03
SE (gi)	0.107	0.003	2.084	1.726	1.153	1.239	0.974	0.870
GCA (mean square)	1660.76**	0.32**	1759.90**	3113.93**	310.71**	166.61**	171.69**	28.99**

** Significnat at 1% level * Significant at 5% level [a]based on one year data

the performance of the hybrids for continuously varying traits, both were considered in the selection of desired hybrids.

Fruit weight is an important trait contributing directly to the yield. None of the hybrids recorded supremacy in heterosis over the best parent. Only five out of 30 crosses registered positive heterosis over better parent. With regard to general combining ability effects *(gca),* LCR 9 and LCR 1 proved to be the best combiners (Table 2). Thirteen hybrids registered significant positive *sca,* out of them, seven showed the involvement of at least one of the best combiners. The cross CLN 2123 A x LCR 9 exhibited merit both in heterosis and combing ability analysis.

Yield is a complex character and is dependent on its component traits and their inheritance. Eleven out of 30 hybrids recorded significant positive heterosis estimates over better parental values. Heterosis for yield was reported by various workers (Mandal *et al.,* 1992; Pujari and Kale, 1994; Kumar, Banerjee and Partap, 1995). 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 is the consequence of multiplicative relationship among 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 contributed by number of fruits and fruit weight. Heterosis for total yield can occur in hybrids in which the above attributes merely show dominance or intermediate level

of expression. For this, the parents must differ with regard to the level of expression of each of the components and neither must has a monopoly at high or low expression in both the unit characters. The result of the present investigation justifies the above statement and fall in line with the works of Aruna and Veeraragavathatham (1996).

The parents LCR 1 and H 24 proved to be best combiners for fruit yield in the present study (Table 2). Out of four hybrids which exhibited positive heterosis over the best parent, one of them (H 24 x CLN 2123A) 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 for fruit yield in tomato by Jamwal *et al.* (1984) and Rai *et al.* (1997).

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). Only the hybrid H 24 x CLN 2123A registered positive heterosis over the best parent and another three hybrids namely LCR 3 x H 24, H 24 x LCR 1 and H 24 x LCR 3 exhibited positive heterosis over the better parent. Significant negative heterosis observed in most of hybrids is in line with the findings of Narayana and Reddy (1980) and Singh and Abidi (1988). *Gca* indicated H 24 as best combiner for this trait. Among the above four hybrids, except H 24 x LCR 3, all the other hybrids showed positive *sca* also. Both additive and epistatic gene actions were inferred for this trait from the present study. None of the hybrids showed positive heterosis over the best parent for the trait OD phenol, while only two hybrids (LCR 1 x LCR 9 and H 24 x LCR 1) had significant positive heterobeltiosis (dii). LE 415 and H 24 appeared to be good parents in terms *of gca.* Out of 13 hybrids showing significant *sca,* eight had one of the above parents. But the peculiarity is that both these best combiners in hybrid condition (including reciprocal) yielded the hybrids with negative significant *sca,* indicating the role of non additive gene action of epistatic nature.

Considering the TLCV resistance, in some cases, the hybrid and one or both the parents had zero value; calculation of heterosis over best/better parent is meaningless which was observed in H 24 x LCR 1, LCR 9 x CLN 2123 A, LCR 1 x CLN 2123 A, LCR 1 x LCR 9 and H 24 x CLN 2123A. In the second season, CLN 2123A x LCR 9, LCR 9 x H 24 and LCR 9 x LE 415, LCR 1 x CLN 2123A and H 24 x CLN 2123A exhibited desired negative heterosis over the best parent indicating the consistency of latter two accessions in both the seasons. The genotype CLN 2123A proved as the best general combiner for TLCV disease resistance in both the seasons whereas H 24 in the first season only. All the above mentioned hybrids also exhibited desirable negative *sca.* In the season I, H 24 x LCR 1, LCR 9 x CLN 2123A and LCR 1 x CLN 2123 A had one parent with positive *gca,* indicating the role of additive x dominance interaction, while H 24 x CLN 2123A exhibited additive x additive interaction for resistance. Both of these interactions could be exploited very well in F_1 generation. In season II, 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 resistance reaction, whereas

additive x dominance interaction played a role in the other two hybrids (LCR 9 x LE 415 and LCR 1 x CLN 2123A). The cross H 24 x CLN 2123A could be best used for selecting recombinants as pure lines in later generations involving additive x additive interaction for TLCV resistance in both the seasons. Its reciprocal counter partner (CLN 2123A x H 24) exhibited unfavorable positive *sca* as well as heterosis indicating the role of either maternal effect or genic-cytoplasmic interaction. Inconsistency of many hybrids in terms of heterosis, *sca* and parents for *gca* over two seasons amply justifies the involvement of environment and modifiers. 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). Resistance to TLCV controlled by polygenes was reported by Berlinger, Daham and Shevach-Orkin (1983) and Kegler (1994) in tomato.

In respect of resistance to Tv, the parents LCR 9, LCR 1 and LCR 3 had proved to be the best combiners for Tv resistance in the season I, whereas in the season II, LCR 1 and H 24 proved to be best combiners. Among the 30 hybrids, only four exhibited favorable negative heterosis over the best parent *viz.,* LE 415 x LCR 3, LCR 9 x H 24, CLN 2123A x LCR 3 and LCR 3 x LCR 1 in the season I. All these hybrids also exhibited favourable negative *sca.* Of the four hybrids, additive x dominance interaction was involved in former three hybrids, whereas latter one exhibited additive x additive interaction for Tv resistance. In the season II, only six hybrids out of 30 showed the desired negative heterosis over better parent, while none of them were superior to best parent. The hybrid H 24 x CLN 2123A in the season I exhibited complementary gene action or the mutual cancellation of unfavourable epistatic genes present in the parents by the genes sponsored, whereas in the season II showed additive x dominance interaction indicating the role of environment and modifiers. Present work confirmed the findings of Kumar (1988) with respect to TSWV resistance, who reported the involvement of additive dominance and duplicate epistasis. Role of modifiers and environment on these virus diseases need to be studied further through temporal and spatial testing of these hybrids.

Regarding total PDI, four hybrids in season I and seven in season II registered favorable heterosis over the best parent. Out of them, LCR 9 x H 24, H 24 x LCR 9 and H 24 x LCR 1 proved consistency in both the seasons.

In conclusion, the parent H 24 was found to possess high favorable *gca* for five out of six characters under study *viz.,* yield, total and OD phenol, TLCV and Tv resistance followed by CLN 2123A for total and OD phenol and TLCV resistance. The hybrid H 24 x CLN 2123A exhibited high heterosis and favorable *sca* for four characters namely yield, total phenol, TLCV and Tv resistance.

Acknowledgement

The first author acknowledges the Council of Scientific & Industrial Research (CSIR) for granting fellowship during his doctoral studies and the present study forms a part of the same.

References

Aruna, S. and Veeraragavathatham, D. (1996). Correlation among yield and yield component traits in tomato *(Lycopersicon esculentum* Mill.). *South Indian Hort.,* **45(1&2):**7-9.

Table 3. Estimates of *sca* effects of F_1 for various traits under study

Cross	Fruit weight[a]	Yield per plant[a]	Total phenol	OD phenol[a]	TLCV incidence (season I)	TLCV incidence (season II)	Tv incidence (season I)	Tv incidence (season II)
LCR9x LCR1	-8.69**	-0.39**	11.33*	35.11**	1.91	10.48**	4.48	1.73
LCR9x LCR3	-3.97**	0.19**	10.06*	-3.62	1.24	3.48	4.68*	-0.14
LCR9xCLN2123A	9.34**	0.20**	0.61	17.39**	-9.22**	-3.57	2.41	5.55**
IXR9V II 24	-5.51**	0.15*	3.48	-5.16	-3.01	-4.87	-8.03**	-3.25
LCR9xLE415	0.36	0.30**	-12.90*	-15.38**	6.75*	-6.20*	-0.81	1.10
LCR1x LCR 9	11.13**	0.16*	-0.67	22.97**	-1.95	0.61	-0.01	-0.52
LCR 1 x LCR 3	-12.61**	-0.44**	-37.33**	-23.79**	8.69**	4.97	-1.72	1.39
LCR 1 x CLN2123A	-5.76**	0.23**	-26.87**	-52.15**	-2.30	-4.24	1.68	-5.12*
LCR 1 x H 24	2.26**	-0.10	8.00	46.34**	-7.90**	-8.11**	-1.16	2.57
LCR1 x LE415	1.25**	0.59**	26.02**	-9.78*	-5.76*	-2.24	-1.98	-2.45
LCR3xLCR9	4.50**	0.19*	3.38	0.10	6.39**	-4.48	-1.52	3.15
LCR3 xLCR1	8.97**	0.48**	18.40**	-2.85	-4.60	0.16	-0.89	6.51**
LCR3xCLN2123A	-1.92**	-0.47**	-6.53	0.62	1.67	0.35	-3.84	-0.97
LCR 3 x H 24	3.67**	-0.17*	28.77**	15.04**	9.22**	1.12	-2.87	-3.41
LCR3x LE415	-2.01**	-0.12	-18.66**	17.66**	2.00	-0.14	-0.90	6.96**
CLN2123AX LCR 9	4.50**	0.00	-10.88	-3.20	3.8S	-7.46*	-4.18	-8.47**
CLN2123AxLCRl	-7.25**	-0.03	-1.05	28.02**	5.41	0.99	-5.45*	-0.46
CLN 2123Ax LCR 3	7.70**	0.06	29.02**	42.45**	3.73	8.53*	-7.71**	1.11
CLN2123AxH24	-0.43	0.52**	3.82	-13.18**	3.60	4.48	10.02**	-1.30
CLN2123AxLE 415	0.61*	-0.30**	-8.78	-19.05**	5.41*	-0.45	2.90	2.69
H 24 x LCR 9	-2.75**	0.51**	5.25	-16.38**	-0.80	11.02**	2.40	-4.26
H 24 x LCR 1	-1.63**	0.17*	6.8S	32.45**	-3.88	0.00	-2 25	-0.91
H 24 x LCR 3	-0.50	0.42**	-1.18	16.40**	-2.72	1.28	0.12	-4.41
H24\CLN2123A	-6.47**	0.27**	33.08**	9.18	-6.81*	-6.37	-14.87**	-5.69*
H24xLE415	6.06**	0.53**	-27.71**	-32.55**	5.36*	3.72	0.64	4.84*
LE415xLCR9	-3.50**	-0.10*	-13.45*	16.27**	4.31	11.98**	-4.11	-6.60**
LE415xLCR 1	-0.75**	0.06	-8.08	7.75	0.00	-2.64	-0.53	1.50
LE415x LCR 3	0.60*	0.42**	19.42**	45.18**	0.22	2.50	-13.23**	-8.93**
LE415xCLN 2123A	-0.55	0.40**	19.90**	21.47**	6.40*	3.29	-10.17**	-2.98
LE415x H24	3.00**	-0.57**	-23.92**	-22.65**	8.43*	4.29	0.54	4.45
SE (sij)	0.244	0.006	4.753	3.936	2.63	2.826	2.222	1.985
SE (rij)	0.287	0.076	5.593	4.632	3.094	3.326	2.616	2.336
SCA (mean square)	109.77**	0.46**	1014.75**	2271.27**	117.74**	48.00**	56.10*	38.45**
RCA (mean square)	57.52**	0.20**	543.23**	1086.38**	43.20*	85.03**	66.60**	46.76**

* Significance at 1% level * Significance at 5% level [a]based on one year data

Baggett, J.R. (1957). Effects of genetic segregation in *Phaseolus vulgaris* on the symptoms induced by bean yellow mosaic virus. *Phytopath.,* **47:** 365-368.

Berlinger, M.J., Daham, R. and Shevach-Orkin, E. (1983). Breeding for resistance to whiteflies in tomatoes in relation to integrated pest control in greenhouses. *Pl. Breed. Abstr.,* **54(8):** 6229.

Bray, H.G. and Thrope, W.V. (1954). Analysis of phenolic compounds of interest in metabolism. *Meth. Biochem. Anal.,* **1:** 27-52.

Fuchs, W.H. (1971). Physiological and biochemical aspects of resistance to diseases. *Mutation breeding for disease resistance.* International Atomic Energy Agency, Vienna. 5-18pp.

Griffing, B. (1956). Concepts of general and specific combining ability in relation to diallel crossing system. *Aust. J. Biol. Sci.,* **9:** 463-493.

Jamwal, R.S., Rattan, R.S. and Saini, S.S. (1984). Hybrid vigour and combining ability in tomato. *South Indian Hort.,* **32:** 69-74.

Johnson, G. and Schaal, L.A. (1957). Cholorogenic and other orthodihydrophenols in scab resistant Russet Burbank and scab susceptible Triumph potato tubers of different maturities. *Phytopath.,* **47:** 253-257.

Kalloo, G. (1986). *Tomato.* Allied Publishers (P) Ltd., New Delhi. 471 pp.

Kegler, H. (1994). Incidence, properties and control of tomato yellow leaf curl virus: a review. *Arch. Phytopath. Plant Prot.,* **29(2):** 119-132.

Kumar, N. (1988). Studies on breeding for resistance to spotted wilt virus disease in tomato *(Lycopersicon esculentum* Mill.) Ph.D. Thesis, Tamil Nadu Agric. University, Coimbatore. 258 pp.

Kumar, S., Banerjee, M.K. and Partap, P.S. (1995). Studies on heterosis for various characters in tomato. *Haryana J. Hort. Sci.*, **24(1):** 54-60.

Mandal, A.R., Hazra, D., Som, M.G. and Maity, T.K. (1992). Exploration of heterosis in tomato *(Lycopersicon esculentum* Mill.) *Bangladesh J. Agric. Sci.,* **19(1):** 145-148.

Martin, M.W. (1970). Inheritance of resistant to curly top virus in tomato line C_5. *J. Amer. Soc. Hort. Sci.,* **95(4):** 424-427.

Narayana, Y.D. and Reddy, D.D.R. (1980). Chemical constituent of root knot nematode resistant varieties of tomato. *Nematol. Med.,* **8:** 51.

Pujari, C. V. and Kale, P. N. (1994). Heterosis studies in tomato. *J. Maharashtra Agric. Univ.,* **19(1):** 83-85.

Rai, N., Syamal, M.M., Joshi, A.K. and Rajput, C.B.S. (1997). Genetics of yield and yield components in tomato *(Lycopersicon esculentum* Mill.). *Indian J. Agric. Res.,* **31(1):** 46-50.

Rick, C.M. and Butler, L. (1956). Cytogenetics of the tomato. *Adv. Genet,* **8:** 267-382.

Singh, D.K. and Abidi, A.B. (1988). Biochemical transformation is leaves of tomato *(Lycopersicon esculentum* Mill.) varieties affected by leaf curl and mosaic disease. *Prog. Hort.,* **20(3&4):** 185-189.

Waswart, S.L. and Walker, J.C. (1961). Inheritance of resistance in cucumber to cucumber mosaic virus. *Phytopath.,* **51:** 423-428.

Williams, W. (1959). Heterosis and genetics of complex characters. *Nature,* **184:** 528-530.

Wynne, J.C., Emery, D.A. and Rice, P.W. (1970). Combining ability estimation in *Arachis hypogaea* II. Field performance of F_1 hybrids. *Crop Sci.,* **10:** 713-715.