



RESEARCH ARTICLE

Elucidating disease progression, combining ability and heterosis for response to leaf curl virus disease in chilli (*Capsicum annuum* L.)

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Received: 23 April 2025; Accepted: 09 July 2026; Available online: Version 1.0: 28 September 2026

Cite this article: Bidwan R, Premakumar, Rohit S, Channabasava, Shirisha KM, Jenia R, Firos TMB, Sindhu D, Mausham Y, Prashantha V, Mohan RA. Elucidating disease progression, combining ability and heterosis for response to leaf curl virus disease in chilli (*Capsicum annuum* L.). Plant Science Today (Early Access). <https://doi.org/10.14719/pst.15211>

Abstract

Development of chilli hybrids resistant to chilli leaf curl virus disease depends on the identification of desirable general combiners and heterotic hybrids. In the current study, 90 hybrids generated following a line × tester mating design using six cytoplasmic genetic male sterile lines and 15 fertility restorer testers were evaluated for response to chilli leaf curl virus disease (ChiLCVD) at 30, 60 and 100 days after sowing (DAS). The parents and hybrids were field screened for ChiLCVD under a randomized complete block design (RCBD) and an alpha lattice design respectively. Percent disease index (PDI) was computed and used to measure ChiLCVD severity. To assess the disease progression, PDI values were used to obtain the disease progress curve and estimate the area under disease progress curve (AUDPC). Wide variability was observed among parents and hybrids for response to ChiLCVD for each stage. The disease progress curve revealed a decline in ChiLCVD progression for S343 derived hybrids at 60 and 100 DAS. CMS10A and AV99 at 30 DAS and CMS7A and S343 at 60 and 100 DAS were found to exhibit significant general combining ability (GCA) effects in the desirable direction. No hybrid showed standard heterosis at 30 DAS and hybrids involving S343 were recorded with significant standard negative heterosis against ChiLCVD at 60 and 100 DAS. Based on our results, we suggest S343 can serve as a valuable source of dominant resistance for accelerating the development of chilli hybrids against ChiLCVD.

Keywords: area under disease progress curve; chilli; general combining ability; heterosis; leaf curl virus; specific combining ability

Introduction

Chilli is an economically significant spice and vegetable crop, contributing to global food systems through its diverse applications in culinary, medicinal and industrial sectors. Among the *Capsicum* species, *Capsicum annuum* L. is the most widely cultivated globally, while others include *Capsicum baccatum* L., *Capsicum chinense* Jacq., *Capsicum frutescens* L. and *Capsicum pubescens* Ruiz & Pav. (1). India is the world's leading producer of dry red chilli, driven by both domestic demand and export markets (2). Among various biotic stresses, Chilli leaf curl virus disease (ChiLCVD) is one of the most devastating, causing substantial yield losses. Four begomoviruses, including Chilli leaf curl virus (ChiLCV), Tomato leaf curl New Delhi virus (ToLCNDV), Tomato leaf curl Joydebpur virus (ToLCJV) and Chilli leaf curl Palampur virus (ChiLCPV) have been identified as the causal agents of ChiLCVD in chilli (3–5). The disease is characterised by symptoms such as leaf curling, puckering, vein thickening and swelling. As infection advances, apical leaves cluster, leading to plant stunting. In severe cases, infected plants fail to produce flowers and fruits and ChiLCVD incidence can reach up to 100 %, posing a serious threat to chilli cultivation (6).

Chilli leaf curl virus disease has emerged as a major concern in chilli growing regions globally over the past two decades (7, 8). Host plant resistance offers an economical and eco-friendly solution to this problem. The availability of cytoplasmic genetic male sterility (CGMS) (9, 10) and the capacity to produce a large number of seeds per hand pollination have facilitated the development and cultivation of chilli hybrids. Thus, breeding ChiLCVD resistant hybrids is a practical and feasible approach to mitigate biotic stress in chilli. Identification of suitable parents, the development of heterotic hybrids that outperform commercial checks and the availability of a pollen control mechanism are crucial factors determining the success rate of hybrid breeding programs. Additionally, the availability of reliable resistance sources governed by dominant alleles is crucial when the objective is to breed resistant hybrids. While most reported resistance sources against ChiLCVD are recessive, a few dominant sources such as S343, IHR4517 and IHR4615 have been identified in chilli (11–14).

In this study, we evaluated 90 chilli hybrids developed using CGMS system for their response to ChiLCVD at 30, 60 and 100 days after sowing (DAS). The disease progress curve was studied to understand the ChiLCVD progression during the seedling (30 DAS),

flowering (60 DAS) and fruiting (100 DAS) stages respectively. Combining ability and heterosis analysis were conducted to identify promising general combiners and heterotic hybrids with ChilCVD resistance.

Materials and Methods

Experimental material

The experimental material comprised 90 hybrids developed during kharif 2020 through a line × tester mating design, involving 6 CGMS lines and 15 fertility restorer testers, along with 5 ChilCVD susceptible checks. A total of 21 diverse parental lines were used, including 18 exotic sources (five CGMS lines and 13 advanced breeding lines (ABLs)) and 3 indigenous sources (one CGMS line and two working collections (WCs)) of chilli germplasm (Table 1).

Design of experiment

Screening against ChilCVD was conducted during summer 2021 at the experimental plots of the Main Agricultural Research Station (MARS), Hebbal, Bengaluru, under natural epiphytotic conditions. This location has previously been reported as suitable for ChilCVD screening due to environmental conditions that favour disease development during the summer season (15). A spacing of 0.60 × 0.45 m was followed for transplanting the hybrids and checks in an alpha lattice design and parents in a randomized complete block design (RCBD) with 2 replications. Infector rows were maintained at regular intervals using susceptible checks to ensure uniform disease pressure. The recommended package of practices was followed for crop management, except for the application of insecticidal sprays.

Sampling and field data recording

Ten plants were randomly selected per replication and screening

was carried out based on a 0–5 scale at 30 DAS, 60 DAS and 100 DAS, corresponding to the seedling, flowering and fruiting stages of chilli development respectively, to assess disease progression across critical growth stages (16, 17). The ChilCVD responses were categorised into highly resistant (HR), resistant (R), moderately resistant (MR), moderately susceptible (MS) and highly susceptible (HS) based on percent disease index (PDI) estimates calculated using equation 1 (13).

$$PDI = \frac{\text{Sum of individual disease ratings}}{\text{Total number of screened plants} \times \text{Highest score in scale}} \times 100 \quad (\text{Eq. 1})$$

Assessment of ChilCVD progression

The PDI estimates were used to compute the area under the disease progress curve (AUDPC) using equation 2 for the seedling (30 DAS), flowering (60 DAS) and fruiting (100 DAS) stages following the trapezoidal method (18). To assess differences in disease progression among stages, AUDPC values were subjected single-factor ANOVA and the results were visualised using error bar graphs. Disease progress curves and AUDPC error bar plots were generated using the ggplot2 package in R software (version 4.4.2).

$$AUDPC = \sum_{i=1}^{n-1} \frac{(y_{i+1} + y_i) \times (t_{i+1} - t_i)}{2} \quad (\text{Eq. 2})$$

Where,

y_i = Mean PDI estimate at i^{th} observation

t_i = Days after sowing at i^{th} observation

n = Total number of observations

Table 1. Details of parental genotypes and checks used in the study

Genotype	Kind of genetic material	World vegetable centre accession code	Source
CMS6A	CGMS line	CCA7242	WVC, Taiwan*
CMS7A	CGMS line	CCA7243	WVC, Taiwan
CMS8A	CGMS line	CCA7244	WVC, Taiwan
CMS9A	CGMS line	CCA7245	WVC, Taiwan
CMS10A	CGMS line	CCA7246	WVC, Taiwan
CMSUAA	CGMS line	-	UAS, Bangalore, India†
AV2	ABL	VI037438	WVC, Taiwan
AV16	ABL	VI059328	WVC, Taiwan
AV31	ABL	AVPP1808	WVC, Taiwan
AV43	ABL	AVPP1809	WVC, Taiwan
AV44	ABL	AVPP1804	WVC, Taiwan
AV46	ABL	AVPP1803	WVC, Taiwan
AV47	ABL	AVPP1806	WVC, Taiwan
AV48	ABL	AVPP1807	WVC, Taiwan
AV49	ABL	AVPP1805	WVC, Taiwan
AV53	ABL	AVPP1814	WVC, Taiwan
AV59	ABL	AVPP1810	WVC, Taiwan
AV99	ABL	AVPP9824	WVC, Taiwan
AV100	ABL	AVPP9905	WVC, Taiwan
PDL1	WC	-	UAS, Bangalore, India
S343	WC	-	Ludhiana, Punjab, India
Arka Haritha	Released hybrid (RH)	-	IIHR, Bangalore, India#
Arka Khyathi	RH	-	IIHR, Bangalore, India
Arka Meghana	RH	-	IIHR, Bangalore, India
Arka Swetha	RH	-	IIHR, Bangalore, India
KBCH 1	RH	-	UAS, Bangalore, India

*WVC: World Vegetable Centre; †UAS: University of Agricultural Sciences; #IIHR: Indian Institute of Horticultural Research; CGMS: cytoplasmic genetic male sterility; ABL: advanced breeding line; WC: wild collection.

Combining ability and heterosis analysis

The PDI estimates were subjected to combining ability analysis to obtain the components of genetic variance (19, 20) followed by heterosis analysis. Arka Khyathi was used as a check to estimate the standard heterosis. This analysis was implemented in Indostat software version 9.1. The relative importance of additive and dominance gene actions was inferred by calculating Baker's predictability ratio from the general combining ability (GCA) and specific combining ability (SCA) variance components (21).

Results

Test of significance

Analysis of variance (ANOVA) for PDI estimates revealed significant differences among crosses for mean sum of squares at 30, 60 and 100 DAS, indicating adequate genetic variability for ChiLCVD response and suitability for further study (Table 2). Significant mean square differences due to line effect, tester effect and line × tester interaction were observed at all three stages, suggesting the involvement of both additive and non-additive genetic components. The estimates of genetic variance components revealed that SCA variance exceeded GCA variance at all stages of disease assessment (Supplementary Table 1). The predictability ratio was low at 30 DAS (0.07) and increased to 0.40 and 0.43 at 60 and 100 DAS respectively. Dominance variance was consistently higher than additive variance and the degree of dominance exceeded unity across all stages, indicating the predominance of non-additive gene action for ChiLCVD response. However, the increasing predictability ratio at later growth stages suggested an increased contribution of additive genetic effects during flowering and fruiting.

Per se response to chilli leaf curl virus disease

High variability was recorded among parents and hybrids in response to ChiLCVD at 30, 60 and 100 DAS. At 30 DAS, six parents exhibited moderately resistant (MR) response, including PDL1 (24.11 %) followed by CMS6A (33.83 %), while the remaining parents showed moderately susceptible (MS) or highly susceptible (HS) response. At 60 and 100 DAS, except for S343 (30.02 % and 29.77 % respectively) followed by PDL1 (31.25 % and 31.75 % respectively), which showed MR, the other parents exhibited MS or HS response (Supplementary Table 2). Among the 90 hybrids, five (CMS7A × AV99, CMS7A × AV2, CMS7A × PDL1, CMS10A × AV99 and CMS10A × PDL1 with a PDI estimate of 16.67 %) and 22 hybrids were identified with R and MR responses at 30 DAS respectively. At 60 and 100 DAS, S343 based hybrids exhibited an MR reaction, while the remaining were MS or HS to ChiLCVD. Among these, CMS6A × S343 (21.67 % and 20.17 %) followed by CMS10A × S343 (23.61 % and 22.61 % respectively) had lower PDI estimates. Notably, CMS10A × S343 was the only hybrid with an MR response at all three stages (Supplementary Table 3).

Resistance to ChiLCVD during the reproductive stage is particularly beneficial for minimising yield loss in chilli and the genotypes AV2, AV99, PDL1 and S343 recorded lower PDI estimates at 60 and 100 DAS (Supplementary Table 2). A comparison of the distribution of PDI estimates among AV2, AV99, PDL1 and S343 based hybrids was visualised through box-and-whisker plots (Fig. 1). The S343-based hybrids had higher PDI estimates at 30 DAS and lower estimates at 60 and 100 DAS, whereas AV2, AV99 and PDL1-based hybrids showed the opposite response. This indicates S343 as a potential source to breed ChiLCVD resistant hybrids at flowering and fruiting stage (Fig. 1).

Table 2. Line × tester ANOVA for percent disease index (PDI) estimates at 30, 60 and 100 days after sowing (DAS) in response to ChiLCVD

Source of variation	df	Mean sum of squares		
		30 DAS	60 DAS	100 DAS
Replications	1	28.30	81.97	192.81
Crosses	89	1014.05***	670.58***	699.23***
Line effect	5	3983.96***	771.91***	760.90***
Tester effect	14	2566.38***	3394.41***	3584.64***
Line × tester effect	70	491.45***	118.58*	117.75*
Error	89	122.58	70.37	72.83

*Significance at $p = 0.05$; **Significance at $p = 0.01$; ***Significance at $p = 0.001$.

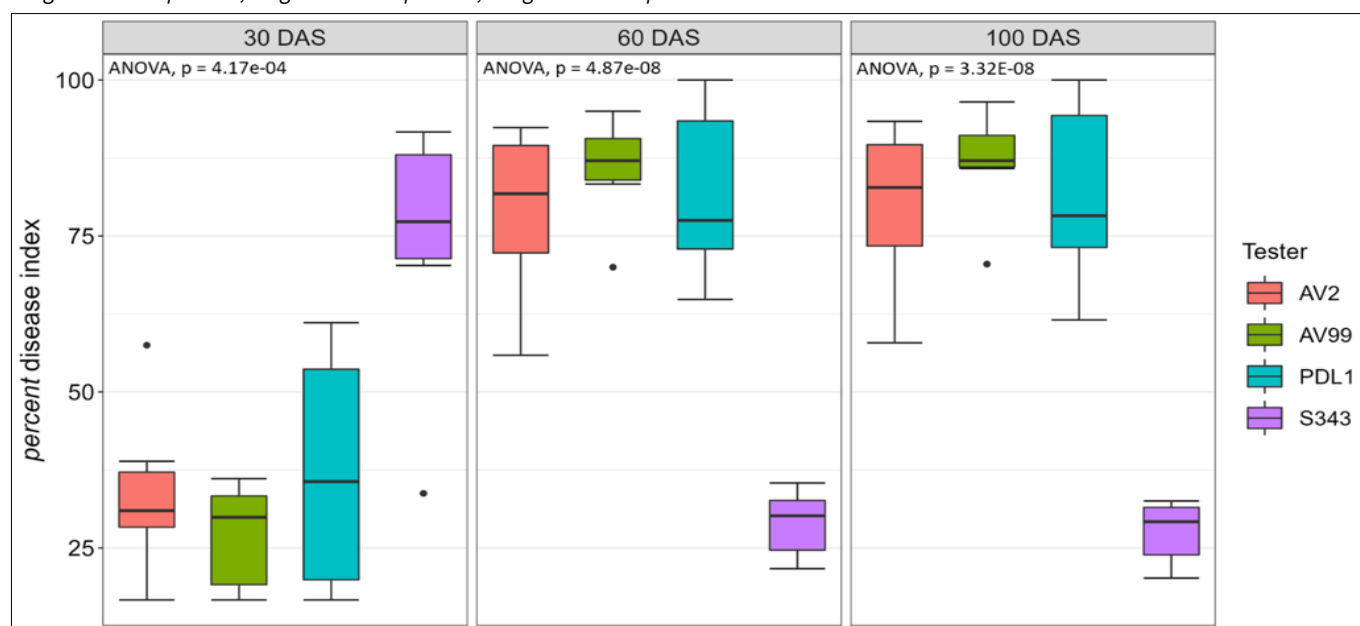


Fig. 1. Box-and-whisker plots showing comparative PDI estimates at 30, 60 and 100 DAS for responses to ChiLCVD infection among chilli hybrids from common tester parents (AV2, AV99, PDL1 and S343)

Progression of chilli leaf curl virus disease (ChiLCVD) under natural epiphytotic condition among hybrids

The Mean of PDI estimates of hybrids with AV2, AV99, PDL1 and S343 as common parents were used for disease progress curve (DPC) analysis and AUDPC estimation. Hybrids based on AV2, AV99 and PDL1 exhibited a typical sigmoid DPC, suggesting a logistic progression of ChiLCVD under epiphytotic conditions in the field. Conversely, S343 derived hybrids showed a non-sigmoid DPC, indicating an initial increase followed by a decline in ChiLCVD progression over time (Fig. 2). The AUDPC was lowest for S343 (3769.70) followed by AV2 derived hybrids (5370.10) (Fig. 2). Independent comparisons of AUDPC for A_1 , A_2 and A_3 intervals

showed significant differences in A_1 and A_3 between S343 derived hybrids and those based on AV99, AV2 and PDL1 (Fig. 3). S343 based hybrids showed significantly higher disease progression in A_1 and lower in A_3 , suggesting an age-dependent decline in disease severity. Among S343 based hybrids, CMS10A $\times$ S343 (2291.05) followed by CMS9A $\times$ S343 (3769.50) recorded lower AUDPC estimates (Supplementary Fig. 1).

Combining ability analysis

Across all three stages (30, 60 and 100 DAS), eight parents exhibited significant negative GCA effects indicating the contribution of favourable alleles to hybrids for resistance against ChiLCVD. Among lines and testers, CMS10A (-15.59) and AV99 (-29.52) at 30 DAS and

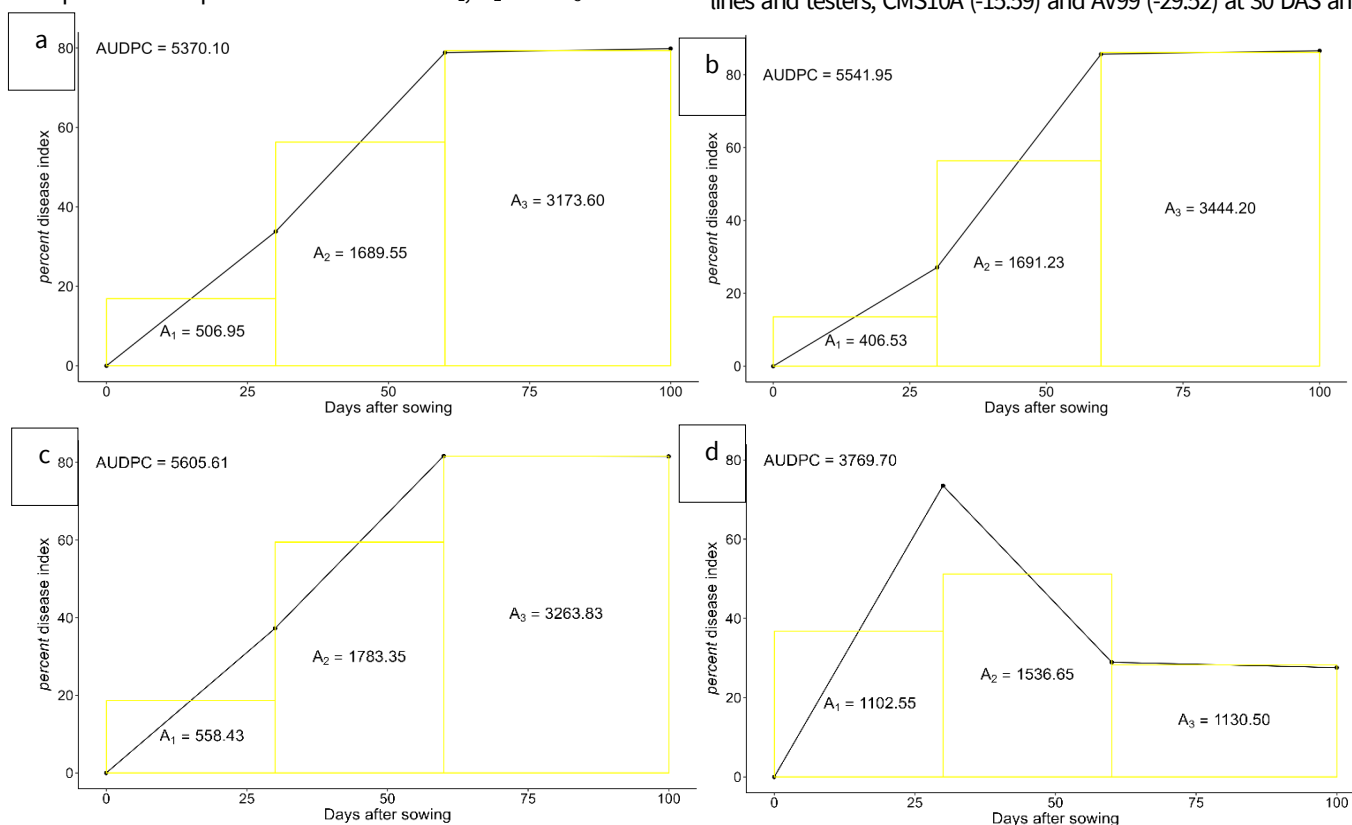


Fig. 2. Disease progress curves and area under disease progress curve (AUDPC) estimates showing changes in chilli leaf curl virus disease (ChiLCVD) severity at 30, 60 and 100 days after sowing (DAS) among chilli hybrids from common tester parents: (a) AV2, (b) AV99, (c) PDL1, (d) S343.

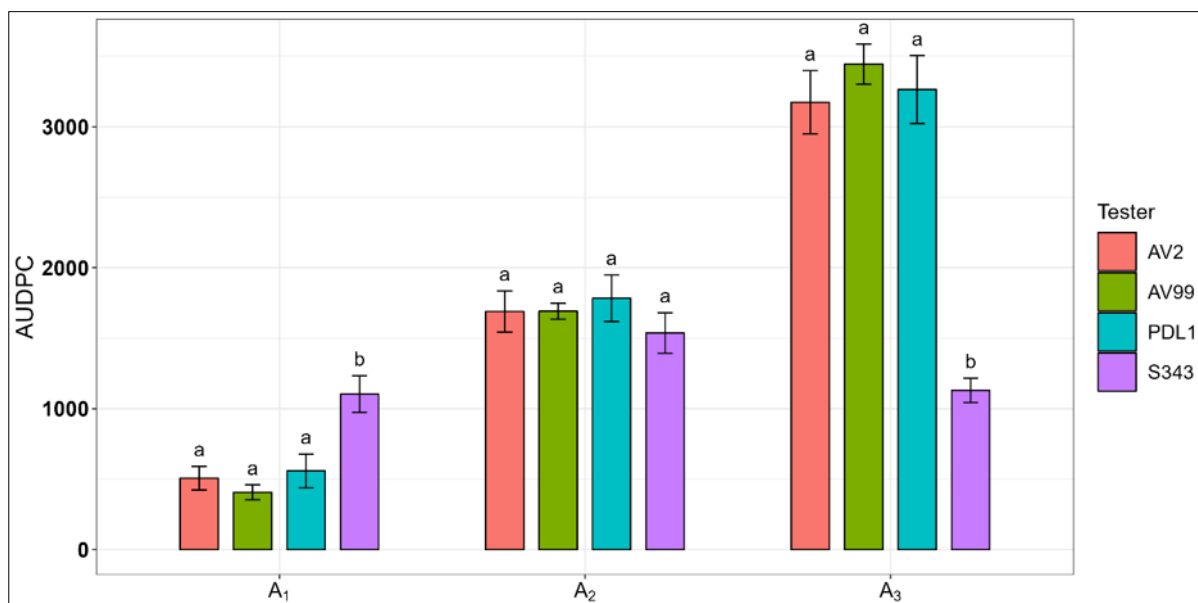


Fig. 3. Area under disease progress curve (AUDPC) from 0–30 days after sowing (DAS) (A_1), 30–60 DAS (A_2) and 60–100 DAS (A_3) for chilli hybrids from common tester parents: AV2, AV99, PDL1 and S343.

CMS7A (-5.87 and -5.84) and S343 (-57.30 and -59.16) at 60 and 100 DAS registered the lowest significant GCA effects (Table 3). Notably, CMSUAA and AV2 had consistent significant negative GCA effects across all three stages.

More crosses showed significant SCA effects at 30 DAS (24) than at 60 (7) and 100 DAS (7), possibly due to stronger dominance gene action at 30 DAS (Supplementary Table 1). Significant negative SCA effects were observed in 18 hybrids across the three stages. At 30 DAS, 12 hybrids, with CMS9A × AV46 (-29.66) having the lowest and at 60 DAS and 100 DAS, five and six hybrids respectively, exhibited significant negative SCA effects, including CMSUAA × AV44 (-17.53 and -16.62 respectively), which showed the lowest values (Supplementary Table 4).

Heterosis analysis

Among the 90 hybrids, five hybrids exhibited significant negative better parent heterosis across the three stages. CMS7A × AV2 (-64.53 %) followed by CMS10A × AV99 (-61.83 %) and CMS7A × AV99 (-61.83 %) at 30 DAS and CMSUAA × AV44 (-38.06 % and -36.06 %) and CMSUAA × AV59 (-20.00 % and -18.24 %) at 60 and 100 DAS recorded the lowest heterobeltiosis (Supplementary Table 5). Arka Khyathi had the lowest PDI among the five checks and was used for standard heterosis estimation (Supplementary Table 3). No hybrid showed significant negative standard heterosis over Arka Khyathi at 30 DAS (Supplementary Table 5). However, 20 hybrids at 60 DAS and 19

hybrids at 100 DAS exhibited significant standard heterosis in desirable direction, particularly S343 based hybrids, which exhibited higher negative heterosis along with MR response to ChiLCVD including CMS6A × S343 (-77.55 % and -79.32 % respectively) followed by CMS10A × S343 (-75.54 % and -76.82 % respectively) (Fig. 4; Table 4).

Discussion

In this study, both additive and non-additive genetic effects in response to ChiLCVD were observed in chilli; however, the predominance of non-additive gene action was evident from the low predictability ratio and high degree of dominance, particularly at 30 DAS. Consequently, exploitation of heterosis through hybrid breeding may be an effective strategy for improving resistance against ChiLCVD. Previous studies have reported the involvement of additive (22) and non-additive (23) gene action in conferring resistance to ChiLCVD as well. Stage-dependent resistance expression is a recognised phenomenon in plant-pathogen interactions, where different genetic mechanisms may contribute to resistance at different developmental stages. The distinction between resistance during the seedling and adult stages, resulting from different gene actions, is well-documented in wheat rust (24). Genotypes such as PDL1 and S343, which exhibit resistance at 60 and 100 DAS, serve as valuable sources of resistance, as severe

Table 3. Estimates of general combining ability (GCA) effects of parental lines and testers for chilli leaf curl virus disease (ChiLCVD) responses at 30, 60 and 100 days after sowing (DAS)

Parent	GCA effect		
	30 DAS	60 DAS	100 DAS
Lines			
CMS6A	2.85	0.07	-0.3
CMS7A	0.90	-5.87***	-5.84***
CMS8A	11.42***	4.40**	4.02*
CMS9A	12.07***	0.63	1.32
CMS10A	-15.59***	6.47***	6.50***
CMSUAA	-11.65***	-5.71***	-5.70***
Testers			
AV31	7.04*	6.14*	6.72**
AV43	8.62**	3.34	4.00
AV44	7.16*	-1.06	-0.48
AV46	6.59*	9.22***	8.96***
AV47	12.66***	10.76***	11.00***
AV48	6.75*	9.23***	9.31***
AV49	-4.29	9.98***	9.56***
AV53	16.12***	8.55***	9.04***
AV59	3.87	2.33	2.99
AV99	-29.52***	-0.59	-0.18
AV100	4.49	8.88***	8.38**
AV2	-22.83***	-7.40***	-6.91**
AV16	-14.18***	2.49	1.98
PDL1	-19.40***	-4.58	-5.22*
S343	16.87***	-57.30***	-59.16**

*Significance at $p = 0.05$; **Significance at $p = 0.01$; ***Significance at $p = 0.001$; GCA: general combining ability; DAS: days after sowing.



Fig. 4. Responses to chilli leaf curl virus disease (ChiLCVD) among CMS6A × S343, CMS10A × S343 and Arka Khyathi.

Table 4. Hybrids exhibiting significant negative standard heterosis and their response to chilli leaf curl virus disease (ChiLCVD) at 60 and 100 days after sowing (DAS)

Hybrid	Standard heterosis		Response to ChiLCVD
	60 DAS	100 DAS	
CMS6A×AV99	-27.48**	-27.71**	HS
CMS6A×AV16	-18.91*	-22.82'	HS
CMS6A×PDL1	-22.30*	-23.61**	HS
CMS6A×S343	-77.55**	-79.32**	MR
CMST7A×AV44	-23.38**	-23.14**	HS
CMST7A×AV59	-19.84*	-19.63'	HS
CMST7A×AV100	-17.27	-18.63'	HS
CMST7A×AV2	-27.48**	-27.71**	HS
CMST7A×AV16	-26.49**	-27.75**	HS
CMST7A×PDL1	-25.18**	-25.43**	HS
CMST7A×S343	-66.31**	-66.65**	MR
CMS8A×S343	-63.31**	-67.40**	MR
CMS9A×AV43	-18.20*	-16.48	HS
CMS9A×AV2	-17.99*	-15.75	HS
CMS9A×S343	-71.22**	-71.52**	MR
CMS10A×S343	-75.54**	-76.82**	MR
CMSUAA×AV44	-35.83**	-34.43**	MS
CMSUAA×AV59	-20.86*	-19.11*	HS
CMSUAA×AV2	-42.10**	-40.64**	MS
CMSUAA×PDL1	-32.84**	-36.90**	HS, MS [#]
CMSUAA×S343	-66.19**	-68.58**	MR

*Significance at $p = 0.05$; **Significance at $p = 0.01$; ***Significance at $p = 0.001$; #HS at 60 DAS and MS at 100 DAS; MR: moderately resistant; MS: moderately susceptible; HS: highly susceptible; DAS: days after sowing; ChiLCVD: chilli leaf curl virus disease.

curling of apical plant parts during the reproductive stage can significantly reduce yield. Several researchers have identified monogenic recessive sources of resistance to ChiLCVD (25–29). However, utilisation of resistance for hybrid development requires dominant inheritance.

In our study, hybrids derived from S343 displayed resistance to ChiLCVD, consistent with findings, where S343 was identified carrying the gene for resistance under dominant gene control (12). Area under disease progress curve was used to characterise temporal disease dynamics through repeated field assessments (30). The sigmoid shaped disease progress curve aligns with epidemiological principles, where logistic disease progression occurs due to resource limitations for the pathogen. Although disease symptoms were higher during the seedling stage, the disease progression was substantially reduced during the flowering and fruiting stages in S343-derived hybrids, suggesting the involvement of age-related resistance mechanisms. Age-related resistance has been widely reported in several plant-pathogen systems, where resistance expression increases as plants mature due to the integration of developmental and defence signalling pathways (31). In virus caused diseases, higher resistance at later developmental stages has been associated with stronger basal defence responses and reduced susceptibility of mature tissues to infection (32). A Previous study has mapped the dominant gene governing ChiLCVD resistance in S343 to chromosome six (33). Moreover, S343 has been reported as moderately resistant to *Meloidogyne incognita* and holds potential for multiple disease resistance (34). It has also been recognised for desirable traits such as red chilli powder yield and colouring matter (35). Although S343 exhibited consistently lower disease severity and AUDPC values under the conditions of the present study, the stability of resistance across diverse agro-climatic environments and its effectiveness against different begomovirus species or strains require further validation. Multi-location and multi-season evaluations, coupled with characterisation of the prevailing virus populations, will be necessary to determine the durability and broader applicability of

S343-derived resistance in chilli breeding programmes.

General combining ability reflects the ability of a parent to transmit favourable alleles to its progenies and is generally indicative of the contribution of additive genetic effects. Consequently, parents with desirable GCA effects for reduced disease severity may be useful sources for incorporating resistance into breeding populations through selection. Conversely, specific combining ability represents the performance of a particular cross associated with non-additive genetic action. Among parents, CMST7A and S343 were identified as desirable general combiners and can be used in future breeding programs for ChiLCVD resistance breeding in chilli. The cross CMS10A × S343 was particularly notable for showing resistance at 30, 60 and 100 DAS. Since significant GCA effects of parents indicate that alleles are transmitted to their progeny, GCA effects of CMS10A at 30 DAS and S343 at 60 and 100 DAS may explain the consistent resistance in CMS10A × S343 across all three stages. None of the S343 based crosses showed significant negative heterobeltiosis, due to high level of resistance in S343 parent. The significant negative standard heterosis observed in several hybrids indicates reduced disease severity relative to the commercial check. However, because disease reactions are assigned based on absolute PDI values, some of these hybrids remained within susceptible categories despite exhibiting standard heterosis. In contrast, S343-derived hybrids expressed both significant heterosis in desirable direction and moderately resistant reactions (Table 4). Previous studies have reported the identification of desirable general combiners and the presence of heterosis for resistance to ChiLCVD in chilli as well (22, 23, 36). Recent efforts have utilised S343 to introgress ChiLCVD resistance into heat tolerant bell pepper lines through combined phenotypic selection and marker-assisted backcrossing (37).

Conclusion

The present study demonstrated substantial variability in chilli leaf curl virus disease (ChiLCVD) response among 90 experimental

hybrids developed using chilli cytoplasmic genetic male sterility (CGMS) lines and restorer testers of exotic and indigenous origin. Hybrids involving the tester S343 exhibited reduced disease severity during the flowering and fruiting stages, as reflected by the disease progression dynamics. Among the parental genotypes, CMS7A and S343 were identified as desirable general combiners, while S343-based hybrids exhibited standard heterosis and enhanced resistance to ChilCVD during flowering and fruiting stages. These findings highlight the importance of S343 as a resistance donor and provide useful parental combinations for the development of ChilCVD-resistant chilli hybrids. Future research should focus on molecular validation of resistance alleles in promising hybrids, elucidation of the genetic mechanisms underlying age-related disease responses and multi-location evaluation of superior hybrids to assess the stability of resistance under diverse environments.

Acknowledgements

The authors are thankful to ICAR-Indian Agricultural Research Institute, New Delhi, India and University of Agricultural Sciences, Bangalore, Karnataka, India, for providing the necessary research facilities and support for conducting the present investigation.

Authors' contributions

BR conducted the experiments, performed field screening and drafted the manuscript. P and RS carried out the data analysis. AMR and C supervised and coordinated the experiments and contributed to the development of experimental material. KMS and PV were involved in the experimental design and field data collection. JR and FTMB contributed to graphical representation of data and preparation of tables. SD and MY assisted in manuscript reviewing and editing. All authors have read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used ChatGPT in order to improve the clarity, grammar and overall quality of the manuscript. After using this tool/service, the author(s) reviewed and edited the content as needed and take full responsibility for the content of the publication.

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