*Review Article*

***Pigeonpea Sterility Mosaic Virus: A brief overview***

ABSTRACT

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| Pigeonpea sterility mosaic virus (PPSMV)belongs to the genus Emaravirusand causes sterility mosaic disease.PPSMV is prevalent in Asian countries, such as Bangladesh, Nepal, Thailand, Myanmar, Sri Lanka, and China.PPSMV is transmitted by the eriophyid mite, *Aceria cajani* (Channabasavanna).PPSMV and the vector eriophyid mite *A.cajani*are specific to pigeon peaand the wild species*C. scarabaeoides* and *C. cajanifolius*.Symptoms of this disease include mosaic and sterililitywith malformation or no flowers symptoms, hence the name of the disease as ‘sterility mosaic. ’Resistant/tolerant cultivars can be used to manage various diseases |

*Keywords: [Pigeonpea sterility mosaic virus, PPSMV, SMD, eriophid mite, Aceria cajani, pigeon pea}*

1. INTRODUCTION

Pigeonpea sterility mosaic virus (PPSMV)belongs to the genus Emaravirus and causes sterility mosaic disease (SMD) in pigeonpea [*Cajanuscajan* (L.) Millsp] (Patil & Kumar, 2015; Kumar et al., 2005; Kumar et al., 2003). This disease is characterized by stunted and bushy plants, leaves of reduced size with chlorotic rings or mosaic symptoms, and partial or complete sterility of flower production (i.e., sterility) (Kumar et al., 2017). The virus PPSMVis a negative-sense, single-stranded RNA with a segmented genomethat is transmitted in a semi-persistent manner by the eriophyid mite *Aceriacajani*Channabassavanna (Acari: Arthropoda) (Kulkarni et al., 2002; Elbeaino et al., 2015). This disease is very specific to pigeonpea and wild species such as *C. scarabaeoides* and *C. cajanifolius*(Kalaichelvi*et al.,*2020).

1. **TAXONOMY**

*Pigeonpea Sterility Mosaic Virus*(PPSMV)belongs to genus Emaravirusunder the family Fimoviridae, order Bunyaviraleswith similarity to Tospovirus and Tenuivirus, withnegative sense ss RNA (Manjunatha*et al*.,2021; Jones et al., 2004).

1. **GEOGRAPHICAL DISTRIBUTION**

Sterility mosaic disease is endemic to India, Nepal, Bangladesh, and Myanmar, and constarts with pigeonpea cultivation(Patil*et al.,*2017).Kumar*et al.* (2004) reported that PPSMVisprevalent in Asian countries, such asBangladesh, Nepal, Thailand, Myanmar, Sri Lanka, and China.In India, Andhra Pradesh, Bihar, Chhattisgarh, Gujarat, Karnataka, Maharashtra, Punjab, Tamil Nadu, Telangana, Uttar Pradesh, and West Bengal will have PPSMV(Singh &Raghuraman, [2011](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0078)).

1. **TRANSMISSION**

PPSMV can be transmitted primarily by the vector and to a minor extent by mechanical transmission or nematodes.

**4.a. Vector transmission**

PPSMV is transmitted by the eriophyid mite *Aceria cajani* (Channabasavanna) (Arthropoda: Acari: Eriophyidae)in a semi-persistent manner. These arethe smallest obligate arthropod plant pests with white to yellow color and spread mainly by wind.  *Aceriacajani* mites inhabit the lower surface of leaves, occupy the symptomatic leaves of PPSMV‐infected plants, and feed without mechanical damage to the host plants. Eriophyid mites cause mixed infections by transmitting emaraviruses and potyviruses. A single mite can transmit up to 53% of the viruses.A population of more than five mites can be transmitted to 100 per cent.  *A. cajani* mites require an acquisition access period (AAP) of 15 min and an inoculation access period (IAP) of 90 min for transmitting PPSMV with no latent period. PPSMV infection of pigeonpea increases *A. cajani* compared with that in healthy plants,which might be a beneficial relationship between the virus and vector. Mites reach the epidermal cells or adjacent layers of mesophyll tissue located on the undersurface of plant leaves with the help of a short stylet. The mites can survive for only 13 h. Mites can multiply within a few weeks in susceptible pigeon pea genotypes. The dispersal of these mites occurs by passive dispersal bythe wind.In India, no *A. cajani* biotypes have been found to be involved in the transmission of PPSMV(Patil and Kumar,2015).

Size of *Aceriacajani* are vary from 200- 250 µm with pale crimsonto orange in color with a short life span of about 2 weeks. They resided in the lower portions of the leaves. Both young adults and adults can transmit the virus but require 48 h after the acquisition of the virus, which can be transmitted by grafting and to some extent by nematodes (Gupta *et al*.,2022).

**4.b. Mechanical Transmission**

PPSMV cancause 10 to 30 per centof infectionsin *Nicotiana benthamiana* and *N. clevelandii* in purified form by sap inoculation, but is not transmissible to pigeonpea or other hosts. PPSMV can be transmitted artificially by *A. cajani* to the French bean and to pigeonpea by grafting(Mielke‐Ehret and Mühlbach, [2012](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0049)). PPSMV is detectable only in the seed coat but not in the cotyledons. PPSMV can infect several cultivated species, including wild pigeon pea, *N. benthamiana*, *N. clevelandii*, *Phaseolus vulgaris* and *Chrozophorarottleri*. *Hibiscus panduriformis* (Malvaceae) is infested with *A. cajani*, but will not be infected byPPSMV, thus acting as a refuge for mite survival and spreading PPSMV(Kumar *et al*., [2007](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0039);Patil and Kumar,2015).

1. **HOST RANGE AND SYMPTOMATOLOGY**

PPSMV can infect cultivated wild species of pigeonpea and other hosts such as*Nicotianabenthamiana, N. clevelandii, P. vulgaris* and *Chrozophorarottleri*. Pigeonpea and a few wild species of *Cajanus*(*C. scarabaeoides* and *C. cajanifoliu*)will be infected by the vector *A. cajani*with a strong communalistic relationship between the virus-infected plants and the vector. *Nicotiana benthamiana*can be infected by sap inoculation, and it has also been transmitted to French bean (*Phaseolus vulgaris* L.) through eriophyid mites(Patil and Kumar,2015).

1. **SYMPTOMATOLOGY**

Plants infected with PPSMVshow mosaic and sterility,with malformation or no flowering symptoms, such as ’sterility mosaic. Plants showa pale green appearance, reduced leaf size, mosaic, mottling, and distortion of leaves, with several secondary and tertiary branches from the axillary leaf. The partial or complete termination of plant reproductive parts results in complete crop failure. Late-infected plants do not show clear symptoms, but ratooned crops showcomplete mosaicism and plant sterility. The symptoms will be categorized into three forms:a) complete sterility with severe mosaic of leaflets, with no flowers and pods during early infection, and pods will be formed if infected beyond 45 days after sowing; b)partial sterility with mild mosaic in a few leafetsthat will be devoid of flowers and bear pods if infected beyond 45 days after sowing; and c) ring spotwith green islands bordered by a chlorotic halo on leaves, which diminish as the plants matures[Fig1(a,b,c,d)]. All these symptoms can be observed in genotypes such as ICP-2376.At the reproductive stage, infected plants can be easily identified from a far distance as they appear green owing to the cessation of reproductive parts with complete or partial sterility(Manjunatha*et al*. 2021;Saiprathap,2020).

1. **PATHOGENICITY/PATHOGENIC CYCLE/PATHOGEN BIOLOGY**

**7.1. Pathogen biology**

PPSMV virus particles are asymmetric, measuring 3–8 mm in diameter, with a coat protein of 32 kDa and 4–5 RNA segments each of 0.8–3.5 kb. Genome organization and sequence characterization of PPSMV-1 and PPSMV-2 revealed that these viruses were comprised of negative-sense segmented RNA, which consists of 4–8 ssRNA segments in the genome. The first PPSMV sequence was renamed PPSMV‐1, which contains five genomic RNA nucleotide fragments of 7022[RNA-dependent RNA polymerase (RdRp)], 2223[a glycoprotein (GP)], 1442[a nucleocapsid protein (NP)], 1563[a movement protein (MP)], and 1689(p5), respectively. Later presence of PPSMV 2 was revealed. In PPSMV-2, the first four RNA fragments shared maximum sequence similarity with FMV (Fig mosaic virus) compared to PPSMV-1. PPSMV‐2 has been found to be linked to six -ssRNA segments of 7009nt, 2229nt, 1335nt, 1491nt, 1833nt and 1194 nt encoding for the RNA-dependent RNA polymerase (RdRp, p1 of 266 kDa), the precursor of glycoprotein p2 (74.5 kDa), the nucleocapsid p3 (34.9 kDa), and movement protein p4 (40.7 kDa), p5 (55 kDa), and p6 (27 kDa) with unknown functions(Manjunatha*et al,*2021). Patil *et al*. (2017) were the first to report mixed infections with PPSMV‐1 and PPSMV‐2 in India.

**7.2. Pathogenic cycle**

The primary source of virus inoculum is infected plant debris, perennial pigeonpea, and *C. scarabaeoides*(Kumar *et al*., [2008](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0034)), whereas the secondary source of inoculum is the transmission of PPSMV by the [eriophyid mite](http://en.wikipedia.org/wiki/Eriophyidae) *Aceria [cajani](http://in.msn.com/default.aspx)* (Manjunatha*et al.*,2021).

1. **ISOLATION AND IDENTIFICATION OF PPSMV**

The virus can be isolated from pigeon pea from different locations in the peninsular region of India and from PPSMV-affected pigeonpea samples infected by graft inoculation and by infective mites(*A.cajani*). The collected samples should be placed in zip-lock plastic bags, transported in cold packs frozen in liquid nitrogen, and stored at 80 °C freezer until further use. One hundred  milligrams of leaf tissue from symptomatic and healthy pigeonpeawill beground in liquid nitrogen for RNA extraction. RNA purity and quality can be checked using a spectrophotometer and stored at − 20 °C(Saiprathap,2020).

After extraction of total RNA, RT-PCR should beperformed with specific primers that contain aggregates of highly flexuous, irregularly branched, filamentous virus-like particles (VLPs) of 8 to 11 nm in diameter and of undetermined length, which resembletenuiviruses along with a major protein of 32 kDa and up to 6 segmented RNA species of size1.1-6.8 kb. Polyclonal antibodies to PPSMV VL Preparations produced in rabbits are effective in detecting PPSMV in plant tissues using double antibody sandwich (DAS). PPSMV can be detected by ELISA in all PPSMV-infected pigeonpea plants, and the virus-specific 32 kDa protein can be detected in extracts of groups of vector mites by western immunoblotting. Fulfilling Koch’s postulates is difficult due tothe unstable nature of the virus and the difficulty of infecting pigeonpea by mechanical inoculation. Although the purified PPSMV VLP preparations were not infective to plants,PPSMV was transmitted experimentally by mechanical inoculation of fresh leaf sap extracts of PPSMV-affected pigeonpea to *N.benthamiana* and *N. clevelandii,* but not to pigeonpea, but not from *N.benthamiana*to pigeonpea. Systemically infected leaves of *Nicotiana* species develop mild chlorosis and some necrotic spots, and ultrastructural studies of PPSMVinfected pigeonpea, *N.benthamiana* and French plants will have 100-150 nm quasi-spherical membrane bound-bodies (MBBs) and fibrous inclusions (FIs). The MBBs were labelled in situ specifically with antiserum to PPSMV, indicating that they contained the PPSMV-specific 32kDa antigen. The FIs found in PPSMV-infected cells may be non-structural inclusion proteins of PPSMV, indicating that it is a previously undescribed virus with an unusual combination of properties(Kumar *et al.,*2004).

Identification of PPSMV‑I and PPSMV‑II infections will be subjected to RT-PCR using specific primers for the RNA-3 segment of PPSMV-I and PPSMV-II, which will be used to detect PPSMV-I and PPSMV-II(Manjunath*et al.,*2022).

1. **EPIDEMIOLOGY**

The spread of PPSMV involves pathogenic viruses, mites, host plants, and environmental factors. PPSMV is an endemic disease that varies with the season and locality (Kumar *et al*., [2008](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0034)).The spread of PPSMV from fields by mites(viruliferous)depends on the distance from volunteer or perennial plant weather conditions favoring eriophyid mites. Plants cultivated under irrigation are more vulnerable to early infection with PPSMV (Dharmaraj *et al*., [2004](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0008)).

**9.1. Factors influencing vectors**

Stubble of pigeonpea in the rainfed field after harvest and plants thriving near water sources with green foliage support the proliferation of viruliferous mites (Dharmaraj *et al*., [2004](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0008)).Sprouting from infected stubbles after summer showers served as the primary source of the inoculum. The spread of PPSMV within fields mainly depends on the accessibility to sources of inoculum,age of the plant, genotype, weather parameters, and mite population. It was evident that *A. cajani* on pigeonpea constituted only one species across the Indian subcontinent, and that no other *Aceria* species, and probably no *A. cajani* biotypes differing in their vectoring ability, were involved in the transmission of PPSMV(Patil and Kumar,2015).

Inter or mixed cropping of pigeon peas with sorghum or millet influences the incidence of the disease. The ratooning of crops also increases disease incidence. Shade and humidity favor high multiplication of mite populations, mostly in hot summer weather.

**9.2. Pathogen factors**

In PPSMV, the size and appearance of its VLPs, and the number and size of its protein and RNA components resemble viruses in the genus Tenuivirus. Ultrastructural studies of PPSMV-infected pigeon peas showed 100 to150-nm quasi spherical membrane-bound bodies (MBBs) and fibrous inclusions (FIs). Under *in situ*,these membrane-bound bodies were labeled with antiserum to PPSMV and indicated that they mainly contained the PPSMV-specific 32-kDa antigen. The fibrous inclusions (FIs) are nonstructural inclusion proteins of the PPSMV virus(Guptal*et al*.,2022)

**9.3. Environmental factors**

mite population is influenced by relative humidity (Kaushik *et al*., [2013](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6638375/#mpp12238-bib-0025)).Temperatures of approximately 20–30 °C were found to be favorable for mite proliferation. However, high temperatures and heavy rainfall are unfavorable for growth(Patil and Kumar,2015).

1. **ECONOMIC IMPORTANCE**

Early infection with PPSMV results in an infection rate of 100 per cent.This disease is known as ‘green plague,’ as the infected plants remain in the vegetative state without flower production, this is epidemic in India and a few other South‐East Asian countries like Myanmar,Bangladesh Sri Lanka, Thailand, and Nepal.  PPSMV is estimated to result in an annual yield loss of over  US $ 300 million in India alone. The disease was first reported in Bihar in 1931. It occurs frequently in the states of Maharashtra, Bihar, Uttar Pradesh, Tamilnadu, Gujarat, and Punjab, with significant yield losses of up to 90% when the infection starts within 45-50 days after sowing, as the plants completely fail to produce flowers and pods(Patil and Kumar,2015).

1. **CONTROL MEASURES**

**11.1. Cultural methods**

Removal of infected plant debris, isolation of new crops from perennial pigeonpea, and crop rotation. Some of the wild relatives of pigeonpeasare refuged by mites because of their thicker leaf cuticle and epidermal cell wall, which obstructs the stylet of mites into the leaf epidermis.The wild species, *C. scarabaeoides* (ICPW 94), is resistant to all PPSMV variants. The existence of distinct PPSMV strains from different regions of the country makes it challenging to combine broad-spectrum resistance from wild pigeonpea relatives that are resistant to multiple strains of PPSMV.Wild species such as *C. scarabaeoides*, *C. cajanifolius*and perennial pigeon pea plants that harbor mites during the off-season should be removed and destroyed(Gupta *et a.l,*2022).

**11.2. Vector control**

The migration of *A. cajani* is difficult to monitor. The presence of *A. cajani* was noted as a result of the symptoms of pigeonpea due to PPSMV secondary infection(Maurya *et al*., 2017).Species of plants, such as Attilocia, should be destroyed because they harbor mites during the off-season(Gupta *et al.,*2022).

**11.3. Phytosanitary and early warning measures**

The summer rain (March to April) contributes to a high mite infestation, with an increased incidence of PPSMV in new pigeonpea. Summer rains support the survival of PPSMV-infected pigeonpeas in the field. Such plants harbor *A. cajani* and allow for high multiplication during the off-season, which spreads to newly sown crops(Manjunatha*et al,*2021; Gupta *et a.l,*2022).

**11.4. Chemical methods**

Complete reduction of PPSMV can be achieved by combining wettable sulfur + propargite and wettable sulfur alone by reducing the mite population (Manjunatha*et al*. 2012). Sprayingfenazaquin at 0.1% 30 days after sowing and 15 days after the first spray resulted in an 80.9% reduction in PPSMV(Rajeswari *et al.*,2016). Seed treatment and foliar spraying of systemic acaricidescurtailed the propagation of *A. cajani* in the field. Spraying propargite at 0.1% at 25 and 40DAS resulted in the lowest PPSMV incidence (7.72%) (Maurya *et al*., 2017).

**.**  The crop was sprayed with Dicofol (Kelthane), 450 ml, Phosalone-600 mi, or 450 ml in 300 liters of water per acre, twice,15 and 30 days after sowing for the vector control. Soil application of chemicals, such as Carbofuron 3G -12 kg or Phorate 10 – 4 kg, should be mixed properly with 10 kg sand around the stem portion of the plant (Gupta *et al.,*2022).

**11.5.Resistant/Tolerant varieties**

ICP 7035 is aPPSMV-resistant variety(Rangaswamy*et al.,*2005).PPSMV Tolerant varieties, such as ICPL 227, Jagruti, Bahar, and ICPL 87119, should be grown(Gupta *et al.,*2022).

References

Dharmaraj, P.S., Narayana, Y.D., Kumar, P.L., Waliyar, F. and Jones, A.T. (2004) Pigeonpea sterility mosaic disease: an emerging problem in northern Karnataka. *Int. Chickpea PigeonpeaNewsl.* 11, 47–49.

Gupta, Prince & Kaur, Manpreet & Roy, Bishal& Joshi, Sarthak & Kumar, Abhishek & Singh, Prabhas Shankar. (2022). Pigeon pea Diseases and their Recent Approaches for Sustainable Management in India: Current Status and Future Prospective.

Kalaichelvi, K. (2020). Report on Pigeonpea Sterility Mosaic Virus (PSMV) Disease Incidence in CO (Rg) 8 in Tamil Nadu. Int.J.Curr.Microbiol.App.Sci. 9(11), 1112-1115. doi: https://doi.org/10.20546/ijcmas.2020.911.12

Kaushik,D., Seweta, S., Chandra, N.B., Chauhan, V.B. and Singh, R.N.(2013). Correlation between mite population (Aceriacajani) and environmental factors causing sterility mosaic disease of Pigeon pea. *Int J Life Sci*, *1*(3), pp.228-232

Kumar, L. & Jones, AT &Waliyar, Farid. (2004). Biology, Etiology and Management of Pigeonpea Sterility Mosaic Disease. Annual Review of Plant Pathology. 3, 1-24.

Kumar, P.L, Jones, A.T., Waliyar, F., Sreenivasulu, P., Muniyappa, V., Latha, T.K.S. and Saxena, K.B. (2007) Pigeonpea sterility mosaic diseaseIn: *Diagnosis and Detection of Viruses Infecting ICRISAT Mandate Crops* (Kumar P.L. and Waliyar F., eds), pp. 15–21. Patancheru, India: ICRISAT. ICRISAT's Methods Manual.

Kumar, P.L., Jones, A.T. and Waliyar, F. (2008) Virus diseases of pigeonpea I In: *Characterization, Diagnosis and Management of Plant Viruses. Vol. 3: Vegetable and Pulse Crops* (Rao G.P., Kumar P.L. and Holguin‐Pena R.J., eds), pp. 235–258. Texas, USA: Studium Press.

Manjunatha, L., Ramappa, H.K., Mahantesha, S. R. V., Gowda, M. B., Rajappa, P. V., Kavitha, T. R. (2012) Management of sterility mosaic disease (SMD) of pigeonpea. Plant Arch 12,1007–1012

Manjunatha L, Ramappa HK, Rangaswamy KT (2018) Role of leaf morphology in defense against sterility mosaic disease of pigeonpea. J Environ Biol 39:298–305

Manjunatha, Lakshmaiah & Ramappa, Honnaghatta & Puyam, Anita & Srinivasa, Nagappa. (2021). Pigeonpea Sterility Mosaic Virus: a threatening virus of pigeonpea, current scenario and its control.

Mielke‐Ehret, N. and Mühlbach, H.P. (2012) Emaravirus: a novel genus of multipartite, negative strand RNA plant viruses. *Viruses*, 4, 1515–1536.

Patil BP, Meenakshi D, Ritesh M (2017) Variability of emaravirus species associated with sterility mosaic disease of pigeonpea in India provides evidence of segment reassortment. Viruses 9,183

Patil BL, Kumar P.L(2015) Pigeonpea sterility mosaic virus: a legume-infecting Emaravirus from South Asia. Mol Plant Pathol. Oct;16(8),775-86. doi: 10.1111/mpp.12238. Epub 2015 Apr 23. PMID: 25640756; PMCID: PMC6638375.

Rahul Kumar Maurya, Birendra Kumar, Rahul Kumar and Mukesh Singh. (2017). Transmission of Pigeon Pea Sterility Mosaic Virus and Management of Sterility Mosaic Disease of Pigeonpea by Different Acaricides under Middle IGP of Bihar.*Int.J.Curr.Microbiol.App.Sci.* 6(8), 3711-3716. doi: <https://doi.org/10.20546/ijcmas.2017.608.448>

Rangaswamy, Katharighatta & V, Muniyappa & Kumar, Lava & Saxena, Kulbhushan & M, Byregowda & N, Raghavendra & K, Pandurangaiah & Kumar, R & Waliyar, Farid & AT, Jones. (2005). ICP 7035 - A Sterility Mosaic Resistant Vegetable and Grain Purpose Pigeonpea Variety. Journal of SAT Agricultural Research. 1.

Sayiprathap, B. R., Patibanda, A. K., Prasanna Kumari, V., Jayalalitha, K., Ramappa, H. K., Rajeswari, E., Karthiba, L., Saratbabu, K., Sharma, M., Sudini, H. K. (2022). Salient Findings on Host Range, Resistance Screening, and Molecular Studies on Sterility Mosaic Disease of Pigeonpea Induced by *Pigeonpea sterility mosaic viruses* (*PPSMV-I* and *PPSMV-II*). Front Microbiol. 1, 13:838047. doi: 10.3389/fmicb.2022.838047. PMID: 35432270; PMCID: PMC9012581.

Singh N, Narula B, Ujinwal M, Langyan S.(2021) Pigeonpea sterility mosaic virus a green plague-Current status of available drug and new potential targets. Ann Proteom Bioinform. 5, 008-026.

Singh, J. and Raghuraman, M. (2011) Emerging scenario of important mite pests in north India. *Zoosymposia*, 6, 170–179.

Kumar, S., Subbarao, B. L., & Hallan, V. (2017). Molecular characterization of emaraviruses associated with Pigeonpea sterility mosaic disease. Scientific Reports, 7(1), 11831.

Kulkarni, N. K., Kumar, P. L., Muniyappa, V., Jones, A. T., & Reddy, D. V. R. (2002). Transmission of Pigeon pea sterility mosaic virus by the eriophyid mite, Aceria cajani (Acari: Arthropoda). Plant disease, 86(12), 1297-1302.

Jones, A. T., Kumar, P. L., Saxena, K. B., Kulkarni, N. K., Muniyappa, V., & Waliyar, F. (2004). Sterility mosaic disease—the “green plague” of pigeonpea: advances in understanding the etiology, transmission and control of a major virus disease. Plant Disease, 88(5), 436-445.

Patil, B. L., & Kumar, P. L. (2015). Pigeonpea sterility mosaic virus: a legume‐infecting Emaravirus from S outh A sia. Molecular Plant Pathology, 16(8), 775-786.

Patil, B. L., & Kumar, P. L. (2015). Pigeonpea sterility mosaic virus: a legume‐infecting Emaravirus from S outh A sia. Molecular Plant Pathology, 16(8), 775-786.

Kumar, P. L., Jones, A. T., & Waliyar, F. (2005). Biology, etiology and management of pigeonpea sterility mosaic. Ann Rev Plant Pathol, 3, 77-100.

Kumar, P. L., Jones, A. T., & Reddy, D. V. R. (2003). A novel mite-transmitted virus with a divided RNA genome closely associated with pigeonpea sterility mosaic disease. Phytopathology, 93(1), 71-81.

Elbeaino, T., Digiaro, M., Uppala, M., & Sudini, H. (2015). Deep sequencing of dsRNAs recovered from mosaic-diseased pigeonpea reveals the presence of a novel emaravirus: pigeonpea sterility mosaic virus 2. Archives of virology, 160, 2019-2029.