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Identification of Source(s) of Resistance to Chilli Leaf Curl Virus Disease in Chilli (*Capsicum annuum* L.)

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ABSTRACT

Chilli is one of the most valuable crops in India and world grown for its multiple uses. Chilli succumbs to numerous viral diseases, among all, chilli leaf curl virus (ChLCV) is a devastating disease hampering the true genetic yield potential. This urges chilli breeders to address the catastrophic viral disease to mitigate losses incurred by farmers following breeding methodologies. Identification of stable resistant sources forms the bedrock for resistant breeding program. With an objective to identify resistant sources for chilli leaf curl disease, twenty-eight chilli genotypes of which seventeen belonging to breeding lines and eleven belonging to germplasm accessions were screened for responses to chilli leaf curl virus infection under natural epiphytotic conditions during summer 2020. A wide range of symptoms including curling, cupping, yellowing of leaves and plants with limited or no flowers and fruits were observed on genotypes screened against ChLCV infection. Analysis of variance for per cent disease index and coefficient of incidence indicated the presence of significant differences among 28 chilli genotypes. Mean and variance of per cent disease index and coefficient of incidence were comparable between breeding lines and germplasm accessions. Two genotypes (Bhut Jolokia and S343) were found to be highly resistant and four genotypes namely PDL1, PDL2, AVRDC2 and LCVT 7 exhibited resistant reaction. The identified resistant genotypes should further be confirmed for their disease reaction following phenotypic assays through artificial inoculation and molecular assays through PCR using chilli leaf curl virus specific primers. Thus, genotypes exhibiting true resistant response can be incorporated in resistance breeding programs.

Keywords : *Chilli, ChLCV, Resistance*, Genotypes

CHILLI is one of the celebrated crops worldwide by virtue of its widespread cultivation for its multiple uses. Chilli has numerous applications in essentially dietary, culinary, pharmaceutical, cosmetic as well as food industries. India being the world's largest producer, consumer and exporter of chilli is planted to largest area of 7.33 lakh ha accounting for 42.81 per cent of world area. In Karnataka, annual production of green chilli is 607.94 thousand MT from an area of 45.53 thousand ha with productivity of 13.38 tons hectare $^{-1}$ (Channabasava, 2020). With respect to pests and diseases, chilli serves as host for plethora of insects and pathogens. Therefore, chilli production is severely constrained by various biotic stresses including infestation by all major group of pathogens (fungus, bacteria and virus).

Chilli is known to be affected by more than 35 viruses. Twenty-four viruses are reported to affect chilli naturally, among them 11 have been reported from India namely Pepper vein banding virus, Pepper veinal mottle virus, chilli leaf curl

virus *etc*., (vijeth *et al*., 2020). Out of these viruses, Chilli leaf curl virus (ChLCV) is the most devastating in terms of incidence and losses incurred. In severe conditions, it could cause 100 per cent marketable yield (fruit) loss (Rao *et al*., 2020). Tropical and subtropical regions of the world where hot pepper is cultivated face heavy losses due to leaf curl disease (Srivastava *et al.*, 2017) thus, gaining the attention of chilli breeders all over the world.

ChLCV disease is caused by genus Begomovirus belonging to family Geminiviridae. Begomovirus members are characterized by twin icosahedral particles (18×30 nm size) with their genome consisting of one or two circular, ssDNA components (2.5-3.0 kb) known as DNA A and DNA B (Zehra *et al*., 2017). The virus, vectored by whiteflies (*Bemecia tabaci*), allows rapid and efficient transmission through indiscriminant feeding. The interaction studies of ChLCV with Asia-I cryptic species revealed that whiteflies with 24 h of acquisition access period and inoculation access period each successfully transmitted the virus with 100 per cent transmission (Madhu *et al*., 2021). The vector population thrives and multiplies best in natural conditions of 25-35^{0}C which corresponds to late winter-summer season in India. Hence, ChLCV is mostly severe to summer crop. However, other season cultivations are infected sufficiently to cause economic losses (Nigam *et al*., 2015) demanding scientific attentions and breeding interventions.

Characteristic field symptoms of leaf curl disease are leaf curling, puckering, rolling, shortening of internodes and petioles, blistering of leaf interveinal areas, thickening and swelling of the veins, older leaves turning out leathery and brittle, crowding of leaves and severely affected plants are stunted and produce no fruit (Srivastava *et al.*, 2017; Vijeth *et al*., 2020 and Rao *et al*., 2020). Typical leaf curl symptoms and increase in disease severity in infected plants are due to the presence of cognate beta-satellites associated with the virus.

Non-genetic approaches for management of ChLCV disease includes use of pesticides to control vectors, removal of diseased plants and agronomic interventions. These approaches have met with limited success. Recommendation to spray insecticides for efficient control of whiteflies has resulted in their indiscriminate or excessive use, contributing to environmental degradation as well as resistance and resurgence in pests. In turn, affecting other seasonal crops grown in crop rotation and combination with other crops.

Under these circumstances, genetic resistance appears to be the most ecofriendly approach, where breeders scout for genotypes that carry genes or combination of genes which can increase plants ability to resist / tolerate infection. The success of disease resistance breeding depends on genetic variability and reliable evaluation tests employed for identification of resistant sources. Screening chilli accessions against chilli leaf curl viral disease would help in recognition of available resistant germplasm against the disease. Further, enabling their utilization in chilli breeding programs. However, evidence of research reports on strong or high-level of resistance against ChLCV disease in cultivated genotypes of chilli is limited. Further, synergistic interaction among different begomoviruses infecting chilli results in breakdown of natural resistance in otherwise resistant chilli plants to begomovirus infection (Singh *et al*., 2016). Till date no validated resistant cultivar is available, atleast in public sector, substantiating the need for identifying strong sources of resistance and in turn developing resistant varieties to mitigate losses experienced by farmers due to ChLCV disease (Kumar *et al*., 2019). Identification and involving ChLCV resistant sources in breeding programs would enable deciphering the inheritance pattern of ChLCV resistance. Further, breeders can device suitable breeding strategies or schemes to introgress resistance into elite horticultural background. Hence current investigation was planned with an objective of screening working collections to identify chilli genotypes resistant to ChLCV infection under natural epiphytotic conditions.

Material and Methods

Experimental Material and Field Evaluation

The study consisted of 28 genotypes, 21 of them were collected from different parts of India and seven from World Vegetable Center, Taiwan where pedigree and place of collection are listed in Table 1. The 28 genotypes included in the investigation could be categorized into seventeen breeding lines and eleven germplasm accessions that bracketed released varieties and wild species. The proportion of genotypes belonging to different categories along

Table 1

Details of chilli genotypes included in the experiment

Genotypes	Pedigree / Source	Place of collection
Breeding lines		
AVRDC2	PSP-11, World Vegetable Centre	Taiwan
AVRDC16	Pant C-1, World Vegetable Centre	Taiwan
BDL1	*C. annuum* × *C. buccatum*	Bengaluru, Karnataka
BDL2	*C. annuum* × *C. buccatum*	Bengaluru, Karnataka
BDL3	*C. annuum* × *C. buccatum*	Bengaluru, Karnataka
LCVT6	Derived from intra species cross	Bengaluru, Karnataka
LCVT7	Derived from intra species cross	Bengaluru, Karnataka
LCVT8	Derived from intra species cross	Bengaluru, Karnataka
PDL1	Line derived from Pride, an elite hybrid	Bengaluru, Karnataka
PDL2	Line derived from Pride, an elite hybrid	Bengaluru, Karnataka
S343	Male parent of CH27 hybrid, PAU	Ludhiana, Punjab
ADL4	Derived from intra species cross	Bengaluru, Karnataka
CMS 6B	World Vegetable Centre	Taiwan
CMS 7B	World Vegetable Centre	Taiwan
CMS 8B	World Vegetable Centre	Taiwan
CMS 9B	World Vegetable Centre	Taiwan
CMS 10B	World Vegetable Centre	Taiwan
Germplasm accessions		
BYADAGI KADDI (BK)	Local Collection	Haveri, Karnataka
BYADAGI DABBI (BD)	Local Collection	Haveri, Karnataka
BHUT JOLOKIA (BJ)	*Capsicum chinense* × *Capsicum frutescens*	Assam, India
GOWRI BIDANUR (GB)	Local Collection	Chikballapur, Karnataka
LCA424	LAM Research Station	Guntur, Andhra Pradesh
APARNA (LCA 1068)	LAM Research Station	Guntur, Andhra Pradesh
PANT-C-1	NP46A × Kandhari	GBPUAT
PUSA SADABAHAR	Pusa Jwala × IC 31339	IARI, New Delhi
TIWARI	Punjab Agriculture University	Ludhiana, Punjab
UTKAL AVA (UA)	OUAT	Bhubaneswar, Orissa
UTKAL RASHMI	OUAT	Bhubaneswar, Orissa

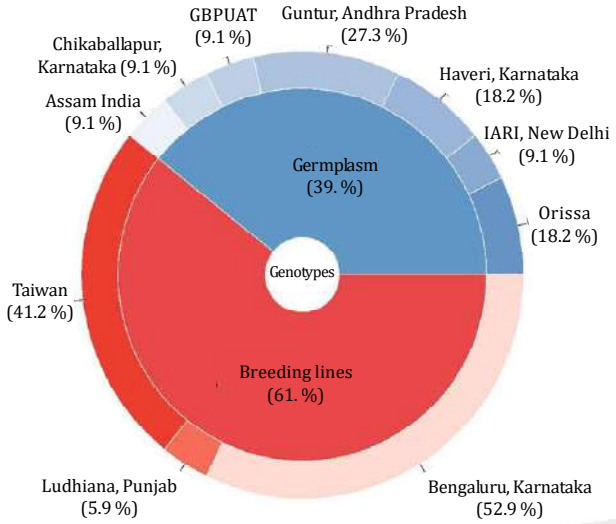

Fig. 1 : Pictorial representation of breeding lines and germplasm accessions used in the study along with place of collection

with place of collection are depicted in Fig. 1. These twenty-eight genotypes were evaluated in randomized complete block design (RCBD) with two replications under natural epiphytotic conditions during summer 2020 at experimental plots of K-block, Department of Genetics and Plant Breeding, College of Agriculture, Gandhi Krishi Vigyana Kendra, Bengaluru. During summer, natural ChLCV epiphytotics resulting due to threshold virus and vector population was taken advantage of to screen genotypes for their responses to natural ChLCV infection. It was evident from previous season (Summer 2019) where prevalence of ChLCV disease was high enough to screen genotypes against ChLCV infection.

Screening of Chilli Genotypes against Responses to ChLCV under Natural Epiphytotic Conditions

Field screening was undertaken to evaluate 28 chilli genotypes against responses to ChLCV disease when the natural ChLCV pressure was at its peak owing to enormous vector population in the study area. Measures to control ChLCV or vector were not followed in the entire experimental plot right from transplanting till final harvesting to avoid any chance of disease escape as a result of decline in white fly population. While, regular agronomic practices were followed to raise a good crop.

ChLCV disease was scored on ten randomly chosen plants from each genotype in each of the replications at 60 days after transplanting when ChLCV manifested clear symptoms. The severity of symptom was recorded on the basis of severity scale (0-5) developed by Banerjee and Kalloo and modified by Kumar *et al*. (2006). The scale used to classify the genotypes based on severity symptoms is presented in Table 2.

Variables measured based on disease scoring were disease incidence and per cent disease index, which were calculated using the following formulae,

$$\text{Disease incidence (DI)} = \frac{\text{No. of plants infected}}{\text{Total no of plants in particular genotype}} \times 100$$

Per cent Disease Index (PDI) : Based on individual scores given to each genotype following disease severity scale (0-5), PDI was calculated by the following formulae using Microsoft excel office 2016. PDI values indicates severity of ChLCV infection, lower the PDI value lower is the susceptibility of a genotype and vice versa.

$$\text{PDI} = \frac{[\text{Sum of individual plant scores}]}{[\text{Total number of plants observed} \times \text{maximum grade}]} \times 100$$

Coefficient of incidence (CI) was calculated for each genotype by multiplying PDI and DI values and by dividing with 100. Further, CI values were used to assign specific disease reaction for 28 genotypes against ChLCV infection (Kumar *et al*., 2006).

Statistical Analysis

Analysis of Variance and Box-whisker plot twenty-eight chilli genotypes were subjected to analysis of variance to detect differences among genotypes for PDI and CI using R software version 4.0.4 (R core team, 2020). The total variation, among chilli genotypes for PDI and CI was partitioned into

TABLE 2

Disease reaction classes based on disease severity adapted from Kumar *et al,* (2006)

Visual symptom	Description of symptoms	Severity score	Disease reaction class
	No symptoms	0	Immune
	0–5% curling and clearing of upper leaves	1	Highly resistant (HR)
	6-25% curling, clearing of leaves and swelling of veins	2	Resistant (R)
	26–50% curling puckering and yellowing of leaves and swelling of veins	3	Moderately susceptible (MS)
	51–75% leaf curling and stunted plant growth and blistering of internodes	4	Susceptible (S)
	More than 75% curling and deformed small leaves, stunted plant growth with small flowers and no or small fruit set	5	Highly susceptible (HS)

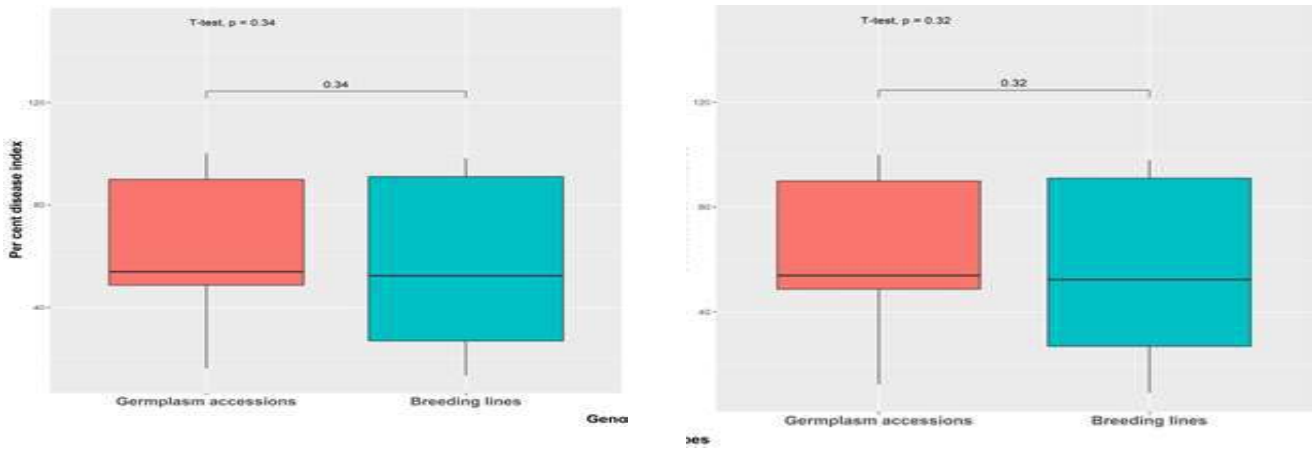

Fig. 2 : Box plots depicting per cent disease index and coefficient of incidence between breeding lines and germplasm accessions

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sources attributable to genotypes, germplasm, breeding lines and residuals. To represent the variability among breeding lines and germplasm, box-whisker plots were plotted to visualize the data through different quartiles for PDI and CI as presented in Fig. 3.

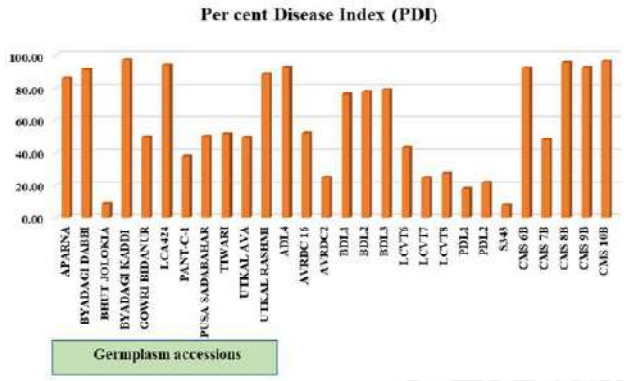

Fig. 3 : Bar graph depicting variability for PDI among germplasm accessions and breeding lines

F test and t test were performed to detect any differences between germplasm and breeding lines for variance and mean of PDI and CI, respectively. A Post hoc test (Turkey's test) was performed to detect significant differences among the means of genotypes for PDI and CI. Means of two genotypes with at least one letter common are not statistically significant. Further, genotypes were grouped into different disease reaction categories based on CI as represented in Table 4.

TABLE 3

Analysis of variance for per cent disease index (PDI), disease incidence (DI) and coefficient of incidence (CI) among breeding lines and germplasm accessions in chilli

Sources of Variation	Degrees of Freedom	Per cent disease index	Coefficient of incidence
Replication	1	4.17	19.58
Genotypes	27	1820.21 ***	1990.83 ***
Germplasm accessions	10	1642.83 ***	1779.23 ***
Breeding lines	16	2005.28 ***	2200.39 ***
Residuals	27	14.50	13.90

*** Significant @ 0.001 per cent (p value <0.001)

TABLE 4

Reaction of chilli genotypes against chilli leaf curl virus disease under natural epiphytotic conditions

Genotypes	Mean Score @ 60 DAT	Per cent Disease Index	Coefficient of Incidence	Response reaction
Germplasm				
BHUT JOLOKIA	0.63	08.84 ^{i}	03.56	HR
PANT-C-1	1.90	37.86 ^{efg}	36.48	MS
UTKAL AVA	2.47	49.38 ^{e}	49.38	MS
GOWRI BIDANUR	2.48	49.62 ^{e}	49.62	MS
TIWARI	2.58	51.67 ^{e}	51.67	S
PUSA SADABAHAR	2.61	52.15 ^{e}	52.15	S
APARNA	4.29	85.83 ^{abcd}	85.83	HS
UTKAL RASHMI	4.33	88.10 ^{abcd}	88.10	HS
BYADAGI DABBI (BD)	4.46	90.83 ^{abcd}	90.83	HS
LCA 424	4.62	94.00 ^{ab}	94.00	HS
BYADAGI KADDI (BK)	4.77	97.14 ^{a}	97.14	HS
Mean	3.19	64.12	63.52	
Breeding lines				
S343	0.48	8.16 ^{i}	4.23	HR
PDL1	1.00	18.00 ^{hi}	12.80	R
PDL2	1.17	22.00 ^{hi}	19.69	R
AVRDC2	1.24	24.81 ^{fgh}	22.91	R
LCVT7	1.28	24.68 ^{gh}	23.60	R
LCVT8	1.44	27.18 ^{gh}	27.18	MS
LCVT6	2.24	43.08 ^{ef}	43.08	MS
CMS 7B	2.39	47.78 ^{e}	47.78	MS
AVRDC 16	2.61	50.12 ^{e}	50.12	MS
BDL1	3.80	76.03 ^{d}	76.03	HS
BDL2	3.80	77.62 ^{cd}	77.62	HS
BDL3	3.95	79.05 ^{bcd}	79.05	HS
CMS 6B	4.59	91.82 ^{abc}	91.82	HS
CMS 9B	4.61	92.22 ^{abc}	92.22	HS
ADL4	4.62	92.38 ^{abc}	92.38	HS
CMS 8B	4.77	95.42 ^{a}	95.42	HS
CMS 10B	4.81	96.27 ^{a}	96.27	HS
Mean	2.87	58.97	56.01	

Two means with at least one letter common are not statistically significant

RESULTS AND DISCUSSION

To identify resistant sources against ChLCV disease, screening of genotypes under natural epiphytotic conditions would be a preliminary step as screening large number of genotypes through challenge inoculation becomes difficult and resource demanding.

The Mysore Journal of Agricultural Sciences

Keeping this in mind, the proposition of field screening implied best to eliminate genotypes that shows susceptibility under natural conditions there by reducing the number of genotypes to screen in artificial epiphytotic conditions.

Generally, in summer cropping periods, whitefly population surges and reaches peak during fruiting stages while in *kharif* it is maximum during the vegetative and flowering stages, (Srivastava *et al.*, 2017). The cropping season 2020 summer witnessed heavy incidence of ChLCV disease in the field coupled with large population of whitefly vector as anticipated. The 28 genotypes screened under the present investigation exhibited a wide range of leaf curl virus symptom variability under natural field conditions. Upward curling of leaves, leaf bending and cupping were also observed. Enations on leaves and vein thickening were pronounced in some plants. Severely affected plants showed bushy appearance (stunted growth) due to shortened internodes with numerous small and curly leaves in the upper portion of the plants. These plants were also devoid of flowers and fruits. Similar observations were documented and reported for first time in farmer's fields of chilli in Rajasthan during 2004 (Senanayake *et al*., 2007). Development of early and severe symptoms on genotypes under investigation suggested that the disease was in epidemic form and screening for ChLCV infection under natural conditions was effective.

Results from analysis of variance (Table 3) indicated significant differences ($p<0.01$) among genotypes for responses to ChLCV disease based on PDI and CI. Genotypes within breeding lines as well as germplasm lines performed significantly different for both PDI and CI as evidenced by significant mean sum of squares (Table 2.). Differential response of genotypes to ChLCV incidence and symptom expression could be attributed to the fact that the disease incidence and its spread are influenced by the occurrence and population dynamics of the vector whitefly and weather conditions in the agroecosystem. Another probable reason could be the presence of different gene combinations involved in governing resistance to ChLCV among 28 chilli genotypes.

Box-whisker plots of PDI and CI between germplasm and breeding lines indicated non-significant differences ($p = 0.3$) for mean PDI and CI. Variance for PDI and CI was comparable between breeding lines and germplasm as both groups included susceptible and resistant genotypes (Fig. 2).

PDI values ranged from to 8.16, (S343) to 97.14 (Byadagi Kaddi) (Fig. 3) indicating prevalence of greater amount of variability for responses to ChLCV disease among chilli genotypes. Low PDI value indicates less severity of disease. Accordingly, least PDI value was observed on S343 indicating as highly resistant genotype based on its CI value (4.23) as presented in Fig. 4. Byadgi kaddi showed highest PDI value (97.14) registering up to 100 *per cent* severity and thus being categorized as highly susceptible genotype based on its CI value (97.14) as shown in Fig. 4. Since byadagi chillies are extensively grown in Karnataka for export purpose for their colour value, introgression of resistance to ChLCV disease into such genetic backgrounds is highly relevant and need of the hour. Genotypes showing contrasting responses (S343(R), BJ(R), BK(S) and BD(S)) for ChLCV disease based on PDI and CI values could be involved in crossing programs to understand genetics of responses to ChLCV after confirming their true disease reaction through artificial disease epiphytotic.

Responses of twenty-eight genotypes to ChLCV disease under natural conditions are presented in Table 4. Out of 11 germplasm lines, Bhut jolokia was found to be highly resistant, these results are in accordance with those reported by Rai *et al.* (2014) and Adluri *et al.* (2017). Three genotypes (Pant c-1, Utkalava and Gowri Bidanur local) exhibited moderate susceptibility and rest of the genotypes expressed susceptible / highly susceptible reaction.

Among Breeding lines S343 showed highly resistant reaction where as PDL1, PDL2, LCVT7 and AVRDC2 were resistant, the resistance response of S343 is in agreement with reports documented by

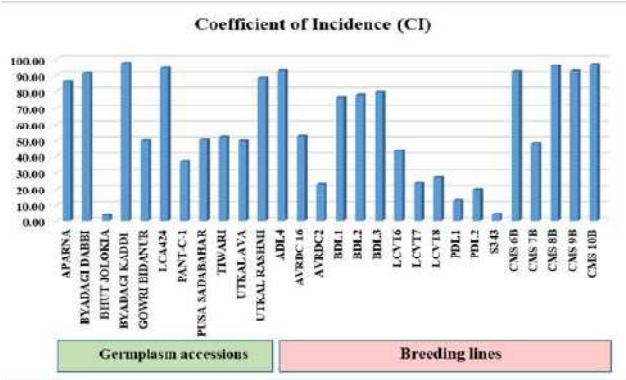

Fig. 4 : Bar graph depicting variability for coefficient of incidence among germplasm accessions and breeding lines

Thakur *et al*. (2019), four genotypes LCVT8, LCVT6, CMS 7 and AVRDC 16 had expressed moderately susceptible reaction while, the rest were either susceptible / highly susceptible. In our study none of the accessions exhibited symptomless or immune response. Out of 28 genotypes, 7 per cent (two genotypes) was categorized as highly resistant, 14 per cent (four genotypes) as resistant and almost 50 per cent of the genotypes were categorized as susceptible (Fig.4). This indicates resistance for ChLCV disease is rare and demands inclusion of more number of genotypes for identification of resistant sources. Phenotypic Coeffecient of Variation (PCV) for PDI and CI was 50.67 and 53.84 respectively indicating the presence of high variability for responses to ChLCV disease infection. Similarly, Genotypic Coefficient of Variation(GCV) was high for PDI (50.31) and CI (53.49). Narrow differences

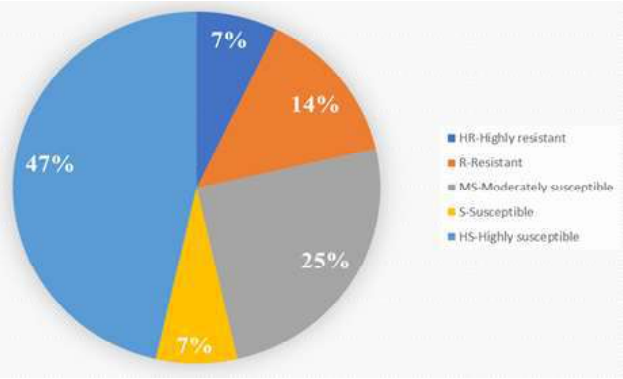

Fig. 5 : Proportion of genotypes falling into different disease reaction categories

between PCV and GCV coupled with high heritability for PDI (98.55 per cent) and CI (98.71 per cent) suggests these traits are less influenced by environment which could be further attributed to uniform disease infection pressure throughout disease progression and maximum expression.

Identification of resistant sources from germplasm is first step in resistance breeding programs. Screening of genotypes under natural epiphytotic conditions serve as guiding light for subsequent steps in breeding programs. However, under natural conditions, resistance exhibited by some lines cannot be inferred as absolute resistance as genotypes might have managed to escape from virus-acquired-whiteflies resulting in insufficient viral loads for disease appearance. Under field conditions, the symptoms may also be due to sucking pests (thrips and mites) that mimics leaf curl symptoms which might create confusion to the breeder while phenotyping for the trait. Apart from the above-mentioned reasons, annual, seasonal and local variations strongly influence the incidence and severity of virus under field conditions. Therefore, genotypes identified as highly resistant and resistant need to be confirmed for their reaction to ChLCV infection through phenotypic assay following artificial / challenge inoculation as well as molecular assays following PCR using ChLCV strain specific PCR primers. Such assays help in identification of true disease response of a genotype.

The consequences of identification of contrasts including highly resistant sources (S343, Bhut Jolokia) and highly susceptible genotypes (Byadgi Kaddi, Byadgi Dabbi) and their involvement in breeding activities in generating segregating material will enable deciphering the inheritance of response to ChLCV infection form a wide spectrum or combination of crosses with different genetic backgrounds (R X R, R X S, S X R, S X S). These activities are also expected to enable a breeder to develop hybrids with high level of resistance if parents are complementing for resistance. Further, resistance can be introgressed into an otherwise elite

The Mysore Journal of Agricultural Sciences

horticultural background employing marker assisted back cross approach if resistance is governed by oligo genes.

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