P.ISSN: 0971-8184 II E ISSN: 0976-1926
DOI: https://doi.org/10.56093/ijpgr.v39i3.182380

Indian Journal of Plant Genetic Resources

RESEARCH ARTICLE

Molecular Tracking and Exploration of Resistance Sources to Chilli Leaf Curl Virus Disease in Chilli

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Abstract

Chilli cultivation is significantly affected by leaf curl disease caused by the Chilli leaf curl virus (ChiLCV), transmitted by whiteflies. This study aimed to identify resistant genotypes to ChiLCV. Forty-one germplasms from the Horticulture Research and Extension Centre, Devihosur, and seven hybrids from the Indian Institute of Horticultural Research, Bengaluru, along with the susceptible check Byadagi Kaddi, were screened under field and glasshouse conditions during 2023-24. The genotypes Arka Gagan (3.57%), Arka Tejasvi (4.17%), and DCA-295 (9.09%) showed resistance, while Arka Saanvi (29.50%) was moderately resistant. The remaining 44 genotypes were susceptible to ChiLCV. Genotypic analysis using ChiLCV coat protein-specific primers confirmed that Arka Gagan, Arka Tejasvi, and DCA-295 were resistant, as they did not show ChiLCV amplification. These genotypes offer promising candidates for developing resistant varieties and hybrids to combat the disease.

Keywords: Chilli, Chilli leaf curl virus, Genotypic, Phenotypic, Resistance

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Received: 15/01/2025

Revised: 07/07/2026

Accepted: 05/08/2026

How to cite this article: Prashanth SV, DS Aswathanarayana, YS Amaresh, MR Govindappa, V Venkataravanappa, BV Temburne and SA Prabhudeva (2026) Molecular Tracking of Chilli Leaf Curl Virus Resistance. *Indian J. Plant Genet. Resour.* 39(3): 97-104.

DOI: https://doi.org/10.56093/ijpgr.v39i3.182380

Introduction

Chilli (*Capsicum annuum* L.) is an important spice and vegetable crop belonging to the family Solanaceae, grown both in tropical and subtropical regions of the world (Kumar and Rai 2005). It has high export potential and huge demand both domestically and internationally. However, its production potential is suppressed by many pests and diseases. Chilli crops also suffer from a large number of fungal, bacterial, viral and nematode diseases. Several viral diseases *viz*., chilli mosaic virus, chilli veinal mottle virus, cucumber mosaic virus, chilli leaf curl virus, pepper vein banding virus. Among the different fungal, bacterial and viral diseases, the leaf curl disease caused by Chilli leaf curl virus (ChiLCV), which contains a ssDNA *Begomovirus* transmitted by *Bemisia tabaci* (Brown *et al.,* 1995), is one of the main constraints hindering its cultivation worldwide.

Chilli leaf curl virus was known to produce different symptoms in plants, as reported by Oraon and Tarafdar (2018). Thakur *et al.,* (2018) reported mild to severe leaf curling, puckering, blistering of leaves, crinkling, narrowing and swollen veins, stunted growth and a bushy appearance in infected plants. Plants that are severely infected may not produce any fruit, while those that are less infected may produce fruits that are significantly smaller in size etc.; these symptoms result in heavy yield losses. In addition to that, thrips, mites and whiteflies have been reported to produce leaf curl

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complex and cause losses in chilli. Earlier some workers conducted studies on viral diseases in chilli; considering the destructive nature, the extent of yield losses due to leaf curl complex ranged from 25 to 80 per cent (Ilyas and Khan, 1996). The chilli leaf curl virus (ChiLCV) disease on chilli was first reported in Pakistan by Shih *et al.,* (2003) and in India by Senanayake *et al.,* (2007).

In view of the economic importance of the disease, the primary objective of the current study was to evaluate and identify sources of resistance to the chilli leaf curl virus by screening a diverse array of chilli genotypes. The evaluation was carried out under natural epiphytotic conditions; the promising genotypes found resistant in field conditions were further screened under controlled environments utilizing viruliferous whiteflies as vectors for the virus. Given the increasing challenges posed by the recurrent development of resistance to pesticides among whitefly populations, exploring alternative management strategies has become critical. Utilizing host plant resistance stands out as a sustainable and environmentally friendly approach for management of viral diseases, particularly those affecting horticultural crops like chillies. Wild relatives and accessions of cultivated chilli species are known to harbour valuable genetic traits that confer disease resistance, making them important resources for breeding programs. By systematically examining the response of various genotypes to virus infection, this study aims to pinpoint specific genetic traits associated with resistance. The findings are expected to contribute significantly to the development of resilient chilli varieties, enhancing productivity while minimizing reliance on chemical interventions. Ultimately, this research not only addresses immediate agricultural challenges but also aligns with broader goals of sustainable agriculture and food security in the face of evolving pest and disease pressures.

Materials and Methods

Screening of genotypes

To find out a suitable resistant source to the leaf curl virus, screening was carried out with chilli genotypes. The germplasms collected from Horticulture Research and Extension Centre (HREC), Devihosur, Haveri and the Indian Institute of Horticultural Research (IIHR), Bengaluru were evaluated against the chilli leaf curl virus under natural epiphytotic field conditions at PG research block during 2023-24, the control measures were taken as per the package of practices of UAS, Raichur to other diseases except for chilli leaf curl disease. Further, per cent disease incidence in each entry was calculated based on the formula given below. Based on the following (0 to 4) disease scale, the ratings were assigned to each entry (Ashwathappa, 2021) for categorization of genotypes. For artificial screening, the genotypes showing resistant reactions in field conditions were further confirmed by individual cage inoculation. Each genotype of resistant lines was raised in por-trays (20 seedlings of test plants along with 20 seedlings of susceptible check, Byadgi Kaddi) and challenge inoculated by a viruliferous whitefly population maintained on susceptible symptomatic chilli plants in the wooden cage covered with nylon net. About 300-500 adults of *B. tabaci* were collected into acquisition bottles, and ChiLCV-infected branches were inserted and allowed to feed for 24 hours as the acquisition access period (AAP). After virus acquisition of the virus, about 10 viruliferous whiteflies of about 10 were collected and inoculated onto each seedling using an aspirator into plastic cages, with an inoculation feeding period of 24 hours. After inoculation, the plants were sprayed with 0.5 ml imidacloprid 17.8 SL to kill whiteflies and allowed for symptom expression in the insect-proof glasshouse. After one week of inoculation, plants expressing symptoms and the number of plants infected in resistant and susceptible lines were recorded at weekly intervals; per cent disease incidence in each entry was calculated based on the following (0 to 4) disease scale; the ratings were assigned to each entry (Ashwathappa, 2021) (Table 1).

Disease incidence was calculated by using the following formula:

$$\text{Disease incidence (\%)} = \frac{\text{Number of infected plants}}{\text{Total number of plants observed}} \times 100$$

Table 1: Disease scale adopted for characterization of genotypes based on ChiLCV incidence

Scale	Host reaction	Description
0	Immune	No symptoms and PCR negative
1	Highly resistant	No symptoms and PCR positive
2	Resistant	1-10 %, curling, very mild clearing, and yellowing of veins
3	Moderately resistant	11-30 %. curling, puckering and yellowing of leaves
4	Susceptible	>30 %, leaf curling, stunted plants, swelling of veins and deformed small leaves

Assessment of area under disease progress curve for ChiLCV in the chilli genotypes

Also, the area under the disease progress curve (AUDPC) was calculated using the formula as suggested by Wilcoxson *et al.,* (1975).

$$AUDPC = \sum_{i=1}^{K} \frac{1}{2} (S_i + S_{i-1}) X\, d$$

Where S_i = Disease incidence at i^{th} day of evaluation, K = Number of successive evaluations of the disease, d = Interval between i and i-1 evaluations of the disease.

Apparent rate of infection (r)

Based on per cent disease index (PDI) values, apparent rate of infection (r) was calculated using the formula as given by (Vander Plank, 2012). These values help to understand the rate of disease progression at given intervals of time.

$r=2.3/t_2\text{-}t_1\{\log(X_2(1\text{-}X_1)/X_1(1\text{-}X_2))\}$

Where *r* is the apparent infection rate in the non-logarithmic phase, X_1 is the disease index at time t_1, X_2 is the disease index at the subsequent week time t_2.

Molecular confirmation of virus in chilli plants through PCR analysis

The leaf samples were collected randomly from resistant genotypes, and DNA was extracted using the CTAB (Cetyl Trimethyl Ammonium Bromide) method (Maruthi *et al.,* 2002). The PCR amplification was carried out in a thermal cycler with coat protein specific primers ChiLCV CPF: CATATGCTCCAGACACTCTG and ChiLCV CPR: CTAACCTTCCGAATCTGGACG with the following conditions; initial denaturation at 94°C for 10 min followed by 35 cycles each consisting of denaturation at 94°C for 45 sec, annealing at 55°C for 1 min, extension at 72°C for 1.30 min with final extension at 72°C for 10 min. Amplified DNA fragments were electrophoresed on a 1% agarose gel according to the procedure outlined by (Sambrook and Russel, 2001).

Results

The results revealed that, based on the final disease incidence, the genotypes Arka Gagan (3.57%), Arka Tejasvi (4.17%) and DCA-295 (9.09%) were found to be resistant. The genotype Arka Saanvi (29.50%) was found to be moderately resistant. However, remaining 45 genotypes DCA-204, DCA-221, DCA-222, DCA-223, DCA-224, DCA-230, DCA-235, DCA-236, DCA-237, DCA-238, DCA-239, DCA-240, DCA-241, DCA-242, DCA-243, DCA-244, DCA-248, DCA-250, DCA-270, DCA-276, DCA-277, DCA-280, DCA-287, DCA-288, DCA-290, DCA-291, DCA-292, DCA-294, DCA-298, DCA-300, DCA-301, DCA-302, DCA-303, DCA-304, DCA-306, DCA-330, DCA-333, DCA-334, DCA-335, DCA-336, Arka Harita, Arka Taanvi, Arka Khyati, Arka Shwetha and Byadgi Kaddi registered susceptible to the chilli leaf curl virus under natural epiphytic conditions with disease incidence ranging from 54.55 to 100 per cent (Table 2).

Table 2: Performance of chilli genotypes against chilli leaf curl virus disease in field (natural epiphytotic) conditions

Sl. No.	Genotype	Disease incidence (%)	AUDPC	Resistance reaction
1	DCA-204	94.12	6300.12	S
2	DCA-221	100.00	5506.44	S
3	DCA-222	100.00	5288.96	S
4	DCA-223	100.00	4551.44	S
5	DCA-224	100.00	4637.48	S
6	DCA-230	90.91	4836.32	S
7	DCA-235	100.00	7043.74	S
8	DCA-236	100.00	6650.00	S
9	DCA-237	100.00	5755.65	S
10	DCA-238	100.00	4694.65	S
11	DCA-239	100.00	5468.73	S
12	DCA-240	81.82	3531.90	S
13	DCA-241	83.33	3033.12	S
14	DCA-242	92.86	3825.06	S
15	DCA-243	100.00	5172.42	S
16	DCA-244	90.00	3395.00	S
17	DCA-248	100.00	4599.97	S
18	DCA-250	100.00	6249.87	S
19	DCA-270	100.00	6550.00	S
20	DCA-276	88.89	3811.03	S
21	DCA-277	90.00	4305.00	S
22	DCA-280	90.00	4655.00	S
23	DCA-287	93.75	4965.59	S
24	DCA-288	100.00	5556.23	S
25	DCA-290	95.00	4497.50	S
26	DCA-291	95.00	5337.50	S
27	DCA-292	100.00	3570.00	S
28	DCA-294	95.00	4392.50	S
29	DCA-295	9.09	286.33	R
30	DCA-298	76.92	2853.74	S
31	DCA-300	85.71	3049.93	S
32	DCA-301	88.89	3500.01	S
33	DCA-302	100.00	5355.00	S
34	DCA-303	93.33	5600.03	S

35	DCA-304	100.00	6055.00	S
36	DCA-306	100.00	5172.14	S
37	DCA-330	100.00	5700.01	S
38	DCA-333	95.00	5092.50	S
39	DCA-334	100.00	3604.77	S
40	DCA-335	95.00	3797.50	S
41	DCA-336	69.00	2257.50	S
42	Arka Tejasvi	4.17	277.30	R
43	Arka Harita	58.33	4389.67	S
44	Arka Taanvi	54.55	2993.01	S
45	Arka Gagan	3.57	212.41	R
46	Arka Saanvi	29.50	1937.95	MR
47	Arka Khyati	100.00	6182.74	S
48	Arka Shwetha	57.89	4034.19	S
49	Byadagi Kaddi	100.00	6099.99	S

I, Immune; HR, Highly resistant; R, Resistant; MR, Moderately resistant; S, Susceptible.

Further, the resistant genotypes were genotypically evaluated with chilli leaf curl virus coat protein-specific primers; the resistant genotypes were found negative for amplification of ChiLCV as compared with a susceptible check at 1.2 kb, confirming that genotypes Arka Gagan, Arka Tejasvi and DCA-295 were found to be resistant to the chilli leaf curl virus both phenotypically and genotypically under natural conditions (Table 2).

The native resistance of these genotypes to the virus was further confirmed with the artificial inoculation, in the presence study, the result showed that genotype Arka Gagan, Arka Tejasvi and DCA-295 (Fig. 1) showed incidence of 5.88, 5.55 and 6.25 per cent respectively in resistant genotypes, whereas severe leaf curl symptoms were observed in the susceptible genotype (73.68% disease incidence) (Table 3 and Table 4).



Fig. 1: The visualization of resistant lines DCA-295, Arka Gagan, Arka Tejasvi and susceptible check Byadagi Kaddi.

Table 3: Performance of chilli genotypes against chilli leaf curl virus disease in glasshouse (artificial inoculation) conditions

Sl. No.	Genotype	Disease incidence (%)	AUDPC	Resistance reaction
1	Arka Tejasvi	5.55	97.12	R
2	Arka Gagan	5.88	226.38	R
3	DCA-295	6.25	196.87	R
4	Byadagi Kaddi	73.68	3278.72	S

I, Immune; HR, Highly resistant; R, Resistant; MR, Moderately resistant; S, Susceptible.

Table 4: Grouping of chilli genotypes based on resistance levels against chilli leaf curl virus disease

Disease Scale	Plant response	Genotype
0	Immune	-
1	Highly resistant	-
2	Resistant	Arka Gagan, Arka Tejasvi and DCA-295
3	Moderately resistant	Arka Saanvi
4	Susceptible	DCA-204, DCA-221, DCA-222, DCA-223, DCA-224, DCA-230, DCA-235, DCA-236, DCA-237, DCA-238, DCA-239, DCA-240, DCA-241, DCA-242, DCA-243, DCA-244, DCA-248, DCA-250, DCA-270, DCA-276, DCA-277, DCA-280, DCA-287, DCA-288, DCA-290, DCA-291, DCA-292, DCA-294, DCA-298, DCA-300, DCA-301, DCA-302, DCA-303, DCA-304, DCA-306, DCA-330, DCA-333, DCA-334, DCA-335, DCA-336, Arka Harita, Arka Taanvi, Arka Kyati, Arka Shwetha and Byadagi Kaddi.

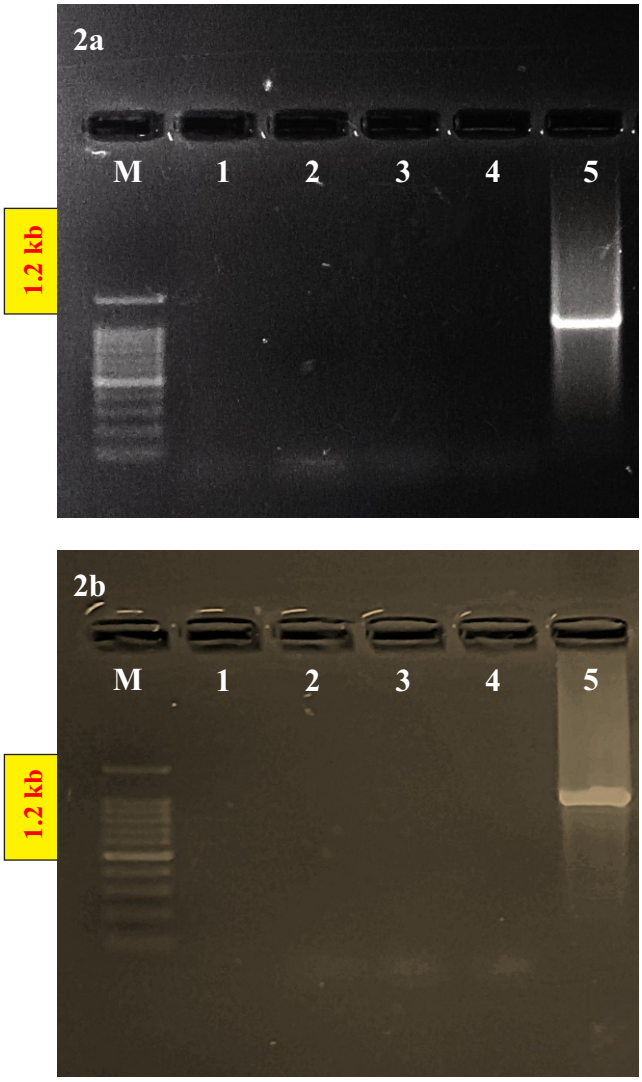


Fig. 2a & 2b: Agarose gel showing the amplification of the ChiLCV-CP primer with different genotypes under both field and glasshouse conditions, respectively.
M: Marker (100 bp ladder), Lane 1: Negative control (Sterile distilled water), Lanes 2, 3 and 4: Resistant genotypes DCA-295, Arka Gagan, Arka Tejasvi showing negative results for ChiLCV-CP primer, Lane 5: Byadagi Kaddi (susceptible check) amplification with ChiLCV-CP primer.

Molecular confirmation revealed that the resistant genotypes were found negative for amplification of ChiLCV as compared with a susceptible check at 1.2 kb (Fig. 2), confirming that the genotypes Arka Gagan, Arka Tejasvi and DCA-295 were found to be resistant to the chilli leaf curl virus both phenotypically and genotypically under artificial conditions also. Therefore, the resistance noted in these genotypes was not a result of whiteflies escaping or showing non-preference during screening, but rather due to inherent resistance mechanisms that either impede virus replication or restrict its movement within the plant. The resistance gene or allele present in these genotypes is the primary factor contributing to their observed resistance and immunity to leaf curl virus. Identifying new and stable chilli genotypes that are immune to leaf curl virus through various screening methods is essential for enhancing chilli crop improvement programs.

The area under the disease progress curve (AUDPC) for ChiLCV was calculated at various standard meteorological weeks, revealing that the AUDPC value was significantly less for resistant genotypes as compared to the susceptible ones. This observation indicates that, a lower AUDPC suggests the genotype either maintains a consistently lower level of disease over time (Table 2 and Table 3), therefore the genotypes Arka Gagan, Arka Tejasvi and DCA-295 have recorded 212.14, 277.30 and 286.33 respectively in natural

epiphytotic conditions and 226.38, 97.12 and 196.87 respectively in glasshouse conditions with less AUDPC as one of the parameters for confirming its status as a resistant genotype.

The apparent rate of infection for ChiLCV was estimated at various meteorological weeks on different genotypes, revealing that a lower r-value suggests the plant variety exhibits a slower rate of disease development. The resistant genotypes showed a slower rate of disease development compared with the susceptible genotypes (Table 5).

Discussion

In the recent past, PCR- based rapid detection methods were developed for viruses (Khan *et al.,* 2006; Chattopadhyay *et al.,* 2008). However, a method integrating the serological-based assay as well as PCR-based tools offer the most efficient system for the rapid detection of viruses (El-gaied *et al.,* 2008). The molecular results confirm that the genotypes Arka Gagan, Arka Tejasvi and DCA-295 were found to be resistant to the chilli leaf curl virus, which helps plant breeders identify resistance genes.

These findings conform with the results of Megharaj *et al.,* (2021), who screened 49 chilli genotypes under open-field conditions against a virus complex that was maintained by the Horticulture Research Experimental Station (HRES), Devihosur, Haveri, Karnataka. The virus-resistant genotypes were identified, namely DCA 295, DCA-107, DCA-154, DCA-222, DCA-224 and DCA-232. The present findings also confirmed the consistent levels of resistance in DCA-295. However, screening work should be a continuous process, since the *Begomovirus* population has most likely changed since then (Singh *et al.,* 2016) from largely monopartite to bipartite species; therefore, confirmation of resistance with recently developed virus strains is required as a continuous process.

The ChiLCV incidence during late-*Kharif* was noted. It might be due to the proximity of alternative hosts for the whitefly population, and the decreased rainfall, favourable relative humidity and temperature might cause increased whitefly activity, leading to predominance of ChiLCV incidence (Bhute *et al.,* 2012). The virus infestation leads to malformation and reduction in leaf areas varied with the cultivars, time of infection and climatic conditions (Abu-Kasim, 1986). The maximum virus incidence could be due to several factors such as crop proximity to alternative hosts, climatic conditions, the supply of poor-quality seed and seedlings and vector activity (Shah *et al.,* 2009). The

Table 5: Rate of disease development among different chilli genotypes against chilli leaf curl virus disease in field (natural epiphytotic) condition

Sl. No.	Genotype	Rate of infection (r)												Average
		05/10/2023	12/10/2023	19/10/2023	26/10/2023	2/11/2023	9/11/2023	16/11/2023	23/11/2023	1/12/2023	8/12/2023	15/12/2023	22/12/2023	28/12/2023	
1	DCA-204	0.046	0.073	0.032	0.120	0.032	0.034	0.000	0.000	0.086	0.066	0.000	0.000	0.000	0.038
2	DCA-221	0.000	0.000	0.082	0.091	0.027	0.027	0.081	0.000	0.099	0.327	0.000	0.000	0.000	0.056
3	DCA-222	0.059	0.084	0.034	0.088	0.000	0.028	0.028	0.029	0.000	0.000	0.435	0.000	0.000	0.060
4	DCA-223	0.000	0.000	0.100	0.121	0.000	0.000	0.094	0.000	0.046	0.108	0.343	0.000	0.000	0.062
5	DCA-224	0.000	0.000	0.000	0.147	0.000	0.000	0.164	0.033	0.036	0.388	0.000	0.000	0.000	0.059
6	DCA-230	0.000	0.000	0.000	0.221	0.000	0.000	0.094	0.231	0.000	0.000	0.000	0.000	0.000	0.042
7	DCA-235	0.108	0.040	0.036	0.285	0.000	0.000	0.300	0.000	0.000	0.000	0.000	0.000	0.000	0.059
8	DCA-236	0.041	0.041	0.064	0.171	0.036	0.000	0.034	0.000	0.336	0.000	0.000	0.000	0.000	0.056
9	DCA-237	0.152	0.000	0.084	0.178	0.029	0.000	0.031	0.000	0.118	0.000	0.000	0.287	0.000	0.067
10	DCA-238	0.000	0.000	0.156	0.089	0.000	0.037	0.138	0.000	0.035	0.041	0.050	0.307	0.000	0.066
11	DCA-239	0.000	0.000	0.202	0.075	0.000	0.096	0.257	0.000	0.000	0.000	0.000	0.232	0.000	0.066
12	DCA-240	0.000	0.000	0.000	0.000	0.000	0.000	0.315	0.046	0.049	0.000	0.057	0.000	0.000	0.036
13	DCA-241	0.000	0.000	0.000	0.000	0.000	0.099	0.117	0.000	0.000	0.220	0.055	0.000	0.000	0.038

14	DCA-242	0.000	0.096	0.000	0.062	0.000	0.049	0.000	0.081	0.000	0.168	0.055	0.000	0.000	0.039	
15	DCA-243	0.000	0.152	0.000	0.233	0.000	0.028	0.029	0.000	0.000	0.148	0.000	0.287	0.000	0.067	
16	DCA-244	0.000	0.000	0.102	0.000	0.000	0.000	0.069	0.000	0.057	0.154	0.060	0.000	0.081	0.040	
17	DCA-248	0.000	0.000	0.000	0.159	0.091	0.000	0.147	0.000	0.000	0.448	0.000	0.000	0.000	0.065	
18	DCA-250	0.096	0.112	0.042	0.280	0.000	0.000	0.315	0.000	0.000	0.000	0.000	0.000	0.000	0.065	
19	DCA-270	0.096	0.000	0.112	0.271	0.000	0.051	0.071	0.000	0.000	0.244	0.000	0.000	0.000	0.065	
20	DCA-276	0.000	0.000	0.000	0.176	0.000	0.000	0.060	0.000	0.113	0.000	0.000	0.148	0.000	0.038	
21	DCA-277	0.000	0.149	0.081	0.089	0.000	0.000	0.026	0.025	0.025	0.107	0.068	0.045	0.000	0.047	
22	DCA-280	0.000	0.000	0.149	0.113	0.083	0.025	0.050	0.053	0.000	0.096	0.045	0.000	0.000	0.047	
23	DCA-287	0.061	0.047	0.040	0.036	0.000	0.065	0.000	0.031	0.140	0.000	0.049	0.068	0.000	0.041	
24	DCA-288	0.000	0.108	0.000	0.140	0.031	0.032	0.069	0.040	0.000	0.049	0.000	0.000	0.300	0.059	
25	DCA-290	0.058	0.000	0.044	0.069	0.000	0.000	0.083	0.050	0.107	0.032	0.144	0.000	0.000	0.045	
26	DCA-291	0.000	0.044	0.098	0.053	0.025	0.050	0.000	0.053	0.060	0.000	0.081	0.063	0.000	0.041	
27	DCA-292	0.000	0.000	0.000	0.149	0.113	0.000	0.057	0.051	0.025	0.025	0.113	0.144	0.213	0.069	
28	DCA-294	0.000	0.000	0.058	0.081	0.032	0.000	0.083	0.050	0.107	0.068	0.107	0.000	0.000	0.045	
29	DCA-295	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.051	0.000	0.000	0.000	0.000	0.000	0.004	
30	DCA-298	0.000	0.000	0.000	0.000	0.000	0.000	0.161	0.094	0.118	0.041	0.045	0.000	0.000	0.035	
31	DCA-300	0.000	0.000	0.000	0.096	0.000	0.000	0.062	0.091	0.000	0.229	0.051	0.000	0.000	0.041	
32	DCA-301	0.000	0.000	0.092	0.059	0.045	0.000	0.000	0.103	0.142	0.031	0.118	0.000	0.000	0.045	
33	DCA-302	0.000	0.000	0.132	0.093	0.026	0.030	0.051	0.126	0.042	0.286	0.000	0.000	0.000	0.060	
34	DCA-303	0.000	0.000	0.204	0.214	0.000	0.000	0.037	0.000	0.091	0.069	0.000	0.000	0.000	0.047	
35	DCA-304	0.149	0.081	0.032	0.158	0.000	0.053	0.029	0.068	0.107	0.000	0.213	0.000	0.000	0.069	
36	DCA-306	0.092	0.059	0.045	0.132	0.028	0.000	0.056	0.029	0.031	0.183	0.000	0.222	0.000	0.067	
37	DCA-330	0.062	0.000	0.091	0.111	0.000	0.036	0.000	0.039	0.000	0.094	0.315	0.000	0.000	0.058	
38	DCA-333	0.058	0.000	0.081	0.140	0.000	0.050	0.000	0.053	0.029	0.000	0.068	0.045	0.063	0.045	
39	DCA-334	0.000	0.000	0.000	0.000	0.000	0.199	0.073	0.062	0.059	0.135	0.051	0.290	0.000	0.067	
40	DCA-335	0.000	0.000	0.000	0.058	0.000	0.044	0.098	0.079	0.050	0.149	0.045	0.063	0.000	0.045	
41	DCA-336	0.000	0.000	0.000	0.000	0.000	0.000	0.105	0.140	0.102	0.000	0.097	0.000	0.000	0.034	
42	Arka Tejasvi	0.000	0.000	0.000	0.000	0.034	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.002	
43	Arka Harita	0.164	0.052	0.088	0.000	0.000	0.000	0.000	0.021	0.000	0.000	0.021	0.000	0.000	0.027	
44	Arka Taanvi	0.000	0.147	0.000	0.079	0.000	0.085	0.048	0.000	0.000	0.000	0.023	0.000	0.023	0.031	
45	Arka Gagan	0.000	0.000	0.000	0.000	0.000	0.038	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.002	
46	Arka Saanvi	0.088	0.000	0.055	0.075	0.000	0.000	0.078	0.000	0.000	0.000	0.000	0.000	0.000	0.023	
47	Arka Khyati	0.000	0.176	0.116	0.057	0.000	0.000	0.148	0.000	0.000	0.000	0.000	0.000	0.287	0.060	
48	Arka Shwetha	0.151	0.115	0.058	0.027	0.000	0.000	0.000	0.026	0.000	0.000	0.000	0.000	0.026	0.031	
49	Byadagi Kaddi	0.062	0.000	0.202	0.000	0.000	0.036	0.039	0.000	0.094	0.315	0.000	0.000	0.000	0.058	

genotypes Arka Gagan, Arka Tejasvi and DCA-295 had the least virus disease incidence, which revealed resistance to chilli viruses.

Recently, the first authentic molecular evidence was reported for the infection of ChVMV in *Capsicum chinense* L. from low- to moderate-temperature geographical areas of North Eastern India (Banerjee *et al.,* 2012). The predominant seed production activities of Solanaceous and Cucurbitaceous vegetable crops act as a host for viruses throughout the year. Due to the wide genetic variability of the genus *Capsicum*, it is possible to search and use germplasm as a source of resistance to various chilli viruses with stable resistance. The marker trait (resistance) linkage is suitable for the isolation of resistant genotypes and the development of resistant cultivars through marker-assisted breeding.

Conclusion

Identifying new and stable chilli genotypes that are resistant to leaf curl virus through various screening methods is essential for improving chilli crops. Investigations into the genotypic reactions to leaf curl virus infection and sources of resistance were carried out to support an effective resistance breeding program. The promising genotypes Arka Gagan, Arka Tejasvi and DCA-295 were found to be resistant under both field as well as glasshouse conditions. Further, it was confirmed through a molecular method using a CP-specific primer. These genotypes can be utilized to develop multiple disease-resistant chilli varieties effectively.

Acknowledgements

We would like to thank the University of Agricultural Sciences, Raichur, Karnataka; Horticulture Research and Extension Centre, Haveri (Devihosur), Karnataka; and Indian Institute of Horticultural Research, Bengaluru for supporting and facilitating the study.

References

Abu-Kassim AB (1986) Virus diseases of horticultural crops in Malaysia. In: *FFTC Book Series*. Food and Fertilizer Technology Center for the Asian and Pacific Region, Taipei, pp 193.

Ashwathappa KV (2021) Molecular characterization of chilli leaf curl virus in Karnataka. Ph.D. Thesis, University of Agricultural Sciences, Raichur, Karnataka, India, pp 95.

Banerjee A, R Dutta, S Roy and SV Ngachan (2012) First report of chilli veinal mottle virus (ChVMV) in Naga chilli (*Capsicum chinense*) in Meghalaya, India. *Virus Dis.* 25(1): 142-143.

Bhute NK, BB Bhosle, BV Bhede and DG More (2012) Population dynamics of major sucking pests of Bt cotton. *Indian J. Entomol.* 74(3): 246-252.

Brown JK, DR Frohlich and RC Rosell (1995) The sweet potato/silverleaf whiteflies; biotypes of *Bemisia tabaci* or a species complex. *Annu. Rev. Entomol.* 40: 511-534.

Chattopadhyay B, AK Singh, T Yadav, CM Fauquet, NB Sarin and S Chakraborty (2008) Infectivity of the cloned components of a begomovirus: DNA beta complex causing chilli leaf curl disease in India. *Arch. Virol.* 153: 533-539.

El-Gaied LF, MI Salama, AM Salem, FN Amani, NA El-Deen and A Abdallah (2008) Molecular and serological studies on a plant virus affecting strawberry. *Arab J. Biotechnol.* 11: 303-314.

Ilyas AK and M Khan (1996) Studies on mosaic complex of chilli (*Capsicum annuum* L.). M.Sc. (Agri.) Thesis, University of Agricultural Sciences, Bangalore, Karnataka, India.

Khan MS, SK Raj and R Singh (2006) First report of Tomato leaf curl New Delhi virus infecting chilli in India. *Plant Pathol.* 55(2): 289.

Kumar S and M Rai (2005) Chile in India. *Chile Pepper Inst. Newslett.* 22: 1-3.

Maruthi MN, J Colvin, SE Seal, G Gibson and J Cooper (2002) Co-adaptation between cassava mosaic gemini viruses and their local vector populations. *Virus Res.* 86: 71-85.

Megharaj KC, PS Ajjappalavara, HB Patil, M Abdul Kareem, S Ratnakar, S Raghavendra, G Hegde and DC Manjunathagowda (2021) Validation for the prevalence of virus complex and identification of virus resistance lines among the genotypes of chilli (*Capsicum annuum* L.). *Vegetos* 34(3): 684-691.

Oraon UB and J Tarafdar (2018) Occurrence and distribution of chilli leaf curl complex disease in West Bengal. *Biomed. J. Sci. Tech. Res.* 3(4): 3515-3519.

Sambrook J and DW Russell (2001) *Molecular Cloning: A Laboratory Manual*, 3rd edn. Cold Spring Harbor Laboratory Press, New York, USA.

Senanayake DMJB, B Mandal, S Lodha and A Varma (2007) First report of chilli leaf curl virus affecting chilli in India. *Plant Pathol.* 56(2): 343.

Shah H, T Yasmin, M Fahim, S Hameed and MI Haque (2009) Prevalence, occurrence and distribution of chilli veinal mottle virus in Pakistan. *Pak. J. Bot.* 41(2): 955-965.

Shih SL, WS Tsai and SK Green (2003) Molecular characterization of tomato and chilli leaf curl begomoviruses from Pakistan. *Plant Dis.* 87: 200.

Singh AK, N Kushwaha and S Chakraborty (2016) Synergistic interaction among begomoviruses leads to the suppression of host defense-related gene expression and breakdown of resistance in chilli. *Appl. Microbiol. Biotechnol.* 100: 4039-4049.

Thakur H, SK Jindal, A Sharma and MS Dhaliwal (2018) Chilli leaf curl virus disease; a serious threat for chilli cultivation. *J. Plant Dis. Prot.* 125: 239-249.

Vanderplank JE (2012) *Disease Resistance in Plants*. Elsevier.

Wilcoxson RD, B Skovmand and AH Atif (1975) Evaluation of wheat cultivars for ability to retard development of stem rust. *Ann. Appl. Biol.* 80(3): 275-281.