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Genetic analysis for resistance to leaf curl disease in Chilli Peppers (*Capsicum annuum* L.) under specific situations

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Abstract

Leaf curl is a serious viral disease of chilli caused by a group of bigomoviruses dominated by chilli leaf curl virus (ChiLcv). With the aim to study the mode of inheritance of the ChiLcv disease resistance, a resistant genotype DLS-Sel.10 was crossed with a susceptible genotype Phule Mukta and F_1, F_2, B_1 and B_2 generations were developed. The parents along with the segregating generations were screened under natural conditions as well as challenged inoculation with viruliferous whiteflies carrying predominant ChiLcv.PCR amplification of viral genome-specific marker confirmed the presence of virus in all the tested plants however, only susceptible plants produced symptoms. The F1 plants showed susceptibility under both natural and challenged inoculation conditions indicating that the resistance is of recessive nature. On Chi-square test, it was found that susceptible and resistant plants of the two F_2populations segregated in a ratio of 3 susceptible : 1 resistant plants and the B_2 population derived from DLS-Sel.10/Phule Mukta//DLS-Sel.10 segregated in a ratio of 1 resistant : 1 susceptible plant suggesting that the Chilcv is goverened by a single recessive gene. The findings of this study will help in breeding for ChiLcv resistance in Chilli.

Key words: Chilli leaf curl virus (ChiLCV), begomoviruses, inheritance, challenge inoculation, natural screening

Introduction

Chilli (*Capsicum annuum* var. *annuum* L.), a member of Solanaceae family, is one of the major cash crops and an essential condiment for India. Its cultivation has spread over to several tropical and sub-tropical countries like Japan, Mexico, Turkey, United States of America, African countries apart from India. India is one of the leading chilli producing and exporting countries in the world. However, chilli production is affected by many biotic stresses and among the viral diseases, it has been reported to be attacked by more than 65 viruses (Nigam et al. 2015). Leaf curl disease has emerged as a serious problem in the chilli growing areas of India causing 100% crop loss during *kharif* (Senanayake et al. 2006). *Kharif* season in India starts with the onset of monsoon from June to September. In Delhi region of the country where the experiment was conducted, the disease generally appears in the end of June about 45-55 days after sowing and spreads rapidly in July. The disease progress becomes slow in August and almost comes to a halt by mid October. Initiation of leaf curl virus is characterized by symptoms like vein thickening on young upper leaves of plants followed by upward/downward leaf curling and leaf thickening. Symptoms start appearing after 10-18 days of infection.

There are several reports of chilli leaf curl disease in India (Mishra et al. 1963; Dhanraj et al. 1970; Senanayake et al. 2007) and is caused by begomoviruses (DNA virus) belonging to family *Geminiviride*. Maximum molecular diversity of geminiviruses and their associated satellite components exist in Southeast Asia (Nawaz-UI-Rehman and Fauquet 2009; Akhter et al. 2013). In India, *Chilli Leaf Curl Virus* (ChiLCV), *Chilli Leaf Curl India Virus* (ChiLCINV), *Chilli leaf curl Vellanad virus* (ChiLCVV), T*omato leaf curl Joydebpur virus* (ToLCJV), *Tomato leaf curl Bangalore virus* (ToLCBaV), *Tomato leaf curl Palampur virus* (ToLCPalV) and *Tomato leaf curl New Delhi virus* (ToLCNDV) are known to be

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associated with chilli leaf curl disease (Khan et al. 2006; Kumar et al. 2011, 2012; Senanayake et al. 2007; Shih et al. 2007). ChiLCV occurring in India has been shown to be a monopartite begomovirus along with DNA beta satellite (Chattopadhyay et al. 2008). The full length genome of ChiLCV consists of a single genomic component equivalent to DNA A that carries all the genes responsible for the successful establishment of the virus in the host (Stanley et al. 2005). However, chillies with severe symptoms are associated with a bipartite begomovirus and a betasatellite (Akhter et al. 2009). Bipartite begomoviruses consist of two genomic components called DNA A and DNA B. Two betasatellites namely, *Tomato leaf curl Bangaladesh betasatellites* (ToLCBDB) and *Chilli leaf curl betasatellite* (ChiLCB) have been found to be associated with chilli leaf curl disease in India (Kumar et al. 2015).

Several factors such as the availability of large population of insect vector, wide host range, emergence of new viral strains/species due to recombination and dissemination of begomoviruses have contributed to their spread and losses caused by these viruses (Varma and Malathi 2003). The management of leaf curl disease in susceptible chilli cultivars is very difficult. For field production, a combination of rotation of insecticides to control the vector and cultural practices to reduce virus reservoirs as well as whitefly populations has been the most commonly followed approach to manage the disease. But these strategies do not provide a safe and sustainable solution to control the disease. Resistance breeding is the only ecologically as well as economically sound approach to reduce disease incidence. An important component of disease resistance breeding includes understanding the genetics of resistance which ultimately decides the choice of suitable breeding methods for further disease resistance program.

We have identified three lines namely DLS-Sel-10, WBC-Sel-5 and PBC-142 as resistant sources to leaf curl disease at our institute through four seasons of natural screening (Srivastava et al. 2017) and the resistance in one of the lines, DLS-Sel-10 has also been confirmed through challenge inoculation (unpublished). There are other reports of resistant sources from different parts of India like BS-35, GKC-29 and Bhut Jhalokia from IIVR, Varanasi (Rai et al. 2014), Saurian 2010, Perennial and Japani Loungi (Ahmad et al. 2016) and S-343, SL 475 and SL 476 (Jindal, 2014; Thakur et al. 2017). Efforts to study genetics of resistance have been made in the past (Bal et al. 1995; Kumar et al. 2009; Anandhi and Khader, 2011) but all these studies were done under natural field conditions which cannot rule out the chance of any escapes during data recording. With advancement in screening technologies, artificial screening methodologies have been used to study genetics of resistance to leaf curl which has been found to be monogenic recessive (Rai et al. 2014; Mathur et al. 2019) as well as monogenic dominant (Thakur et al. 2019). This gives an indication that resistance genetics basically depends on the resistance source that is being used in improvement program. *In situ* efforts to compare the genes conferring resistance in tomato to leaf curl with genes in chilli have also been made (Mangal et al. 2017) but it does not provide much information on genetics of resistance. With the background of leaf curl disease being a complex disease caused by a group of viruses, the present study was therefore undertaken to understand the genetics of resistant gene from our resistance source DLS-Sel-10 to begomovirus complex as well as the pre dominant virus in this complex. This study will eventually help in breeding leaf curl resistant lines and highlights the importance of virus specific resistance breeding.

Materials and methods

Population development for inheritance studies

The resistant source DLS-Sel-10 (P_2) has previously been screened under natural epiphytotic condition against chilli leaf curl disease for four consecutive seasons and the line exhibited consistent resistance in all the four seasons of evaluation (Srivastava et al. 2017). DLS-Sel-10 along with a susceptible genotype Phule Mukta (PM: P_1) were crossed to generate F_1s in the present study with an aim to study the inheritance leaf curl disease. The F_1 seeds obtained were used to raise plants and these plants were selfed by wrapping the flowers with cotton wad and mature seeds from self-fruits were collected and sown to get F_2 generation. F_1 plants were also backcrossed with PM and DLS-Sel-10 to generate BC_1P_1 and BC_1P_2 populations, respectively. With the seeds of parents, F_1, F_2, BC_1P_1 and BC_1P_2 in hand, these were screened for leaf curl disease.

Identification of predominant virus in the field

As emphasised earlier leaf curl disease in chilli is said to be a complex disease caused due to infestation by a group of viruses. Challenge inoculation against all

these viruses is quite difficult so we made an attempt to identify the pre-dominant virus causing leaf curl at our station. Breeding lines grown for evaluation during *kharif*, gets infected with leaf curl disease due to prevalence of the disease during this period. Twenty four different breeding lines heavily infested with leaf curl were selected and one plant from each genotype were chosen to collect leaf samples. These samples were tested for the presence of common begomo viruses causing leaf curl in chilli using virus specific primers which have been designed for each of these viruses (sequences have been submitted for patent). The samples were tested for ChiLCV, ToLCNDV, ToLCJV, ToLCBaV and ToLCPaLV to identify the most common virus among these.

Screening under natural epiphytotic conditions

One set of parents, F_1 and F_2 were screened for leaf curl disease through natural infestation under field condition. F_2 population (132 plants) along with parents (20 plants each) and F_1 (15 plants) were raised in a net house during *kharif*, 2016. The net house was left open for a period of about three weeks and when some of the plants started showing symptoms, the net was closed expecting that enough whitefly population might have built up inside the net. Closed net allowed the whiteflies to multiply inside resulting enough population to build up which ensured that none of the plants being screened for leaf curl disease resistance could escape the infestation by whitefly.

Screening through challenge Inoculation

One hundred and sixteen healthy F_2 transplants of chilli along with F_1 and parental genotypes (DLS-Sel-10 and PM) were raised in plastic pots of four inch size. Similarly 84 BC_1P_1 individuals and 78 BC_1P_2 individuals were also raised. Whitefly colonies were reared and maintained on eggplants under controlled conditions in insect rearing cages. A temperature of 28-35°C, 30-50% relative humidity and 14 hr photoperiod was maintained which yielded optimal whitefly population. For screening, these healthy whiteflies were converted to viruliferous whiteflies by allowing them to feed on chilli plants infected with ChiLCV. After 24 hours of feeding (acquisition access period) on the infected chilli plants, whiteflies were considered to be viruliferous and were used for challenge inoculation of the healthy seedlings (of about 35 days) of test genotypes. Each individual plant was covered with plastic bottles with a height of 15 cm and diameter of 9 cm and their top portion was removed with the help of a hot iron solder. Black muslin cloth was fixed on the removed top portion which helped to avoid accumulation of excess moisture inside the cage as well as escape of whiteflies. A small hole was made at the side of the bottle to release the viruliferous whiteflies and the hole was then plugged with cotton to prevent whitefly escape. For screening, individual plant inoculation was done and five viruliferous whiteflies were released per plant. Inoculated plants were sprayed with Dimethoate (2 ml/l) after 15 days of inoculation to kill the whiteflies. The inoculated plants were kept in the glasshouse for symptom expression and symptom severity was scored on individual plants according to scale mentioned in Table 1 (Kumar et al. 2006). The disease scoring was recorded from 7 days after inoculation till 28 days at weekly intervals. All the plants with disease score of 0, 1 and 2 were categorized as resistant while those with score of 3, 4 and 5 were grouped as susceptible.

Detection of viruses in plants subjected to challenge inoculation

DNA was extracted from all the test plants subjected to challenge inoculation with ChiLCV. This included the resistant parent DLS-Sel-10, susceptible parent (PM), F_1, F_2, BC_1P_1 and BC_1P_2-individuals. Leaves were collected from the plants, 28 days after

Table 1. Infection type classification given by Banerjee and Kalloo, 1987 and modified by Kumar et al. 2006)

Class	Grade	Description of symptoms
Immune	0	No symptom
Highly resistant	1	0 to 5% curling and clearing of upper leaves
Resistant	2	6 to 25 curling, clearing of leaves and swelling of veins
Moderately susceptible	3	26 to 50% curling, puckering and yellowing of leaves and swelling of veins
Susceptible	4	51 to 75% leaf curling and stunted plant growth and blistering of internodes
Highly susceptible	5	More than 75% curling and deformed small leaves, stunted plant growth with small flowers and no or small fruit set

inoculation, when all the plants had given their response after infestation with ChiLCV. DNA extracted from different plants was used to test for the presence of ChiLCV using the virus specific primers designed and which do not show cross amplification with other begomoviruses (unpublished data). The 15 μl PCR reaction mixture consisted of 1 μl DNA (50 ng/μl), 1.0 U *Taq* DNA polymerase (Hi media Laboratories, Mumbai, India), 0.6 uL of 10 mM dNTP mix (Hi media Laboratories, Mumbai, India), 0.6 μl (20 pM) of forward and reverse primers each and 1.5 uL of 10 × PCR buffer having 17.5 mM $MgCl_2$ (Hi media Laboratories, Mumbai, India). Amplification conditions used were, one cycle of 94°C for 3 min; 10 cycles of 94°C for 0.5 min, 65-55°C decreasing by 1°C per cycle for 1 min, and 72°C for 1 min; 30 cycles of 94°C for 0.5 min, 55°C for 1 min, and 72°C for 1 min; and a final cycle of 72°C for 5 min. Amplified products were resolved on 1.5% agarose gels with Tris/Acetate/EDTA (TAE) stained with ethidium bromide, at a constant voltage of 60 V for 3 h using a horizontal gel electrophoresis system (BioRad, USA) and visualized and photographed under UV light in a gel documentation unit (Alpha imager, Cell biosciences, Santa Clara, CA).

Statistical analysis

F_1, F_2, BC_1P_1 and BC_1P_2 generations of the cross between PM × DLS-Sel-10 were recorded for their expression for leaf curl disease. To estimate the number of genes conditioning expression of resistance to leaf curl disease in this population, a Chi-square test was done (Babiker et al. 2009). A Chi-square test for goodness-of-fit was tested with the hypothesis of monogenic control of resistance to leaf curl disease.

Results

Identification of predominant virus in the field

Out of the twenty four infested samples tested for detection of pre dominant viruses, ChiLCV and ToLCNDV was detected as the major viruses infecting chilli at our experimental station (Figs. 1 and 2). The PCR method resulted in specific detection of each of the begomovirus without any cross amplification. Out of 24 samples, 23 samples showed presence of 453 bp fragment corresponding to ChiLCV (Fig. 1) whereas 11 out of 23 samples amplified a fragment of 702 bp corresponding to ToLCNDV (Fig. 2).

Natural epiphytotic screening

Twenty eight days after closing of the net, out of the 20 resistant and 20 susceptible plants subjected to

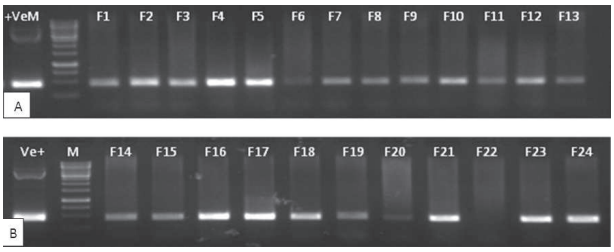

Fig. 1. (A and B): Detection of chilli leaf curl virus (ChiLCV) with the specific Primer (BM861 and BM862) gave amplification of 453 bp fragment in most of the symptomatic samples (M: 1 kb ladder)

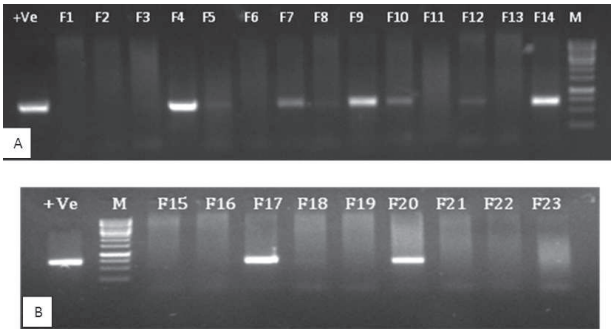

Fig. 2. (A and B): ToLCNDV specific primer BM794 and BM795 gave 702 bp fragments in a few samples (M: 1 kb ladder)

natural epiphytotic screening, 19 plants were found to be resistant and one susceptible of DLS-Sel-10 while all the 20 plants of PM were found susceptible. Similarly, all the fifteen plants of F_1 hybrid were susceptible while the population of 132 F_2 plants segregated into 27 resistant plants and 105 susceptible plants (Table 2).

Screening after challenge inoculation

Under challenge inoculation, all the plants of DLS-Sel-10 showed resistance response while all plants of PM showed susceptible response. F_1 individuals were found to be susceptible and the population of 116 F_2 plants segregated into 26 resistant and 90 susceptible plants (Table 2). BC_1P_1 population of 84 plants was all susceptible while 78 plants BC_1P_2 segregated into 34 resistant plants and 44 susceptible plants.

Detection of ChiLCV

The ChiLCV showed amplification in all the plants subjected to challenge inoculation. This included both the resistant individuals as well as plant showing susceptibility. Resistant parent (P_2) showed a faint band while susceptible parent (P_1) and F_1 showed an intense band. On testing the F_2 and backcross

Table 2. Number of F_2 individuals of PM x DEL-Sel-10 cross and leaf curl disease score range of R, MR, MS and S

Population	Natural Screening (NS)		Challenge Inoculation (CI)		Expected ratio (R:S)	χ^2 Value(cal)		χ^2 Value (Tab)
	Number of plants		Number of plants			NS	CI	
	Resistant	Susceptible	Resistant	Susceptible				
DLS-Sel-10	19	1	15	0				
Phule Mukta	0	20	0	15				
F_1	0	15	0	15				
F_2	27	105	26	90	1:3	1.43	0.41	3.84
BC_1P_1	-	-	0	84	0:All	-	0	3.84
BC_1P_2	-	-	34	44	1:1	-	1.28	3.84

individuals for presence of ChiLCV, different plants showed different intensity of band. Plants with intense band were found to show susceptible response while those with fainter bands had resistance response (Fig. 3). The same has been confirmed through quantitative

Fig. 3: Detection of ChiLCV in P_1 (Phule Mukta), P_2 (DLS-Sel-10), F_1 and part of F_2 individuals screened through challenge inoculation along with their phenotypic response, M: Marker Lane

PCR where the resistant genotype (DLS-Sel-10) had significantly lower viral load when compared with PM (under communication).

Genetics of resistance

F_1 individuals were found to be susceptible, both under natural screening as well as challenge inoculation, indicating the resistant gene to be recessive in nature. A Chi-square test for goodness of ût was tested with the hypothesis of monogenic control of resistance to ChiLCV. The genetic model was considered to be appropriate for a probability (P) value >0.05. The calculated value of χ^2 was 1.453 for F_2 under natural epiphytotic condition and 0.41 under challenge inoculation. For BC_1P_1, observed χ^2 value was 0 while it was 1.28 for BC_1P_2. These values were smaller than the tabulated χ^2 value 3.84. Non-significance of calculated χ^2 values indicate that the observed and expected frequencies are in close agreement.

Discussion

Study conducted to identify the pre-dominant viruses causing leaf curl disease at our experimental station indicated that ChiLCV is the most common virus causing leaf curl in chilli along with occurrence of mixed infection with ToLCNDV in some of the samples. The results are in line with several earlier reports which indicate similar results wherein chilli plants showing leaf curl symptoms have been found to harbor mixed infection with different begomoviruses (Khan et al. 2006; Senanayake et al. 2007; Shih et al. 2007; Rai et al. 2014; Thakur et al. 2019). Studies of Kumar et al. 2015 have identified ChiLCV as the most widely distributed begomovirus associated with leaf curl disease in chilli across India, followed by PepLCBV and ToLCNDV. Occurrence of different begomoviruses causing leaf disease in chilli in different parts of the country necessitates the importance of virus specific breeding. Resistance breeding against viruses should start with identification of pre-dominant viruses causing disease in a particular region followed by identification of resistant sources to these viruses and then the genetic study of resistance against these viruses. As ChiLCV has been identified as pre-dominant virus of our location, it can be concluded that DLS-Sel-10 exhibits resistance to this virus.

Virus detection in challenge inoculated plants showed presence of virus in all the plants irrespective

of the plant response to the disease. This indicated successful inoculation using viruliferous whiteflies and confirmed no escapes in challenge inoculation studies. But resistant plants from resistant source (DLS-Sel-10), F_2 population and backcross population showed presence of virus in spite of resistant response. This highlights that the resistant source we used is a symptomless carrier of ChiLCV. Similar results indicating resistant sources to be symptomless carrier has also been reported in tomato (Friedmann et al. 1998) and chilli (Thakur et al. 2019). Viral copy in resistant source is much less in comparison to susceptible genotypes (Legarrea et al. 2015). This is supported by the appearance of low intensity bands of viral amplicons during PCR detection of ChiLCV. The resistant source acquires the virus but hampers its further multiplication to allow manifestation of resistant phenotype (Gowda et al. 2003).

To understand the nature and the number of gene (s) governing resistance to leaf curl disease in chilli, F_1, F_2, BC_1P_1 and BC_1P_2 from the cross of PM (as female parent) and DLS-Sel-10 (as pollen parent) were evaluated for resistance to leaf curl disease under natural epiphytotic conditions as well as challenge inoculation with ChiLCV. DLS-Sel-10 was found to show resistance both under natural as well as challenge inoculation except for one plant under natural screening. Natural screening was done with whiteflies present naturally which can carry different complex viruses present in nature. This single plant DLS-Sel-10 might have been infected with a whitefly carrying a virus to which DLS-Sel-10 was susceptible or infection with multiple viruses might have resulted susceptibility in the resistant source or a resistance breaking strain ChiLCV might have evolved (Mansoor et al. 2003). Challenge inoculation confirmed that DLS-Sel-10 was resistant to ChiLCV, the pre-dominant virus of our region. PM exhibited susceptibility under both natural and challenge inoculation. Similarly all plants of F_1 hybrid developed from the cross between PM × DLS-Sel-10 were found to be susceptible under both natural and artificial screening, suggesting thereby the gene controlling resistance is recessive in nature. Recessive nature of resistance is again supported by the fact that F_2 population segregated into resistant and susceptible individuals under both natural and challenge inoculation where the susceptible individuals were again more than the resistant ones. On assuming monogenic nature of the resistance genes, the resistant and susceptible individuals in F_2 population should segregate in the ratio of 1:3 and this is what we observed under both natural and challenge inoculation. From the study of BC_1P_1 population developed from backcrossing with susceptible parent, it was observed that all the plants of the population showed complete susceptibility. Backcross population with the resistant parent (BC_1P_2) segregated into resistant and susceptible individuals in the ratio of 1:1. The segregation pattern of backcross population confirmed the monogenic recessive resistance of DLS-Sel-10. Kumar et al. (2009) have also reported the genes to be recessive in nature under both field and artificial screening. Recessive resistance to virus has been reported in other crops like mungbean (Solanki et al. 1982; Ammavasai et al. 2004). An earlier study on inheritance of resistance to PepLCV (Rai et al. 2014) in an inter-specific cross of PBC-535 (*C. annuum*) × Bhut Jolokia (*C. chinense*) have also revealed monogenic recessive nature of resistance. In our previous study with WBC-Sel-5 as resistant source (Mathur et al. 2019), we have reported resistant gene to leaf curl again to be monogenic and recessive in nature. In contrast to this, there is recent report that resistant gene from the resistant source S-343 to leaf curl in chilli is monogenic dominant in nature (Thakur et al. 2019). Study on genetics of resistance from Bhut Jhalokia was against PepLCV (Rai et al. 2014) and from S-343 was against ToLCJV (Thakur et al. 2019). Different nature of genetics of resistance may be due to different resistant sources studied as well as the virus against which resistance is being studied.

Authors' contribution

Conceptualization of research (AS, MM); Designing of the experiments (AS, MM, PK, AT, AK); Contribution of experimental materials (AS, BM); Execution of field/ lab experiments and data collection (PKM, VS); Analysis of data and interpretation (PKM); Preparation of the manuscript (PKM, MM).

Declaration

The authors declare no conflict of interest.

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