International Journal of Plant & Soil Science

Volume 35, Issue 24, Page 253-260, 2023; Article no.IJPSS.111195
ISSN: 2320-7035

The Cucumber Mosaic Virus: A Review

Mukta Joshi [a*], T. K. Narute [a++] and Dhanashree Sarnobat [a#]

[a] Department of Plant Pathology and Agricultural Microbiology, MPKV, Rahuri, Maharashtra, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJPSS/2023/v35i244316

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/111195

Review Article

Received: 20/10/2023
Accepted: 25/12/2023
Published: 30/12/2023

ABSTRACT

Cucumber mosaic virus is a member of genus Cucumovirus from the Brommoviridae family known to have a very wide host range and is one of the most prevalent viruses in the globe. The research work of cucumber mosaic virus strains responsible for various diseases in vegetable and pulse crops; ornamentals, medicinal and aromatic plants and weeds reported in world from last 71 years (1951–2022) have been reviewed in this article. It includes historical background, taxonomy and classification, Geographical distribution, Host range and virus propagation, Virus Structure and Genome organization, Replication, Movement, survey, symptomatology, Insect transmission, Evolution and Management. The mode of spread of the diseases in nature through insect vectors, search of alternate hosts/reservoirs and diagnostics methods for sensitive detection of the viruses at early stages of infection in plants and in propagating materials are important factors to study virus and disease epidemiology. These reviews summarised in this article focused on the epidemic nature of CMV, their capability to infect a variety of economically important plants and proper identification and designing the effective management strategies which would help to strengthen the understanding of the researchers and students in particular discipline help to design the management strategies.

Keywords: Cucumovirus; Brommoviridae; taxonomy; evolution; epidemiology; genome; replication.

[++] *Ex. Head;*
[#] *Assistant Professor;*
**Corresponding author: E-mail: Joshimukta46@gmail.com;*

1. INTRODUCTION

"It can be challenging to identify viral diseases in the field that are caused by CMV based on symptoms at certain stages of disease development. In some circumstances, the relevance of symptoms is limited in determining the viral agent responsible for the disease. The identification of viruses using molecular and serological techniques will be more accurate, trustworthy, time- and cost-efficient and more accurate. Due to its quick spread by more than 60 aphid species in the field, CMV is devastating and results in significant losses in fruits, vegetables and ornamentals" [1]. "In some crop and weed hosts, transmission through planting material is also important" [2].

The CMV disease has a huge impact on the industrial scale of production of different crop. it can also devastate subsistence farmers who depend on their crops to feed their families and provide income. Once established it is very difficult to eradicate and manage the disease. the disease is more severe in the areas where monocrop is grown. The disease is vector transmitted and CMV can transmitted by more than 60 species of aphid species. Gallitelli [3] concluded "after several studies that cucumber mosaic virus can be transmitted by several aphid species and is known to infect more than 1000 species of different plants in 365 genera of 85 families. Such a wide host range may provide viral inoculum throughout the year for infection. In many crops, no resistant variety developed or is commercially available yet and the control measures used are quite demanding and less effective as CMV has a wide host range and many aphid species can efficiently transmit the CMV virus".

2. HISTORY

Cucumber mosaic virus (CMV) was first described as a disease of cucurbits by Doolittle [4] in Michigan and Jagger [5] in New York. "In early 1940s CMV was reported in Maharashtra and later from many parts of the country" by Kamat and Patel [6]; Rao [7]. The occurrence of CMV was reported first time on chilli in India by Bhargava [8,9]. The virus can infect a large number of indicator plant species and has been isolated from over 500 naturally infected species. Cross-protection was used in the 1930s to discriminate isolates of CMV with differences in phenotypes or host range (strains). CMV was not purified reliably until the middle 1960s. Later serology and hybridization technology were used to detect and differentiate two major subgroups of CMV. The nucleotide sequence and the genome organization of one strain of each CMV subgroup were determined between 1984 and 1990, while biologically active cDNA clones of several CMV strains were developed in the early 1990s. The major functions of each of the five encoded proteins have been assigned, although each protein is also involved in other host–virus relationships.

3. TAXONOMY AND CLASSIFICATION

"Isolates of CMV are differ in symptoms, host range, transmission, serology, physicochemical properties, and nucleotide sequence of the genomic RNAs. CMV isolates can be classified into two major subgroups (subgroup I and subgroup II) on the basis of serological typing, peptide mapping of the coat protein, sequence similarity of their genomic RNA. The 69% to 77% identity was observed in the nucleotide sequence between pairs of isolates belonging to each of these subgroups, depending on the RNA segment compared and the pair of isolates, dissimilarity being highest for RNA2. Nucleotide sequence identity among isolates within a subgroup is above 88% for subgroup I and above 96% for subgroup II, indicating a higher heterogeneity of subgroup I. The biological, serological and nucleotide sequence homology of numerous CMV strains have been documented earlier and divided it into two subgroups I and II" [10,11]. "Subgroup I have been further divided into IA and IB based on 5′ non-translated region of RNA 3 and CP gene" [12]. "Most of the Indian CMV strains have been clustered in IB subgroup" [13]. Identification of the viruses by molecular and serological methods will be of more accurate, reliable, less time consuming and also cost effective.

4. GEOGRAPHIC DISTRIBUTION

"CMV is one of the common viral diseases have worldwide occurrence, reported to infect several crop and weeds species in both temperate and tropical regions. Subgroup I most prevalent as most of the isolates belong to subgroup I. Subgroup II isolates are found more frequently in cooler areas or seasons of temperate regions. This has been associated with lower temperature optima for planta virus accumulation shown for the few isolates characterized for this property. Most isolates in subgroup IB have been reported from East Asia, which is presumed to be the

origin of this subgroup. CMV sub group I was found to be predominant to Costa Rica. CMV subgroup II was detected in the Atlantic region Subgroup IB isolates also have been reported from other areas, for example, the Mediterranean region, California, Brazil, and Australia. Those in the Mediterranean could have been introduced recently from East Asia" [14,15] identified "a CMV-banana isolate as a member of CMV subgroup IB by sequence analysis of three RNA genomes". "A CMV strain infecting Gladiolus was also identified as a member of subgroup IA based on distinct phylogenetic relationships with Indian isolate of IB" [16]. "The identification of CMV subgroup II isolate causing severe mosaic in cucumber" was reported by Kumari et al. [17] based on its complete genome for the first time in India. Deloko et al. [18] reported "the occurrence of CMV on tomato and pepper,showed that CMV infects tomato and pepper in the West Region of Cameroon to a varying extent"

5. HOST RANGE AND PROPAGATION

"The CMV has a very wide host range as it is known to infect the majority of the horticultural as well as weed species. The host range of the collective isolates of CMV is over 1300 species in more than 500 genera of over 100 families, with new hosts reported each year [14] and which has the widest host range of any known virus" [19]. Due to high rate of mutation some newly described strains have lost their ability to infect many typical hosts while infecting new hosts of CMV. It could be a strategy to overcome the constraints of infection and adaption to new hosts. CMV able to infect the majority of horticultural crops (vegetables, fruits and ornamental crop) and also different weed species. These weed species may act as reservoirs of the virus as well as provide shelter for insect vector. The different indicator plant used to study the reaction of CMV infection, include cucumber (*Cucumis sativus*), tomato (*Solanum lycopersicum*), and tobacco (*Nicotiana tabacum*), all systemic hosts of CMV, as well as cowpea (*Vigna unguiculata*) and *Chenopodium quinoa*, symptoms were observed to limited to an inoculated leaf, whereas some legumes strains reported to infect systemically. Most of the isolates of CMV were reported to propagate in many hosts like squash (marrow) (*Cucurbita pepo*), tobacco*, N. clevelandii*, or *N. glutinosa, Musa* spp. In India, the occurrence of CMV has been noted in a number of hosts comprising Egyptian henbane [20]; Gladiolus [21], Brinjal [22], Sarpagandha and Jatropha [23,24], banana [25].

6. VIRUS STRUCTURE AND GENOME ORGANIZATION

CMV is known to infect various crop species and weed species. So, particle size of CMV also varies from crop to crop. According to report on CMV of different crop, CMV has an isometric cored particle size ranges from 28nm in capsicum to 35 nm in prunus. CMV is a multicomponent single stranded virus having three positive-sense RNAs (RNA1, RNA 2 and RNA 3), as well as a fourth sub genomic RNA (RNA 4) produced from RNA 3 [26,27]. By contrast, RNA 3a encodes for 3a movement protein (MP) [28], RNA 4 encodes for 3b coat protein (CP) [26,29] and RNA 1 and RNA 2 encode for 1a and 2a proteins involved in virus replication [30]. "Contain about 18% RNA, its total genome size was 8.621 kb and was broken into three parts. The largest part was 3.389 kb; the second was 3.035 kb; and the third was 2.197 kb. The RNA was found surrounded by a protein coat consisting of 32 copies of a single structural protein which forms isometric particles. Completely sequenced genome of a CMV isolate causing severe symptoms of mosaic in cucumber and its phylogenetic analysis with other 21 CMV isolates grouped it into subgroup II strains. The genome consists of RNA 1 (3,379 nucleotides), RNA 2 (3,038 nucleotides) and RNA 3 (2,206 nucleotides) and also reported that RNA1 and RNA2 were closely related to the Japanese isolate while RNA3 clustered with an American isolate" [17].

The 111 kDa 1a protein is encoded by RNA1, and the 13–15 kDa 2b protein, which is translated from subgenomic RNA 4A that is co-terminal with RNA2's 3'end, is encoded by RNA2. On the other hand, RNA1 encodes the 98 kDa 2a protein. Although in an a+1 reading frame, the ORFs expressing the 2b and 2a proteins overlap. The 25 kDa 3b protein, which is expressed from an RNA4 that is co-terminal with the 3' end of RNA3, and the 30 kDa 3a protein are both encoded by RNA3. All three genomic and subgenomic RNAs have highly conserved 224–338 nt 3' nontranslated regions that create several pseudoknots and a tRNA-like structure. Compared to RNA3, the 5' nontranslated sections of RNA1 and RNA2 are more conserved in sequence with one another. In addition, CMV generates an unidentified function RNA5 that co-terminates with the 3' nontranslated sections of

RNA1 and RNA2. Only class II strains are encapsidated for RNA4A and RNA5. Low amounts of tRNAs were also encapsulated by CMV particles; they have been identified in the literature as CMV RNA6 [14] Certain CMV strains have the ability to encapsidate faulty RNAs generated from CMV RNA3, however this is rarely documented.

7. REPLICATION

"The site for the replication of CMV is cytoplasm. The CMV mostly replicates in vacuole membrane or tonoplast. The RNA1 and RNA2 encode proteins 1a and 2a proteins respectively, as well as possibly a few host proteins, which are involved in genome replication and internal transcription of sgRNA4 from the minus-strand copy of RNA3 together with the purified CMV replicase also includes a 50 kDa host protein whose purpose is unknown. The putative N-terminal proximal methyltransferase domain of the CMV 1a protein is believed to be important in capping the RNAs, while the putative C-terminal proximal helicase domain is required for the unwinding of the viral RNAs during replication. It has been discovered that the 1a protein can attach to a number of tonoplast intrinsic proteins; however, it is unknown what functions these play in the replication of the virus. Both in vivo and in vitro, interactions occur between the N-terminal region of the 2a protein and the C-terminal region of the 1a protein. The 2a protein's phosphorylation stops it from interacting with the 1a protein. The conserved domains present in RNA are present in the C-terminal portion of the 2a protein. polymerases, and as a result, they constitute the core of the CMV replicase along with the 1a protein. The tonoplast-associated replicase binds to the tRNA-like structure and different pseudoknots found in the 30 non translated regions of the positive-sense CMV RNAs to start CMV replication. Subsequently, each genomic RNA is converted into minus-sense RNA, which serves as a template for the synthesis of fresh plus-stranded genomic RNAs. By recognising the subgenomic promoters found on the minus-sense RNAs, the minus-sense RNA2 and RNA3 also function as templates for the production of the two plus-sense subgenomic RNAs (4 and 4A). The CMV replicase replicates satellite RNAs and faulty RNAs, but not the subgenomic RNAs themselves. The CMV replicase replicates satellite RNAs and faulty RNAs, but not the subgenomic RNAs themselves. Diverse host species have shown variations in the relative levels of accumulation of the distinct CMV genomic RNAs and satellite RNAs, most likely as a result of host-specific variations in template copying" [14].

8. MOVEMENT

"Cell to cell movement is most important step of infection in the life cycle of virus. Short distance cell to cell movement of the virus happen through plasmodesmata" [31,32], Robard and Lucas 1990). "The virus genome encodes an essential protein for the infection called movement protein" [33,34]. "Based on the sequence homology of viral protein with 30kDa MP of TMV, 3a viral protein acts as movement protein. As of yet, no particular plant protein has been shown to be involved in the mobility of cells among themselves. It appears that CMV travels both within and between epidermal cells, passing through mesophyll cells and onto vascular cells. Because the virus preferentially travels to and between mesophyll cells in the absence of the 2b protein, the 2b protein also affects the course of virus migration. All of these cell types allow the virus to reproduce, but the sieve components of the vasculature do not. Virion assembly may occur within sieve elements as a result of capsid protein and RNAs migrating from nearby vascular cells. In certain species, but not all of them, virion assembly is required for systemic infection" [14].

9. SYMPTOMATOLOGY

"CMV exhibits complex symptoms *viz*., mosaic, leaf distortion, mottle, veinal chlorosis and stunting causing a considerable loss in plant vigour and yield" [35]. "The variety, plant age during infection, temperature and viral strain can all affect the symptoms. leaf chlorosis is one of the typical signs, which can progress to seriously blight the growing point and ultimately result in plant mortality. The presence of constriction, wrinkling with vein distortion and inward leaf roll are further symptoms of chlorotic mottle in leaves" [36]. "Cucumber mosaic virus (CMV) isolate, causing leaf mosaic and distortion, malformed flowers or colour breaking on the petals of *Catharanthus roseus* in Serdang, Selangor, Malaysia, was as Malaysian periwinkle isolate (CMV-MP)" [37]. "CMV isolate showed severe mosaic symptoms on *Nicotiana* spp. and *Cucumis* spp. The isolate induced leaf deformation and mild filiform type symptoms in tomato" [17]. "CMV at the early stage was severely stunted in growth, leaves were distorted and fruits were unsalable because of obvious

crease". Agrios [38]; Zitikaite et al. [39]. Tripathi et al. [40] observed "the virus causes variable symptoms from mild chlorosis to severe chlorotic streaks on leaf lamina depending on the pathogen strain and the weather conditions. They explained symptoms with more pronounced which included necrosis of emerging leaves and internal tissues of pseudo stem when banana plants are infected with severe strains of the virus. Fruits showed mosaic symptoms and bunches bear malformed fruit or no fruit. Plants died in case of very severe infection especially when plants get infected soon after planting". Vinodhini et al. [41] collected "infected plants showing mosaic with chlorotic and necrotic rings, veinal necrosis, mosaic mottling, leaf filiformity and malformation and concluded the natural co-existence of chlorosis inducing CMV strain with CaCV and GBNV on hot pepper in India". Diningsih et al. [42] collected of "a symptomatic indicator plant (*Solanum lycopersicum*) following the inoculation with the mosaic virus from the Impatiens plant (previous research findings). The results of this study indicated that, CMV was amplified successfully from the test plants".

10. TRANSMISSION

Many plant species have reported cases of CMV seed transmission, with efficiency ranging from less than 1% to 50%. In addition to pollen, viruses can be found in the embryo, endosperm, and seminal integuments. Seed transmission efficiency is affected by RNA1 and potentially protein 1a. Aphids act as a nonpersistent vector for the horizontal transmission of CMV. There are around 80 species of aphids known to spread CMV; the two most effective and well-researched vectors are *Aphis gossypii* and *Myzus persicae.* The precise mix of viral isolate and aphid species, as well as the buildup of particles in the source leaf, determine the transmission effectiveness. Changes in plant volatile profiles can alter the vector behaviour. For example, the cucumber mosaic virus (CMV) caused dynamic volatile alterations in its natural squash host, resulting in more attractiveness of host to *Aphis gossypii* [43]. CMV infection also resulted in enhanced attraction of its aphid vector *M. persicae* towards host *Arabidopsis thaliana*. The viral suppressor of host RNAi expressing 2b protein in transgenic *A. thaliana* can induce odour dependant aphid attraction [44].

"Non-persistent plant viruses can also affect plant defence responses to change the feeding behaviour, preference, or fitness of insect vectors. For instance, CMV infected tobacco plants produced increased reactive oxygen species (ROS) and initiated defensive signalling in their tissues, altering aphid feeding behaviour and increasing virus retention and transmission" [45]. "CMV infection also rendered plants (*Nicotiana benthamiana* and *Arabidopsis thaliana*) unattractive and unpleasant to aphids, speeding up aphid dispersal" [46,47]. "CMV infection increases plant SA signalling, which is linked to ROS, to limit aphid fertility and induce winged aphids in A*rabi*dopsis plants as a result, its virus propagation is improved" [48].

11. EVOLUTION

Genetic drift linked to population bottlenecks during systemic colonisation of the host and, likely also, during host-to-host transmission, has been demonstrated to counteract genetic diversity. Different evolutionary constraints have been revealed by sequence analysis for several viral proteins, indicating distinct evolutionary dynamics. The exchange of RNA sequences through recombination or the reassortment of genomic regions is a second source of genetic diversity. It has been demonstrated through experimentation that recombination takes place between the 3' nontranslated regions of the genomic RNAs, accounting for up to 11% of the population. It has been demonstrated that isolates infecting Alstroemeria may have a higher fitness level for recombinants in the 3' nontranslated region in some hosts. Recombination in the RNA3 was also common in combined CMV and TAV infections**.** Stem-loop structures in the RNA appear to aid recombination between CMV strains or between CMV and TAV strains. Analysis of the genetic makeup of field populations of CMV reveals that selection acts against most recombinants and that recombinant RNAs are not common, despite a wealth of data supporting frequent recombination. Pseudorecombination, or the reassortment of genomic segments, is another process of genetic exchange. Reassortants that interchange any genomic sequence between distinct CMV strains have been discovered; these strains proliferate effectively in laboratory conditions. The various phylogenies found for each genomic RNA show that reassortment may have been a significant factor in the development of CMV. Natural reassortants have also been reported. Similar to recombinants, reassortant isolates are uncommon in field populations and appear to be selected against. There is evidence of selection acting against genotypes originating

from genetic exchange, which points to the co-adaptation of the various virus genes, which reduces fitness when it is disturbed. Studies of the CMV population structure in California and Spain show a metapopulation structure with localised extinctions and recolonizations, indicating the possibility of population bottlenecks that are most likely connected to unfavourable seasons for the aphid vectors and/or host plants. Interestingly, population structure satellite RNA does not follow this pattern [14].

The various phylogenies found for each genomic RNA show that reassortment may have been a significant factor in the development of CMV. Natural reassortants have also been reported. Similar to recombinants, reassortant isolates are uncommon in field populations and appear to be selected against. There is evidence of selection acting against genotypes originating from genetic exchange, which points to the co-adaptation of the various virus genes, which reduces fitness when it is disturbed. Studies of the CMV population structure in California and Spain show a metapopulation structure with localised extinctions and recolonizations, indicating the possibility of population bottlenecks that are most likely connected to unfavourable seasons for the aphid vectors and/or host plants. Satellite RNA analyses conducted during CMV plus satellite RNA epidemics in Italy and Spain have revealed an undifferentiated population of satellite RNAs. The distinct population structure for both CMV and its satellite RNAs suggests that rather than satellite expansion being associated with a specific CMV strain, the satellite RNAs spread as a molecular parasite on the CMV population.

12. MANAGEMENT

The planting of resistant crops can help control CMV, yet many crop species lack resistance to a wide variety of CMV strains. Because the virus spreads nonpersistently, the use of insecticides to eliminate the aphid vectors of the virus has had only patchy success. The virus would have spread even before the aphids were killed by the insecticide. Instead, insecticides are employed to lower the population of aphids and so slow the infection's progressive progression. In order to remove the source of infectious material from field borders, it is crucial to clear weeds because many of them are asymptomatic hosts and operate as reservoirs for the virus. This holds true for removing diseased crop plants from the ground while they are still growing. The use of transgenic plants producing RNA silencing- or protein-mediated resistance are examples of additional resistance sources.

13. CONCLUSION

A broad-spectrum resistance to CMV and other viruses infecting the same crop species is possible through the use of pyramiding viral segments from different subgroups, despite the fact that most of these approaches have only produced resistance to members of one of the two major subgroups. RNAs have also been employed to offer CMV resistance. Although this has been successful, there are worries concerning both the use of mild strains of a deadly pathogen and the use of CMV for cross-defense against highly pathogenic strains.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Palukaitis P, Garcia-Arenal F. Cucumber mosaic virus. Descriptions of plant viruses. CMI/AAB. 2003;400:1-23.
2. Hsu HT, Barzuan L, Hsu Y, Bliss H, Perry KL. Identification and subgrouping of Cucumber mosaic virus with mouse monoclonal antibodies. Phytopathol. 2000; 90:615-620.
3. Gallitelli D. The ecology of Cucumber mosaic virus and sustainable agriculture. Virus Research. 2000;71(1-2):9-21.
4. Doolittle SP. A new infectious mosaic disease of cucumber. Phytopathol. 1916;6 (2):145-147.
5. Jagger IC. Experiments with the cucumber mosvertaic disease. Phytopathol. 1916;6 (2):149-151.
6. Kamat MN, Patel MK. Notes on two important plant diseases in Bombay. Stat. Bull. PI. Prot., New Delhi. 1951;3:16.
7. Rao DG. Studies on a new strain of banana mosaic virus in South India. In: Proc. National Seminar on Banana Production Technology. C.R. Muthukrishnan and J.B.M Abdul Khader (Eds.). Tamilnadu Agricultural University, Coimbatore, India. 1980;155-159.
8. Bhargava KS. Some properties of four strains of cucumber mosaic virus. Ann. Appl. Biol. 1951;38:377-438.

9. Bhargava KS. Natural occurrence of cucumber mosaic virus on chilli in India. Indian Phytopathol. 1951;20:380-381.
10. Anderson BJ, Boyce PM, Blanchard CL. RNA4 sequences from Cucumber mosaic virus subgroup I and II. Gene. 1995;161: 193-194.
11. Palukaitis P, Roossinck M, Dietzgen RG, Francki RIB. Cucumber mosaic virus. Adv. Virus Res. 1992;41:281-348.
12. Roossinck MJ, Bujarski J, Ding SW, Hajimorad R, Hanad K, Scott S, Tousignant M. Virus taxonomy-eighth report of the International Committee on Taxonomy of Viruses. In: Family Bromoviridae. Academic Press. San Diego, Calif. 2002;923-935.
13. Srivastava A, Raj SK. High molecular similarity among Indian isolates of cucumber mosaic virus suggests a common origin. Curr Sci. 2004;87:1126-1131.
14. Garcia-Arenal F, Palukaitis P. Cucumber Mosaic Virus. Scottish Crop Research Institute, UK. 2008;615-619.
15. Vishnoi R, Kumar S, Raj SK. Molecular characterization of a Cucumber mosaic virus isolate associated with mosaic disease of banana in India. Phyto-parasitica. 2013;41:545–555.
16. Dubey VK, Aminuddin, Singh VP. Molecular characterization of cucumber mosaic virus infecting gladiolus, revealing its phylogeny distinct from the Indian isolate and alike the Fny strain of CMV. Virus Genes. 2010;41:126-134.
17. Kumari R, Bhardwaj P, Singh L, Zaidi AA, Hallan V. Biological and Molecular Characterization of Cucumber mosaic virus Subgroup II Isolate Causing Severe Mosaic in Cucumber. Indian J. Virol. 2013; 24(1):27-34.
18. Deloko DCT, Chofong NG, Ali IM, Kachiwouo IG, Songolo FO, Nkang Manock ARN, Kamgaing M, Fonkou T, Njukeng AP. Detection of cucumber mosaic virus on *Solanum lycopersicum* L. and *Capsicum annuum* L. in the Western region of Cameroon. Journal of Agriculture and Food Research. 2022;8:2666-1543.
19. Edwardson JR, Christie RG. In: Cucumoviruses (ed). CRC handbook of viruses infecting legumes. CRC Press. Boca Raton, Florida.1991;293-319.
20. Samad A, Raj SK, Srivastava A, Chandra G, Ajayakumar PV, Zaim M, Singh BP. Characterization of a cucumber mosaic virus isolates infecting Egyptian henbane (*Hyoscyamus muticus* L.) in India. Acta Virol. 2000;44:131-136.
21. Raj SKA, Srivastava T, Chandra G, Singh BP.. Characterization of cucumber mosaic virus isolate infecting *Gladiolus* cultivars and comparative analysis of serological and molecular methods for sensitive diagnosis. Curr. Sci. 2002;83:1132-1136.
22. Sudhakar N, Nagendra-Prasad D, Mohan N, Murugesan K. First report of cucumber mosaic virus subgroup II infecting *Lycopersicon esculentum* in India. Plant Disease. 2006;90:1457.
23. Raj SK, Kumar S, Pratap D, Vishnoi R, Snehi SK. Natural Occurrence of Cucumber mosaic virus on *Rauvolfia serpentina*, a new record. Plant Disease. 2007;91:322.
24. Raj SK, Kumar S, Snehi SK. First report of cucumber mosaic virus on *Jatropha curcas* in India. Plant Disease. 2008;92:171.
25. Aglave BA. Krishnareddy M, Patil RV, Andhale MS. Molecular identification of a virus causing banana chlorosis disease from marathwada region. Int. J. Biochem. 2007;3:13-23.
26. Hubili N, Francki RIB. Comparative studies on tomato aspermy and cucumber mosaic viruses. III. Further studies on relationship and construction of a virus from part of the two viral genomes. Virology. 1974;61:443-449.
27. Paden KWC, Symons RH. Cucumber mosaic virus contains a functionally divided genome. Virology. 1973;53:487-492.
28. Suzuki M, Kuwata S, Kataoka J, Masuta C, Nitta N, Takanami Y. Functional analysis of deletion mutation of Cucumber mosaic virus RNA3 using as *in vitro* transcriptions system. Virology. 1991;183:108-113.
29. Schwinghamer MW, Symons RH. Fractionation of Cucumber mosaic virus RNA and its translation in a wheat embryo cell-free system. Virology. 1975;63:252-262.
30. Hayes RJ, Buck KW. Complete replication of a eukaryotic virus RNA in vitro by a purified RNA dependent RNA polymerase. Cell. 1990;63:363-368.
31. Gibbs A. Viruses and plasmodesmata. In intercellular communication in plants: Studies on plasmodesmata. 1976;149-164.
32. Hull R. The movement of viruses in plants. Annu. Rev. Phytopathol. 1989;27:213-240.
33. Atavekov JG, Taliansky ME. Expression of plant virus -coded transport function by

different viral genomes. Adv. Virus Res. 1990;38:201-248.
34. Doem CM, Lapodot M, Beachy RN. Plant virus movement protein. Cell. 1992;69:221-224.
35. Rashid MH, Khalequzzaman KM, Alam MS, Uddin SA, Green SK. Screening of different sweet pepper lines against cucumber mosaic virus and chili veinal mottle virus. Int. J. Sustain. Crop Prod. 2007;2(3):1-4.
36. Zitter TA, Murphy JF. Cucumber mosaic. The Plant Health Instructor; 2009. DOI: 10.1094/PHI-I-2009-0518-01
37. Mazidah M, Yusoff K, Habibuddin H, Tan YH, Lau WH. Characterization of Cucumber Mosaic Virus (CMV) Causing Mosaic Symptom on *Catharanthus roseus* (L.) G. Don in Malaysia Pertanika J. Trop. Agric. Sci. 2012;35(1):41-53.
38. Agrios GN. Plant Pathology (5th Ed.). Elsevier Academic Press, San Diego. 2005;922.
39. Zitikaite I, Staniulis J, Urbanaviciene L, Zizyte M. Cucumber mosaic virus identification in pumpkin plants. Zemdirbyste Agric. 2011;98(4):421-6.
40. Tripathi S, Patil BL, Verma R. Viral Diseases of Banana and Their Management. Chapter 17; 2016. DOI: 10.1007/978-981-10-1406-2-17
41. Vinodhini J, Rajendran L, Abirami R, Karthikeyan G. Co-existence of chlorosis inducing strain of Cucumber mosaic virus with Tospoviruses on hot pepper (*Capsicum annuum*) in India. Sci. Rep. 2021;11(1):87-96.
42. Diningsih E, Yanda RF, Rahardjo IB, Wegadara M, Thamrin M. Identifying Cucumber mosaic virus (Cucumovirus, Bromoviridae) isolates on *Impatiens* (Balsaminaceae): A preliminary study using molecular characterization. Acta Hortic. 2022;1334: 193-198.
43. Mauck KE, De Moraes CM, Mescher MC. Biochemical and physiological mechanisms underlying effects of cucumber mosaic virus on host-plant traits that mediate transmission by aphid vectors. Plant Cell Environ. 2014;37:1427-1439.
44. Wu D, Qi T, Li W, Tian H, Gao H, Wang J, Ge J, Yao R, Ren C, Wang X, Liu Y, Kang L, Ding S, Xie D. Viral effector protein manipulates host hormone signalling to attract insect vectors. Cell Res. 2017;27:402–415.
45. Guo H, Gu L, Liu F, Chen F, Ge F, Sun Y. Aphid-borne viral spread is enhanced by virus-induced accumulation of plant reactive oxygen species. Plant Physiol. 2019;179:143-155.
46. Westwood JH, Groen SC, Du Z, Murphy AM, Anggoro DT, Tungadi T, Luang-In V, Lewsey MG, Rossiter JT, Powell G, Smith AG, Carr JP. A trio of viral proteins tunes aphid-plant interactions in A*rabi*dopsis thaliana. PLoS One. 2013;8: e83066.
47. Donnelly R, Cunniffe NJ, Carr JP, Gilligan CA. Pathogenic modification of plants enhances long-distance dispersal of non-persistently transmitted viruses to new hosts. Ecology. 2019;100:e02725.
48. Shi X, Gao Y, Yan S, Tang X, Zhou X, Zhang D, Liu Y. Aphid performance changes with plant defense mediated by cucumber mosaic virus titre. Virol J. 2016;13:70.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/111195