2016 INTERNATIONAL YEAR OF PULSES

Yellow Mosaic Disease in Pulses

Diagnosis and Management

ISO 9001-2008 Certified
ICAR-Indian Institute of Pulses Research
Kanpur 208024

Yellow Mosaic Disease in Pulses

Diagnosis and Management

Naimuddin
Mohd. Akram
N.P. Singh

ISO 9001-2008 Certified
ICAR-Indian Institute of Pulses Research
Kanpur 208024

Printed : August, 2016

Publication no. : 7/2106

Published by : Dr. N. P. Singh, Director
ICAR-Indian Institute of Pulses Research,
Kanpur

Edited by : Mr. Diwakar Upadhyaya

Citation : Naimuddin, Akram, M. and Singh, NP. (2016).
Yellow Mosaic Disease of Pulse Crops: Diagnosis
and Management.
ICAR- Indian Institute of Pulses Research, Kanpur.

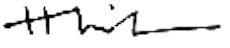
Printed at : Maheshwari & Sons, Moti Nagar, Lucknow. Ph. : 0522-4071816

Foreword

Enhancing food production for increasing population of the nation is biggest challenge Indian Agriculture faces today. Pulses are important components of Indian diet and known to provide nutritional security to millions of Indian who are religiously or culturally vegetarians. Since most of the pulses are grown under rainfed conditions any aberration in monsoon disturbs pulse production scenario and this appears to be one of the most important reasons responsible for lack of a significant growth in the total area under rainfed pulses. Monsoon aberration often leads to widening of gap between demand and supply and resulting price rise brings focus on the research and developmental issues of pulses. Annual production of ~20 mt pulses during 2013-2014 and lowering of this figure in subsequent years due to deficient rains over a large part of the country have proved that we have technology for increasing pulse production but it requires favour of rain God.

Mungbean and urdbean being short duration crops are considered important for increasing area and production of pulses as they can fit easily between two crops of a suitable cropping system. Further their incorporation into cereal dominant cropping systems is essential to maintain the sustainability of the system. However, like other pulses these crops also face a number of biotic constrains and of them yellow mosaic disease (YMD) is most prevalent and known to cause serious damage to their productivity, often rendering the crops totally unproductive.

During more than five decades since YMD was first reported, researchers have worked on various aspects of this potentially damaging disease. Development of many YMD resistant varieties with reduced maturity period has resulted in reviving the popularization of these crops and increasing area under their cultivation. However, reports of inconsistency in stability and degree of resistance keeps researchers busy in improving the resistance and looking for new sources of resistance. Of late, progress has been made in understanding the diversity in YMD causing viruses but much still remains to be investigated. Outbreaks of whitefly, the vector of YMD in different parts of the country during last two years has brought back focus on the need to understand virus-vector-host interactions and diversity in vector population with special reference to its spatial and temporal buildup. At ICAR-Indian Institute of Pulses Research, Kanpur, progress in diagnostics for YMD causing viruses has been worth mentioning. I am happy that Drs. Naimuddin and Mohd. Akram have come up with this publication on various aspects of YMD. I am confident that this publication will be useful to the researchers, extension personnel, farmers, students and teachers and help in mitigating the production losses caused by YMD in pulse crops.



(N.P. Singh)
Director

Preface

Yellow mosaic disease (YMD) is one of the most damaging diseases affecting pulse crops especially mungbean and urdbean. Though YMD has always been considered as an important biotic constrains to mungbean and urdbean production, during last two-three years it assumed epiphytotic proportion attracting the attention of all those involved in research and development related to pulse crops. Considering this and the year 2016 being the “International Year of Pulses-IYP-2016”, it was thought to bring out a publication on YMD of pulses. The bulletin 'Yellow Mosaic Disease of Pulse Crops: Diagnosis and Management' is an attempt to compile information on various aspects of YMD. This is aimed at providing better understanding of YMD to the scientific community, students and pulse growers. It contains reviews on most of the issues pertaining to YMD *viz*., history and distribution, symptomatology, transmission, host range, causal viruses and their nomenclature, disease cycle, host plant resistance, inheritance of resistance, genome and genome organization of the viruses involved in YMD and available management options.

Since more than one virus are known to be associated with YMD in different pulse crops, their distinction based on symptoms or host reaction is not possible. PCR based diagnosis protocols for detection, identification and differentiation of YMD causing viruses developed at ICAR-IIPR, Kanpur have also been elaborated.

Authors are hopeful that this publication will be a ready reference for various aspects of YMD in pulses.

Authors

Contents

Introduction

The yellow mosaic disease (YMD) is known to affect a number of legumes in the country. Symptoms of the disease are broadly similar in all the host plants and hence it is quite often taken as caused by the same virus. However, in the last decade rapid advancement has been made towards understanding of viruses involved in YMD. At present four distinct viruses *viz*., *Mungbean yellow mosaic India virus (*MYMIV*), Mungbean yellow mosaic virus (*MYMV*), Horsegram yellow mosaic virus (*HgYMV*), Dolichos yellow mosaic virus (*DoYM) are known to cause YMD in different leguminous crops and are collectively called legume yellow mosaic viruses (LYMVs) (Qazi *et al.,* 2007). YMD is known to occur in mungbean (*Vigna radiata*), urdbean (*V. mungo*), cowpea (*V. unguiculata*), soybean (*Glycine max*), pigeonpea (*Cajanus cajan*), rajmash (*Phaseolus vulgaris*), dolichos (*Dolichos lablab*), mothbean (*V. aconitifolia*) and horsegram (*Macrotyloma uniflorum*), however, the worst affected crops are mungbean, urdbean, cowpea, mothbean and soybean.

Mungbean and urdbean also known as greengram and blackgram, respectively are important pulse crops grown throughout India. These two legumes along with other legumes serve as an important source of protein to a large portion of the Indian population that traditionally practices vegetarianism. Pulse crops are grown round the year under different agroclimatic conditions. Mungbean and urdbean are however, primarily cultivated as *kharif* (rainy season) crop, occupying about 25% of the total area under all pulse crops. With the development of short duration varieties, these crops have been successfully introduced as spring/summer crops in northern plains. In peninsular India, these crops are cultivated not only as *kharif* crop but also as *rabi* and summer crops. Cultivation of these crops especially of urdbean in rice fallows in many parts of South India and parts of Orissa has resulted in the increased contribution of these crops to the national pulse basket.

In India mungbean occupied 3.38 million hectares in 2013-14 with a production of 1.61 m/t and a productivity of 474 kg/h. Urdbean occupied 3.06 m hectares with a total production of 1.70 million tonnes and a productivity of 535 kg/hectare. These two crops are known to be affected by yellow mosaic disease that is a single most important biotic constraint to the production of these crops in the country.

History of yellow mosaic disease

History of yellow mosaic disease (YMD) goes back to the pre-partition times of the country. YMD was first reported in cowpea in Lyllpur (presently Faisalabad, Pakistan) by Vasudeva (1942). Soon after, it was reported from Pune in western India on *Phaseolus lunatus* and *Dolichos lablab* (Capoor and Varma, 1948, 1950). Later, a similar yellow mosaic disease was first reported in mungbean by Nariani (1960) who named the putative causal agent as MYMV. A similar disease of mungbean was later reported from Pakistan by Ahmad and Harwood (1973). At present YMD is a major constraint to the production of most of the major legume crops in Indian sub-continent (Qazi

et al., 2007). YMD has been reported in urdbean (Williams *et al*., 1968), mungbean (Nene, 1973), pigeonpea (Williams *et al*., 1968, Nene, 1972), horsegram (Muniyappa *et al*., 1975) and Frenchbean (Singh, 1979). YMD in accessions of wild *Vigna* has also been described recently (Naimuddin *et al*., 2011a,b,c, Gautam *et al*., 2014).

Symptoms

As the name suggests, the disease is characterised by yellow mosaic symptoms in leaves of affected plants. Nariani (1960) and Williams *et al.* (1968) described symptoms of yellow mosaic disease on mungbean and urdbean, respectively. Symptoms first appear as small yellow flecks in young leaves. Subsequently emerging leaves exhibit more conspicuous and irregular yellow and green patches alternating each other. Leaf size is generally not much affected. In highly susceptible genotypes however affected plants bear few pods that are smaller in size and deformed. Yellow

Fig. 1. Yellow mosaic symptoms in legume crops. A=Soybean, B=Rajmash, C=Mungbean, D=Horsegram, E=Urdbean, F=Dolichos, G=Cowpea, H=Pigeonpea, I=Wild *Vigna*

spots may also be observed on such pods. Seeds developing in such pods may also show yellow spots on the green seed coat. In urdbean two types of symptoms *viz*., yellow mottle and necrotic mottle were considered to be caused either by different strains of the same virus or by different viruses. However, Nair *et al.* (1974a) clarified that these two types of symptoms were in fact due to the varied reactions to different genotypes to the same virus isolate. More or less similar types of symptoms of YMD are present in soybean, cowpea, dolichos, horsegram and other leguminous crops (Fig. 1).

Occurrence and distribution

Yellow mosaic disease first reported on munbean by Nariani (1960) and on urdbnean by Williams *et al*. (1968) is now known to occur throughout the country wherever these crops are

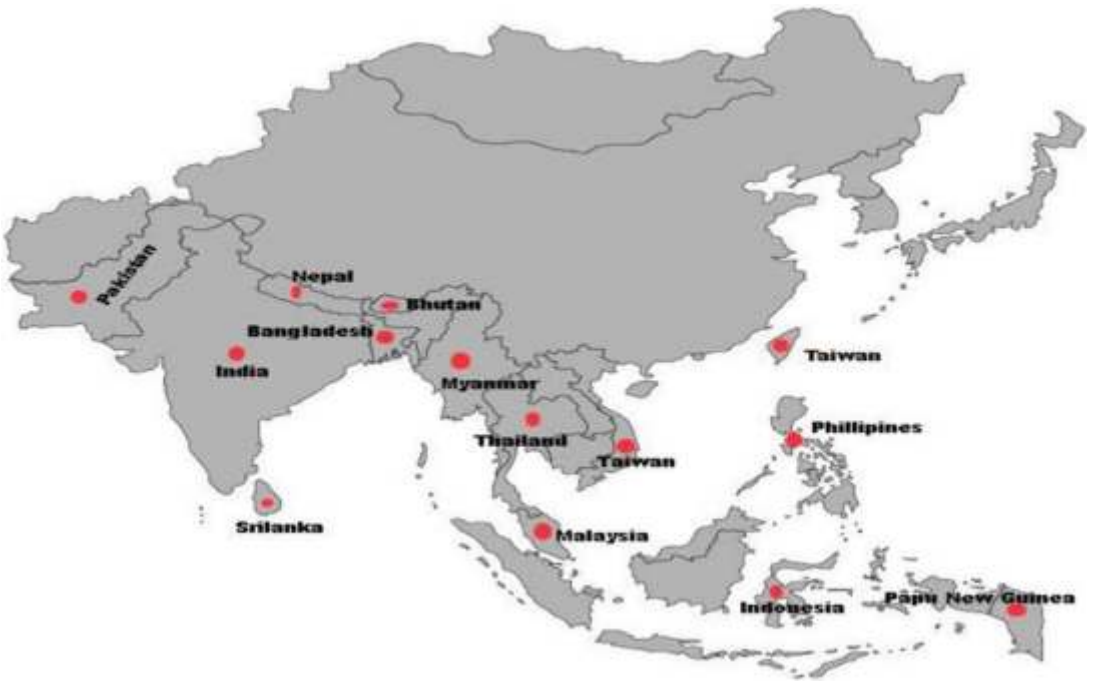


Fig. 2. Distribution map of YMD in different countries (Karthikeyan *et al*., 2014)

grown. It often assumes epiphytotic proportions in northern plains and central and South zones of the country. It is reported to occur in Uttar Pradesh, Uttarakhand, Haryana, Punjab, Himachal Pradesh, Delhi, Madhya Pradesh, Rajasthan, Chhattisgarh, Bihar, Jharkhand, Orissa, Andhra Pradesh, Gujarat, Karnataka, Kerala and Tamil Nadu. YMD of mungbean and urdbean is reported to occur only in Asian countries *viz*., India, Sri Lanka, Bangladesh, Philippines, Pakistan, Myanmar, Thailand, Nepal, Indonesia, Malaysia, Taiwan (Fig. 2) (Karthikeyan *et al*., 2014) and is

considered to be a potential threat to the cultivation of not only mungbean and urdbean but also of soybean and cowpea particularly in Indian sub-continent (Varma and Malathi, 2003).

Yield loss

Yield losses caused by the disease depend up on the stage at which crop is infected (Singh *et al.,* 1982, Gill and Singh, 1999) and may vary from 10-100% (Chenulu *et al.*, 1979, Marimuthu *et al.,* 1981, Nene 1972). YMD intensity of 25% and above influences pod formation and yield in urdbean (Gurha *et al*., 1982, Singh *et al*., 1982). YMD adversely affects all the three yield contributing parameters viz., pods/ plant, seeds/pod and 1000 seed wt in both mungbean and urdbean. Infection adversely affects the colour, texture and size of the seeds also (Vohra and Beniwal, 1979). Often early infected plants suffer more than those infected in later stages of growth (Singh *et al*., 1982). In mungbean, early infection at 20 days after sowing resulted in 62.9% reduction in number of pods and 83.9% reduction in yield as compared to plants in which symptoms appeared at 30-40 days after sowing (Ahmad, 1991). The yield reduction was maximum (69-78%) in mungbean when plants were infected up to 2 weeks after germination (Sharma and Varma, 1982). Per cent reduction in grain yield of mungbean plants infected at 30, 40 and 50 days after sowing was 57.6, 44.3 and 36.5, respectively (Thakur and Agrawal, 1991). Yield losses in plants infected after 60 days are insignificant (Vohra and Beniwal, 1979).

Fig. 3. High incidence of YMD in urdbean and mungbean

Nair and Nene (1974) studied the relation of age of the plant at which it is infected (YMD) and yield of the affected urdbean. The yield loss was not significant if the plants are infected in the 8th week or later. Virus infection reduced number of pods/plant and it was responsible for the overall yield reduction rather than the seeds/pod.

In addition to yield attributing characters, YMD is also reported to reduce height and fresh weight of mungbean and urdbean plants upto 38.2% and 28.5%, respectively. Shape, size and appearance of pods and seeds of diseased plants are considerably distorted although seed germination remains unaffected (Chand and Varma, 1980, 1983) but is delayed in seeds produced from plants infected 10 days after planting (Vohra and Beniwal, 1979). Khattak *et al.* (2000) found decrease in the grain yield and other yield attributing characters not to be significantly correlated with the YMD incidence. It supports the view that it is the severity of the infection rather than the incidence that influences the level of adverse effect on the yield contributing parameters. In monetary terms, it is estimated to cause an annual loss of over US$ 300 millions in mungbean, urdbean and soybean (Varma *et al*., 1992). It may be concluded that the extent of yield loss depends on age of the plant at which it gets infected and on the level of disease severity. In YMD susceptible variety, if the crop is affected at an early stage of growth (Fig. 3), complete yield loss is not uncommon.

Causal Viruses

Since the first description of the disease (YMD) in mungbean and urdbean, the causal virus of the disease was always considered whitefly transmitted. It was however, Honda *et al.* (1983) who on the basis of electron microscopic observations of purified virus preparations and leaf dip preparations provided evidence that the MYMV had geminate particles. Infectivity of purified virus was also proved in mungbean seedlings. These finding established that the causal agent of YMD in mungbean is in fact a geminivirus. Muniyappa *et al*. (1987) described the association of geminate particles in YMD infected plants of mungbean, soybean, lima bean, french bean, groundnut and bambarra ground nut. Presence of similar geminate particles was also shown in plants inoculated with MYMV/MYMIV isolates from blackgram, cowpea and pigeonpea by immunosorbent electron microscopy using polyclonal antibody to *Squash leaf curl virus* (Srivastava, 1989, Varma *et al*., 1992). Association of geminivirus-like particles with yellow mosaic disease of *Dolichos lablab* has also been demonstrated (Raj *et al*., 1989). Presence of geminate particles and their reaction to polyclonal antibodies to other whitefly transmitted geminiviruses (WTGs) further established that the aetiological agent of YMD not only in mungbean and urdbean but also in other legumes is a whitefly transmitted geminivirus, which is now recognized as the species of the genus *Begomovirus* under the family *Geminiviridae*.

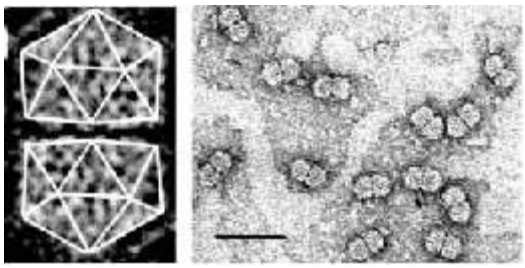


Fig. 4. Geminate particles of a begomovirus

At present, four species of begomoviruses, *Mungbean yellow mosaic India virus* (MYMIV), *Mungbean yellow mosaic virus* (MYMV), *Horsegram yellow mosaic virus* (HgYMV), *Dolichos yellow mosaic virus* (DoYMV) are known to cause YMD in various leguminous crops (Qazi *et al*., 2007, Ilyas *et al.,* 2010). Three of them *viz*., MYMIV, MYMV and HgYMV are known to be involved in causing YMD in mungbean and urdbean in India (Malathi and John, 2008). Virus particles of these viruses are isometric and geminate in morphology (Fig. 4). Particles are 18-30 nm in size.

Nomenclature of causal viruses

Nariani (1960) named the putative virus causing YMD in mungbean as MYMV. For decades MYMV was considered as the causal agent of YMD in many pulse crops. The genome of the Thailand isolate of MYMV was characterized by Moringa *et al.* (1993). Soon after, genome of the virus causing YMD in blackgram in North India was also characterized (Verma *et al.,* 1991, Mandal *et al.,* 1997). Genome sequence data of North Indian isolate of MYMV was found to have <89% similarity *i.e*., the begomovirus species demarcation limit (Fauquet *et al*., 2008) with the

Thailand isolate of MYMV and hence the former was considered as a distinct species and named as *Mungbean yellow mosaic India virus* (MYMIV). Subsequently, the virus causing yellow mosaic disease in horsegram was characterized and it also came out to be a distinct species differing from both MYMIV and MYMV and has been named as *Horsegram yellow mosaic virus* (Muniyappa *et al*., 1987, Baranbas *et al*., 2010). The virus causing yellow mosaic disease in dolichos has also been shown to be a distinct begomovirus species and is designated as *Dolichos yellow mosaic virus* (DoYMV) (Maruthi *et al*., 2006, Akram *et al.,* 2015). DoYMV is generally known to be confined to dolichos bean only. However, yellow mosaic disease of dolichos has been shown to be caused by MYMIV in Varanasi (Singh *et al.,* 2006) and a mixed infection of DoYMV and MYMIV has been detected in cowpea (Naimuddin and Akram, 2010).

Disease Transmission

In mungbean and urdbean, YMD appears within 2 weeks after sowing. This gives an impression as if the disease is transmitted through seed or some factors present in the soil. Nair (1971) was the first to rule out transmission of YMD through seed or soil. He also studied sap transmission of the causal virus of YMD and based on the results of inoculations of a large number of leguminous plants as well as commonly used test plants ruled out the mechanical sap inoculation of the causal virus of the YMD in mungbean and urdbean.

Vector

Nariani (1960) established that the YMD of mungbean is transmitted by whitefly *Bemisia tabaci* Genn. (Fig. 5). The extensive work on host-virus-vector relationship was carried out at Pantnagar in North India by Nair (1971) and Rathi (1972). Murugesan and Chelliah (1977) have also investigated virus-vector relationship. The causal virus of YMD in mungbean and urdbean at that time was named as *Mungbean yellow mosaic virus* and hence in the following text this name is used though at present we know that the virus causing YMD in mungbean and urdbean at Pantnagar is *Mungbean yellow mosaic India virus* (MYMIV). The results of the extensive studies by Nair (1971) and Rathi (1972) are abstracted as under:

Fig. 5. Adult whitefly

- Whitefly may acquire MYMV/MYMIV within 15 minutes of acquisition feeding period.
- Incubation period (of virus in vector) is four hours.
- Whitefly requires a minimum of 15 minutes (inoculation feeding period) to transmit the virus to healthy host plant.
- Starvation (of whitefly) before acquisition and inoculation feeding increases efficiency of acquisition and inoculation, however, the starvation has more pronounced effect on the acquisition feeding than on inoculation feeding.
- A single whitefly can transmit the virus.
- Female whitefly is more efficient in transmitting the virus and may retain it for up to 10 days. Males are however less efficient and generally retain the virus up to 1 day.
- MYMV is not transmitted to the offspring of the viruliferous whitefly indicating no transovarial transmission of MYMV/MYMIV.

PCR based protocols have also been employed to ascertain the identity of the virus (MYMV) in the host and vector in host-vector-virus relationship studies (Govindan *et al*., 2014).

Although in pulse crops whitefly is known to occur as sucking insect and not as a major pest.

In fact, it is known more for being the vector of YMD rather than as a pest of pulse crops. The outbreak of whitefly in cotton and many other crops during the year 2015 has generated interest in the whitefly as a pest of agricultural importance. Therefore, it would not be out of place to add here some information about the white fly.

Bemisia tabaci Genn. (Hemiptera: Aleyrodidae), the vector of yellow mosaic disease causing viruses is a small fly with a delicate and light yellow coloured body with two pairs of white wings. The adults are around 1mm in size. Females are slightly larger than males (McAuslane, 2009, Chu *et al*., 2003). It is polyphagous insect and has an extremely wide host range attacking more than 500 species of plants from 63 plant families (Greathead, 1986, Mound and Halsey, 1978). It is widespread in India. *B. tabaci* has many common names such as tobacco whitefly or cassava whitefly, cotton whitefly, silver leaf whitefly, *etc*. In India, *B. tabaci* is known to infest many important crops mostly those cultivated during warm season, like - cotton, tobacco, cucumber, gourds, eggplant, beans, cowpea, urdbean, mungbean, soybean, dolichos, luffa, pumpkin, sweetpotato, tomato, watermelon, okra, chilli, cassava, *etc*. Many ornamental plants and weeds like- Chinese rose, hollyhock, *Eclipta* spp., Calendula, *Coccinia* sp., *Euphorbia, Ipomoea* spp., *Malva* sp., *Hibiscus* spp., sowthistle, *Sida* spp., *Ageratum*, *Xanthium* sp*., etc*. are also reported as host of whitefly. Whitefly feeds generally on lower surface of leaves. It completes its life cycle in less than 2 weeks to more than ten weeks depending upon temperature and plant host. Females usually lay eggs between 200 and 400. There are six life stages *viz*., egg, four nymphal stages and adult. Temperature around 27-30^{0}C and humidity around 70% favour development of this insect. Overlapping generations occur throughout the year.

Whitefly infestation in crops like cotton, okra, tomato, *etc*., where its population build up is very high adversely affects their growth and yield. While feeding on the plants, whitefly excretes 'honeydew', a carbohydrates rich material that encourages development of molds and leads to the fungal sooty molds giving the affected plant part a blackened appearance. Intensive agriculture can be said to be the most important human intervention that appears to have helped whitefly survive, reproduce, spread and proliferate. Although in case of pulse crops, whitefly hardly assumes status of a pest, but it is known to thrive in these crops. In fact infestation of whitefly in pulse crops becomes more important because it is the vector of yellow mosaic disease (YMD) causing viruses and is responsible for spread of the disease. Whitefly build up is more in urdbean than in mungbean.

B. tabaci is considered as a species complex containing numerous genetic and biological variants undergoing continuous evolutionary changes (Brown, 2000, Perring, 2001). Of many biotypes recognized (Lisha *et al*., 2003, Brown, 2007, Prasanna *et al*., 2015), the 'B' biotype (silver leaf whitefly) due to its widest distribution, attributed to its ability to colonize many diverse plant hosts and remarkable ability to transmit a number of geminiviral diseases is considered a potential threat to many crops. This biotype said to have originated in Middle East region has been reported from India also (Banks *et al*., 2001).

Review of literature reveals that there are many reports on YMD management by targeting the vector of the disease (whitefly) through chemicals. However, as far as management of whitefly

in pulse crop is concerned, there is no management strategy exclusively targeting the whitefly. In fact the insecticides used against whitefly target other insects like thrips, jassids, *etc*., also. And it is probably because of this reason that the use of insecticides against whitefly results in increased yield but this increase cannot be attributed exclusively to the reduction in whitefly population in the crop or to the reduction in yellow mosaic disease.

Seed transmission

In YMD affected plants of a susceptible mungbean and urdbean genotype, yellow patches may often be seen on the pods. Seeds in such pods also show yellow patches on the seed coat. This led us to investigate whether virus (MYMV/MYMIV) is carried through seed. Seeds showing yellow pathches on the seed coat were collected from YMD affected mungbean genotype T44 at ICAR-IIPR, Kanpur. Such seeds were subjected to PCR tests using MYMIV specific primers. Results indicated that the virus (MYMIV) was present in the seeds (Fig. 6). However, the virus could not be detected in seedlings raised from MYMIV positive seeds (Fig. 7). Our findings indicate that the virus is lost during the process of germination and is not transmitted through seeds. In a recent publication Kothandaraman *et al*. (2016) concluded that MYMV is seed borne in urdbean. The seedlings from MYMV positive seeds however did not show any symptoms of YMD though the virus (MYMV) was detected in 32 % of tested seedlings. They however, did not explain the absence of symptoms in these seedlings. Also whitefly transmission from PCR positive symptomless seedlings was not demonstrated. Nevertheless they concluded MYMV to be seed borne in urdbean. It is well accepted that a virus is said to be seed borne only when it is carried to seedlings and serves as source of primary inoculum. This publication does not provide answer to it and hence this can not be accepted as a case of seed borne virus simply due to the presence of the virus in the seed.

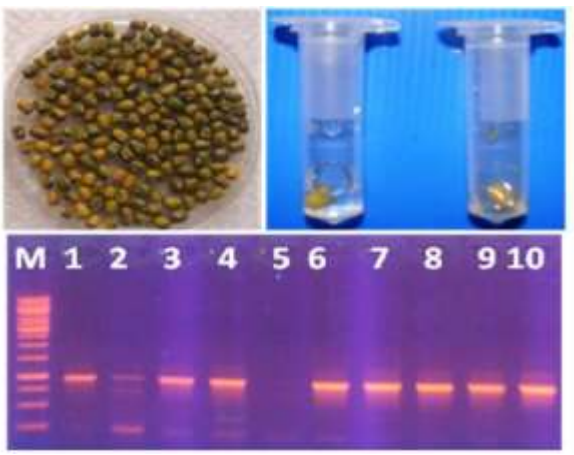


Fig. 6. Detection of MYMIV in different part of plants

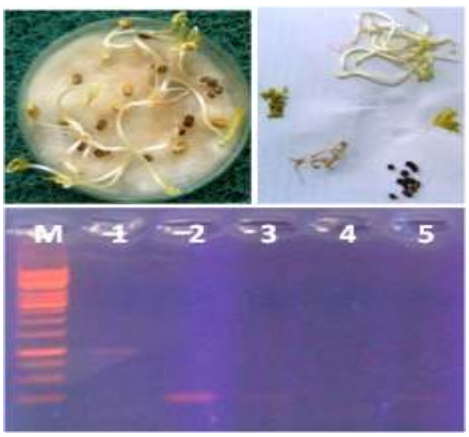


Fig. 7. Detection of MYMIV in different part of 72 hrs old seedling

Host-Virus Interaction

Host range

Mothbean (*Vigna aconitifolia*), soybean (*Glycine max*), Phasemy bean (*Phaseolus lathyroides*), horsegram (*Macrotyloma uniflorum*), black tapery bean (*P. acutifolius*) were reported as host of the virus causing YMD in mungbean and urdbean (Nariani, 1960). Later, pigeonpea, common bean, and weeds namely, *Brachiara ramosa, Eclipta alba, Cosmos bipinnatus* and *Xanthium strumarium* were also reported as host of MYMV (Nene, 1972). These reports were based on whitefly transmission tests and symptoms. Based on symptoms, 75 weed hosts were reported to be the offseason hosts and reservoirs of *Mungbean yellow mosaic virus* (Raychaudhari *et al*., 1977). Based on genome characterization, the causal viruses of YMD in many leguminous hosts have been shown to be *Mungbean yellow mosaic virus* or *Mungbean yellow mosaic India virus* or *Horsegram yellow mosaic virus* or *Dolichos yellow mosaic virus.* In fact the identity of the virus in the weeds reported to be the hosts of legume yellow mosaic viruses has not been proved unequivocally. At IIPR, Kanpur we have for the first time found MYMIV infection in yellow vein affected weed, *Ageratum conyzoides* using MYMIV specific primers in PCR tests (Naimuddin *et al*., 2014).

Disease cycle

Since a number of cultivated plant species and weeds are hosts of MYMV, availability of inoculums throughout the year can be explained. However, the differences in the prevalence and severity of the disease may be attributed not only to the prevalence of alternate/collateral hosts (of LYMVs) in the vicinity of the crop, but also to the availability of vector, whitefly also. The vector is polyphagous but its population is generally influenced by weather parameters.

Under North Indian conditions, mungbean and urdbean are cultivated in spring/summer and *kharif* seasons. Long duration pigeonpea is also a host of MYMV/MYMV and remains in the fields till April. The YMD affected pigeonpea may provide primary inoculums of MYMV/MYMIV for spring/summer sown crop. Besides, weeds hosts may also act as source of primary inoculums of MYMV/MYMIV for spring/summer crops. Beans grown as winter season also get MYMV/MYMIV infection and may serve as a source of primary inoculum for spring/summer crop. Once the virus is introduced into the spring/summer sown crops, it perpetuates and is carried over to *kharif* sown crops of not only mungbean and urdbean but also to *kharif* sown pigeonpea. Virus may also be introduced in to the *kharif* crop from weed hosts through whitefly vector (Fig. 8). Weeds reported to be the hosts of the yellow mosaic virus are described under the heading of host range. They may be playing a reservoir of yellow mosaic disease causing viruses. Howevere,

extensive studies are required for characterizing geminiviruses in weeds so as weeds harbouring the crop infecting viruses are identified. This will indeed help devising strategies for the management of YMD of pulse crops.

Fig. 8. Yellow mosaic disease cycle

Since pulse crops are sown in different seasons, influence of environmental factors on disease development is very obvious. Though environmental factors appear to have no effect on the disease *per se*, but the vector population and its build up, availability of primary source of inoculum may be influenced by these factors. It is because of these reasons that the temporal and spatial variations on disease (YMD) are often reported. Effect of sowing date is more pronounced in South as the crop is raised throughout the year. Maximum incidence of YMD was observed in mungbean sown from March to May, while the crop sown from July to December had low incidence of disease in Tamil Nadu (Murugesan and Chelliah, 1977). Sharma and Varma (1982) found incidence of the YMD less in summer (April-June) than in the rainy season (July-October) mungbean crops under Delhi conditions, whereas Singh and Gurha (1994) reported that under North Indian conditions incidence of YMD was more in summer sown (March-April) crops than in *kharif* (July-August) sown crops. At present, this trend is however not observed. For last many years our experience is that YMD is more in *kharif* sown crop than in spring/summer crop.

Disease rating scale

For identification of host plant resistance a system is required to record the host reaction of test genotypes. In eighties, disease rating scales used were based on either per cent disease incidence (Chuadhary *et al*., 1981) or the crop stage at the time of infection and symptom severity (Singh *et al*., 1987). Subsequently, many researchers have proposed scales for rating disease reaction of mungbean and urdbean genotypes against YMD (Mayee and Datar, 1986, Singh *et al*., 1987, Singh *et al*., 2004). Making use of all these scales, a system has been developed under All India Coordinated Research Project on MULLaRP and is being used to record the disease reaction of mungbean and urdbean genotypes against YMD at AICRP (MULLaRP) centres across the country (Table 1).

Table 1 : Disease rating scale for grading the resistance of mungbean and urdbean genotypes.

Rating scale	Description	Reaction category
1	No visible symptoms on leaves or very minute yellow specks on leaves	Resistant (R)
2	Small yellow specks with restricted spread covering 0.1 to 5 % leaf area	
3	Yellow mottling of leaves covering 5.1 to 10 % leaf area	Moderately resistant (MR)
4	Yellow mottling of leaves covering 10.1 to 15 % leaf area	
5	Yellow mottling and discoloration of 15.1 to 30 % leaf area	Moderately susceptible (MS)
6	Yellow discoloration of 30.1 to 50 % leaf area	Susceptible (S)
7	Pronounced yellow mottling and discoloration of leaves and pods, reduction in leaf size and stunting of plants covering 50.1 to 75 % foliage	
8	Severe yellow discoloration of leaves covering 75.1 to 90 % of foliage, stunting of plants and reduction in pod size	Highly susceptible (HS)
9	Severe yellow discoloration of entire leaves covering above 90.1 % of foliage, stunting of plants and no pod formation	

Host plant resistance

Fig. 9. Symptoms of YMD in urdbean during screening for resistance in field

Infector row technique is commonly employed to screen mungbean and urdbean genotypes against YMD under field conditions. A highly susceptible genotype is often used as infector row and is planted between two rows of test genotypes (Fig. 9). A number of workers have identified sources of resistance against YMD in mungbean and urdbean. However, in very few of them, identity of the causal virus of YMD have been unequivocally proved. Recently, infectious clones of MYMV (Sudha *et al*., 2013b) and MYMIV (Bag *et al*., 2014) have been employed to ascertain the resistance of mungbean and urdbean genotypes, respectively.

Many other workers have reported field resistance of urdbean genotypes from different parts of the country (Table 2). Singh and Awasthi (2004) evaluated 98 germplasm accessions of urdbean against YMD and found thirteen genotypes (HPU-227, UH-86-40, IPU-94-2, IPU-981, KU-300, IPU-94-1, ND-6, Pant U-19, K-11686, PDU-2, PLU-800, PLU-603 and NDU-7) as resistant. Basandrai *et al* (2003) reported six urdbean cultivars (LU 1129, PLU 117, PLU 1077, UG 218, UG 786 and UG 1120) and Gupta (2003) reported 13 urdbean lines (KU 96, TU 98-14, KU 96-1, UG 774, UG 737, UPU 97-10, KU 315, NP 6, PLU 96-8, PLU 44, PLU 131, PLU 463 and KU 96-8) as resistant to YMD. Biswas and Verma (2001) found urdbean genotypes DPU 84-14, DPU 102, UG 218, UG 389, UL 257 and UL 310 as resistant to highly resistant to MYMIV under both field and glasshouse conditions. Arutkani *et al.* (1999) screened 52 urdbean genotypes against YMD under

field as well as through whitefly inoculations and found PDU 102 as resistant. Panigrahi and Baisakh (2013) identified urdbean lines PDU 1-15, PDU 1-9 and Sarala-13 as resistant to YMD in Orissa.

Sudha *et al.* (2013b) screened 78 mungbean genotypes against MYMV and found 28 as resistant in field screening. Agro-inoculation of these genotypes with infectious cloned viral DNA of two strains (VA 221 and VA 239) revealed only three genotypes (ML 1108, KMG 189 and SP 84) to be resistant to VA 221 strain, while only one genotype ML 818 showed resistance to strain VA 239, but it was susceptible to strain VA 221. These findings highlight the importance of confirming resistance to YMD using infectious clones of the virus. Bag *et al.* (2014) screened 344 accessions of urdbean. Of the eight genotypes found resistant in field screening and tested using whitefly inoculation, only four accessions *viz.*, IC144901, IC001572, IC011613 and IC485638 showed resistant reaction to MYMIV. Resistance of these four genotypes was further confirmed through agro-inoculation of the infectious cloned viral DNA under controlled conditions.

Many reports of field screening are available and are summarized in Table 2. Manivannan *et al.* (2001) screened 551 green gram genotypes and rated EC300072, K141, LGG424B and LM108B as resistant to YMD. Singh and Awasthi (2009) screened 84 genotypes of mungbean and found ten (GM-13, GM-21, GM-125, GM-138, M-125, GM-164, GM-850, GM-899, M-126 and PLM-214) resistant to YMD. Kumar *et al.* (2006) screened 50 genotypes of mungbean and found NDM 2, ML 337, and PDM 90-1 to be resistant to YMD. Biswas *et al.* (2005) identified mungbean genotypes MGG 443, ML 5, ML 337, ML 459, ML 513, ML 610, P 9271 and P 9272 as resistant to five variants of MYMIV namely Pp1, Bg3, MbD, MoL and MbS through whitefly inoculation. Chhabra *et al.* (1979) reported mungbean genotypes ML 1, ML 3, ML 5 and LM 170 as resistant to YMD in Punjab.

YMD is also prevalent in neighbouring Bangladesh, Nepal and Pakistan and sources of resistance have been reported from there also (Munawwar *et al.,* (2014). Karim *et al.* (2010) found mungbean genotype BMX-97008-8 as resistant to YMD in Bangladesh. Ilyas *et al.* (2007) screened 101 genotypes of mungbean and found genotypes CMG-523, CM-mung-97, 96002, 98010, C10/95-3-21 and C1/95-3-45 as resistant to YMD in Pakistan. In Nepal, Neupane *et al.* (2004) found mungbean genotypes VC 6372 (45-8-1), VC 6370 (30-65), and VC 6368 (46-7-2) as resistant to YMD.

Table 2: Summary of the sources of resistance in mungbean and urdbean reported by different workers from different countries

Country	Crop	Number of genotypes screened	YMD resistant genotypes	Reference	Remarks
India	Urdbean	63	Cultivars 4-5-2 and UL29	Chhabra and Kooner (1981)	Used inoculation through whitefly vector
India	Urdbean	1565	UG 234, UG 248, UG259, UG 261, UG 265, UG 361, Mash 1-1 and UL169	Singh *et al.* (1987)	
India	Urdbean	64	KU39, JKU64, JKU9, JKU33, JKU28, JKU33, JKU50, JKU12, JKU9 and JKU1	Acharya *et al.* (1993)	
India	Urdbean	52	PDU 102	Arutkani *et al.* (1999)	
India	Urdbean	15	DPU 84-14, 'DPU 102, UG 218, UG 389, UL 257 and UL 310	Biswas and Varma (2001)	Used field as well as Whitefly inoculations
Pakistan	Urdbean	132	53R	Bashir and Zubair (1992)	Pakistan
Pakistan	Urdbean	118	25 genotypes were identified as resistant	Ahmad (1975)	
India	Urdbean	38	KU 96, TU 98-14, KU 96-1, UG 774, UG 737, UPU 97-10, KU 315, NP 6, PLU 96-8, PLU 44, PLU 131, PLU 463 and KU 96-8.	Gupta (2003)	
India	Urdbean	18	IPU-95-13	Sharma *et al.* (2004)	
India	Urdbean	98	HPU-227, UH-86-40, IPU-94-2, IPU-981, KU-300, IPU-94-1, ND-6, Pant U-19, K-11686, PDU-2, PLU-800, PLU-603 and NDU-7	Singh and Awasthi (2004)	
Pakistan	Urdbean	134	45014, 45015, 45016, 45021, 45022, 45024, 45028, 45030, 45032, 45036, 45047, 45049, 45062, 45064, 45083, 45090, 45091, 45092,	Bashir *et al* (2006)	

Country	Crop	Number of genotypes screened	YMD resistant genotypes	Reference	Remarks
			45093, 45094, 45096, 45097, 45101, 45114, 45116, 45126, 45134, 45138, 45139, 45141, 45157, 45159, 45162, 45163, 45164, 45173, 45174, 45235, 45336, 45339, 45340, 45342, 45788 (43 lines highly resistant)		
India	Urdbean	12	Uttara, IPU 2-43 and VBG 4-008	Gopalaswamy *et al.* (2012)	
India	Urdbean	62	PU 01-415, IPU 94-1, IPU 94-2, IPU 96-1,IPU 99-16, IPU 99-192, PLU 3435, BG 369, HPL 180, PDU 14, PGRU 95018, YS/RC 238, KUG 479, *Mash* 114, *Mash* 338 and *Mash* 1-1	Kumar and Bal (2012)	
India	Urdbean		PDU 1-15, PDU 1-9 and Sarala-13 (mutant lines)	Panigrahi and Baisakh (2013)	
India	Urdbean	56	22 genotypes	Obaiah *et al.* (2013)	
India	Urdbean	344	IC144901, IC001572, IC011613 and IC485638	Bag *et al.* (2014)	Used agroinoculation to confirm the resistance
India	Urdbean	100	NDU 12-1, IPU 10-23, KUG 586, Mash-338, NDU 12-300, PU 09-35, UH 07-06, Uttara, VBG 10-008, and VBN 6, Kopergaon, RUG-44, VBG 09-005, and NDU 11-201	Equbal *et al.* (2015)	
Pakistan	Mungbean	157	None	Ahmad (1975)	
India	Mungbean	28	ML-5, ML 337, ML 405, ML 408, MUM 2, Pusa 8773, MH 85-61, MH 85-	Patel and Srivastava (1990)	

Country	Crop	Number of genotypes screened	YMD resistant genotypes	Reference	Remarks
			91, ML 537, NDM 8813, NDM 88-14, Pant M-3 and UPM 79-11, and VGG3 45		
India	Mungbean	26	ML 395, ML 505 and ML 543	Chhabra and Kooner (1994)	These genotypes were reported as resistant to whitefly also.
India	Mungbean	126	ML267, ML337, ML393, ML395, ML409, ML443, ML452, ML462, ML466, ML473, ML505, ML506, ML512, ML513, ML514, ML517, ML522, ML532, ML535, ML553, ML587, ML591, ML593, ML604, ML605, ML613, MUG225, MUG249, MUG255, MUG262, PDM 54, PDM 84-139, PDM 84-143, PDM 219, Pusa 8731	Singh *et al.* (1996).	
India	Mungbean	551	EC300072, K141, LGG424B, LM108B	Manivannan *et al* (2001)	
India	Mungbean		MGG 443, ML 5, ML 337, ML 459, ML 513, ML 610, P 9271 and P 9272	Biswas *et al.* (2005)	
India	Mungbean		VC 6372 (45-8-1), VC 6370 (30-65), and VC 6368 (46-7-2)	Neupane *et al.* (2004)	
Pakistan	Mungbean	110	85 highly resistant NM-49-9, NM-92, NM-1,	Bashir *et al.* (2006)	
Pakistan	Mungbean	254	Nil	Nadeem *et al,* (2006)	
India	Mungbean	5050	NDM 2, ML 337 and PDM 90-1	Kumar *et al*. (2006)	These genotypes also carried least population of whitefly
India		38	RMM 18, RMM 35, RMM 48, RMG 754, RMG 843, RMG 852, RMG 926, RMG 938 and RMG 951	Khedar *et al.* (2006)	

Country	Crop	Number of genotypes screened	YMD resistant genotypes	Reference	Remarks
Pakistan	Mungbean	101	4CMG-523, CM-mung-97, C10/95-3-21, C1/95-3-45	Ilyas *et al.* (2007)	
India	Mungbean		ML nos. 935, 1165, 1194, 1240, 1252, 1256, 1262, 1265, 1268, 1270, 1278, 1286, 1294, 1296, 1329, 1330 and 1331.	Kaur *et al.* (2007)	These genotypes were also reported to have resistance to Cercospora leaf spot and bacterial leaf blight
India	Mungbean	84	GM-13, GM-21, GM-125, GM-138, M-125, GM-164, GM-850, GM-899, M-126 and PLM-214	Singh and Awasthi (2009)	
India	Mungbean	40	NM 57 and NM 94	Yadav and Brar (2010)	
Bangladesh	Mungbean	1010	BMX-97008-8	Karim *et al.* (2010)	
Pakistan	Mungbean	162	Nil	Akhtar *et al.* (2011)	Used whitefly inoculation and grafting
Pakistan	Mungbean	100	Mungbean lines 014043, 014133, 014249, 014250	Iqbal *et al.* (2011)	
India	Mungbean	136	43 R	Zhimo *et al.* (2013)	
India	Mungbean	78	ML 1108, KMG 189 and SP 84 resistant to MYMV strain VA 221; ML 818 resistant YMV strain VA 239	Sudha *et al.* (2013b)	28 genotypes showing resistance to YMD in the field were subjected to agro-inoculation with two different MYMV strains VA 221 and VA 239.
India	Mungbean		MUM2 and ML5	Pathak and Jhamaria (2004)	
India	Mungbean	35	IPM 2-14 and PDM 139	Suman *et al.* (2015)	

Molecular insight

Factors responsible for replication of MYMIV have been studied. Rouhibakhsh *et al.* (2011) demonstrated that agro-inoculated plants with AV 2 mutants K73R, C86S and the double mutant C84S, C86S increased severity of symptoms compared with the wild type. These mutants caused a 50-fold increase in double-stranded super coiled and single-stranded DNA accumulation, the mutations W2S and H14Q, G15E showed a decrease in double-stranded super coiled and single-stranded viral DNA accumulation. It has been hypothesized that due the alteration of the ratio between open circular and super coiled DNA forms by the AV2 mutants may modulate the

functions of the replication initiation protein (Rouhibakhsh *et al.*, 2011, 2012). The viral constructs having N′ terminal deletion of 75 and 150 amino acids affected systemic spread and pathogenicity in cowpea, mungbean and blackgram plants contrasting to frenchbean which developed symptoms similar to wild type. Assembly of particles and whitefly transmission were not seen in all the mutations (Haq *et al*., 2011). Suyal *et al.* (2013a,b) demonstrated that the involvement of host factors AtMCM2 and RAD51 in MYMIV DNA replication. The interaction of NAC083, a member of NAC transcription factor family, with MYMIV-Rep protein indicated its possible role in MYMIV DNA replication (Suyal *et al.*, 2014).

In order to obtain a critical insight to unravel how *V. mungo* respond to MYMIV a PCR based suppression subtractive hybridization technique was employed to identify genes that exhibit altered expressions. It has been shown that 345 candidate genes expressed differentially in compatible or incompatible reactions. The enhanced expression of transcripts involved in phenylpropanoid and ubiquitin-proteasomal pathways believed to confer resistance against MYMIV (Kundu *et al*., 2015). Such studies were not conducted in mungbean-virus interaction. Attempts have also been made to see the feasibility of developing the transgenic against MYMV in tobacco plants (Sunitha, *et al*., 2012, 2013, Gnanasekaran *et al*., 2015) and soybean (Singh *et al*., 2013b).

Proteomics of mungbean-MYMV interaction was studied through 2D-PAGE using leaf protein extracts at virus stressed flowering phase of uninfected (control) and MYMV-infected susceptible (cv. VBN Gg2), resistant (cv. KMG 189) parents and their susceptible (CM 15-1-1-F2S) and resistant (CM 15-7-13-F2R) F_2 progenies. It has been concluded that the proteins expressed differentially in resistant and susceptible genotypes and also suggested that the proteins of stress/defense and signaling/transport were involved in re-programming the defense mechanism (Cayalvizhi *et al*., 2015).

Inheritance

In case of mungbean, resistance to YMD has been found to be governed by a single recessive gene (Saleem *et al*., 1998). Khattak *et al.* (1999) also worked out the mode of inheritance of resistance to YMD in crosses involving a highly resistant mungbean line (NM 92), a moderate resistant line (ML-5), a tolerant line (6601), 2 moderate susceptible lines (VC 2272 and Pusa Baisakhi) and 4 susceptible lines (VC 1560D, VC 3902A, Berken, and Emerald) and found a single recessive gene to be involved in imparting resistance against YMD. They however did not find any maternal effect in the inheritance of YMD resistance in mungbean. Some recent studies have also shown YMD resistance to be under the control of single recessive gene. Jain *et al.* (2013) studied the progenies of the crosses involving four YMD resistant (Sonamung, KM6 220, KM6 201 and Samrat) and four susceptible mungbean genotypes and Dhole and Reddy (2013) studied the inheritance of resistance to YMD in the progenies and recombinant inbred lines developed from the cross between TM-99-37 (resistant) × Mulmarada (susceptible). Both these studies found susceptibility to YMD dominant over resistance. A single recessive gene was indicated responsible for imparting resistance to YMD. Mishra (2003) also found a single recessive gene responsible for conferring resistance to YMD in mungbean and indicated that the expression of the gene

responsible for YMD resistance is affected by the action of modifying genes present in the nucleoplasm. These modifying genes were the cause of variation in degree of resistance/susceptibility in progenies derived from a single cross and hence development of highly resistant mungbean lines would depend on the accumulation of favourable modifying genes.

In other studies, two recessive genes have been shown to impart resistance to YMD in mungbean (Singh *et al*., 2013a). When one gene is present in the homozygous recessive condition in different plants, it confers MR and MS reactions. When both genes are present together in the homozygous recessive condition, plants produce resistant reactions (R) (Dhole and Reddy, 2012). Shukla *et al*. (1978) using YMD resistant (Tarai Local), moderately resistant (L-80), tolerant (L294-1 and LM-214) and susceptible (Jawahar-45 and G-65) lines of mungbean also demonstrated resistance to YMD under the control of two recessive genes in all the crosses. Verma and Singh (1988) analyzed the disease (YMD) reaction of the progenies of a cross between YMD susceptible (T44) and resistant line (Tarai Local) of mungbean and found that the resistance was under the control of two recessive genes. They also concluded that there was no maternal effect on the control of resistance. The literature indicates divergent conclusions from the inheritance (of YMD) studies in both crops (mungbean and urdbean). The differences in the results may be attributed to the different sources of resistance and differences in the virus strain/isolate. Many studies report control of resistance to YMD to be governed by two recessive genes. The role of individual gene needs to be worked out. Studies on inheritance of resistance in mungbean and urdbean are summarized in the Table 3.

Table 3: Summary of inheritance of resistance to YMV in mungbean and urdbean

Crop	Crosses	Nature of resistance	Reference	Remarks
Mungbean	Tarai Local (resistant), L-80 (moderately resistant), L294-1 and LM-214 (tolerant) and Jawahar-45 and G-65 (susceptible)	Digenic, recessive	Shukla *et al*. (1978)	
Mungbean	Susceptible T44 and the resistant line Tarai Local	Digenic, recessive	Verma and Singh (1988)	The F1 means of the reciprocal crosses were almost identical indicating the absence of maternal effects
Mungbean	PDM 116 (R)x SML 32 (S) PDM 116 (R)x G65	Digenic, recessive	Pal *et al.* (1991)	
Mungbean Cultivated X Wild	Susceptible (T44 and K851)x resistant, wild *V. radiata* var. *sublobata* line PLN15.	Resistance controlled by one dominant and one recessive gene in both resistant parents.	Reddy and Singh (1993)	
Mungbean	crosses involving three resistant (HUM 1, DPM 90-1 and DPM 90-2) and four susceptible (MH 84-1, K 851, China Mung and Kopergaon)	Digenic recessive	Singh and Singh (1996)	

Crop	Crosses	Nature of resistance	Reference	Remarks
Mungbean	Resistant (NM-92 and NM-93)x susceptible (VC-1973A, VC-2254A, VC-2771A and VC-3726A)	Monogenic recessive	Saleem *et al*. (1998)	
Mungbean	Crosses involved resistant (NM2), moderately resistant (ML5), tolerant (6601), moderately susceptible (VC 2272 and Pusa Baisakhi), and 4 susceptible lines (VC 1560D, VC 3902A, Berken and Emerald).	Monogenic recessive	Khattak *et al*. (1999)	No maternal effect
Mungbean	Crosses involved (HUM 1, JM 721, K 861, LGG 450, Pusa Baisakhi, HUM 2 and TARM 1	Monogenic recessive	Mishra (2003)	The expression of the gene responsible for MYMV resistance is affected by the action of modifying genes present in the nucleoplasm. These modifying genes cause variation in the degree of resistance/ susceptibility in progenies derived from a single cross.
Mungbean	susceptible (K 851, BDYR-1 and Pusa Bold-1) and resistant (BDYR-2, ML 818 and ML 682) parents	All 3 crosses showed the presence of recessive genes governing resistance. Duplicate type of gene action.	Ammavasai *et al*. (2004)	
Mungbean	Crosses involved mungbean genotypes -3902A, NM92, VC2272, 6601, Pusa Baisaki, ML-5, Berken, Emerald, VC1560D and NM98	one major recessive gene	Khan *et al* . (2007)	No maternal effect in the inheritance of YMD resistance
Mungbean	6 crosses between resistant and susceptible genotypes of mungbean	2 recessive genes	Dhole and Reddy (2012)	Original to be seen for getting names of the genotypes used in crosses
Mungbean	UPM99-03 (R)xPusa 9531, PM4 (R)xK851(S), PDM139xEC398885, IPM99-125xEC398885	Digenic recessive	Aski *et al*. 2015	Results indicated that MYMIV

Crop	Crosses	Nature of resistance	Reference	Remarks
				resistance is governed by two recessive genes,viz. r1 and r2 segregating independently
Ricebean X mungbean Mungbean X mungbean	Interspecific cross TNAU RED (rice bean) R to YMV × VRM (Gg) 1-S to YMVand intra KMG 189 Mungbean R to YMV × VBN (Gg) 2 S to mYMV	Single recessive gene	Sudha *et al.* (2013a)	
Mungbean	R (ML818 and Satya) x S (SML 32 and Kopergaon)	2 recessive genes	Singh *et al.* (2013a)	
Mungbean	crosses involving five susceptible (KM6 202, LGG 478, KM6 204, PUSA 9871 and K 851) and four resistant (Sonamung, KM6 220, KM6 201 and Samrat) mungbean genotypes	Single recessive gene	Jain *et al.* (2013)	
Mungbean	cross 'TM-99-37' (resistant) × Mulmarada (susceptible)	Single recessive gene	Dhole and Reddy (2013)	
Urdbean	Resistant Pant U19 and Pant U26	Monogenic dominant, but different genes were involved in the 2 cultivars	Kaushal and Singh (1988)	
Urdbean	Crosses involved resistant (BR61, Sel1 and NP21) and susceptible (UL2) lines	2 recessive resistance genes	Verma and Singh (1989)	
Urdbean	Mash 1-1 (R) x and Local Mash (S) Saradhu (R) x Local Mash (S)	Monogenic recessive	Pal *et al.* (1991)	
Urdbean	Cross involved resistant line IW3390 of wild progenitor species *V. mungo* var. silvestris and susceptible lines UL2 and RU2) of urdbean	one dominant and one recessive gene in both resistant parents	Reddy and Singh (1993)	
Urdbean	Crosses involved resistant lines (Pant U-19, UH 81-7 and UG 400) and a susceptible line (HPBU 52)	2 recessive genes	Singh *et al.* (1998)	
Urdbean	Crosses involved resistant (DPU 88-5, DPU 88-1 and UG 400) and susceptible (Barabanki Local) line of urdbean	2 recessive genes	Sirohi *et al.* (2002)	
Urdbean	crosses of black gram involving eight highly resistant cultivars (DPU 88-31, NP 21, PLU 710, PDU 6, IPU 98-8, UPU 85-86, UG 27, and DUS 19) and six susceptible cultivars (PDU 1, IPU 99-182, IPU 99-168, PGRU 95013, UH 80-38, and UH 82-2)	Monogenic dominant	Gupta *et al.* (2005)	
Urdbean	Cross involved DPU 88-31(resistant) × AKU 9904 (susceptible)	Monogenic dominant	Gupta *et al.* (2013)	
Urdbean	Cross involved CO 5 and LBG 623 as susceptible (s) and PU 31, VBN (Bg) 4, VBN (Bg) 6 as resistant (r) genotypes. Co 5 × PU 31, Co 5 × VBN (Bg) 4,	Digenic recessive	Prasad *et al.* (2015)	various types of interaction, i.e. duplicate

Crop	Crosses	Nature of resistance	Reference	Remarks
	× VBN (Bg) 6, LBG 623 × VBN (Bg) 4 and LBG 623 × VBN (Bg) 6			interaction (Co 5 × PU 31 and Co5 × VBN (Bg) 6), complementary interaction (Co 5 × VBN (Bg) 4) and inhibitory interaction (LBG 623 × VBN (Bg) 4 and LBG 623 × VBN (Bg) 6) was reported.
Intrerspecific	Mungbean x urdbean Mungbean x *Vigna* sublovata	Digenic recessive	Pal *et al.* (1991)	
Urdbean Inter sub specific	Urdberan cv. VBN (Bg) 4' (resistant) x *Vigna mungo* var. *silvestris* 22/2 (susceptible)	Monogenic dominant	Vinoth and Jayamani (2014)	

Genome Characterization and Variability

Genome and genome organization

Begomoviruses are categorized into three types based on their genome organization. The begomoviruses causing yellow mosaic disease in different leguminous plants including pulse crops fall in to type I that comprises virus species with bipartite genome, *i.e.*, the genome of these species consists of two molecules of single stranded DNA referred as DNA-A and DNA-B (Fig. 10). In begomoviruses with bipartite genome, DNA-A and DNA-B differ in sequence except for a common region (CR) of 200-250bp that is nearly identical in the genome components of any given virus, but differ between virus species. A unique feature of isolates of MYMIV and MYMV is the divergence in the nucleotide sequence of CR in their DNA-A and DNA-B. Though DNA-A contains all viral information necessary for replication and encapsidation (Rogers *et al*., 1986, Sunter *et al.*, 1987, Townsend *et al*., 1986), but both components are required for infectivity (Hamilton *et al*., 1983, Stanely, 1983).

Bipartite nature of the genome was known for three (MYMIV, MYMV, HgYMV) of the four well known viruses causing YMD in different pulse crops. Till 2007 (Qazi *et al*., 2007) only DNA-A component of the DoYMV genome was known. At IIPR, Kanpur we have discovered DNA-B of the DoYMV (KJ481025) thus confirming genome of DoYMV also to be bipartite (Akram *et al*., 2015). Thus, at present the genome of these four LYMVs is known to be bipartite, *i.e.* consisting of two single stranded circular DNA molecules *viz*., DNA-A and DNA-B each approximately of 2.7 kb in size (Fig. 10).

In bipartite begomoviruses, the genes in DNA-A and DNA-B are separated by an intergenic region (IR) that includes a segment of ~200 nt called the common region (CR), a highly conserved region between both components. All the *cis* elements required for replication are located in the CR, which varies from virus to virus with the exception of a highly conserved ~30 nt long segment with potential to form a stem loop structure, also called as hairpin structure. In LYMVs, there are generally six-seven open reading frames (ORFs) in DNA-A and two ORFs in DNA-B. Two ORFs (AV1 codes for coat protein, and AV2 for pre coat protein) are in virion-sense orientation or positive sense and four-five in complimentary or negative sense orientation (Fig. 10). AC1 codes for replication initiator protein (*rep*, also known as AC1 or AL1), AC2 for transcription activator protein (*TrAP*, also known as AC2) and AC3 for replication enhancer protein (*REn*, also designated as AC3). Two other genes, AC4 and AC5 have been described but their role in replication of the virus or pathogenesis is not known. The ORF AV2 is said to be a signature gene of old world begomoviruses. In DNA-B, there are two ORFs, the BV1 in virion sense and the BC1 in complimentary sense orientation. ORF BV1 codes for the nuclear shuttle protein which is required for the movement of viral DNA- both ss- and dsDNA- between nucleus and cytoplasm of host cells, whereas ORF BC1 codes for the movement protein which regulates cell to cell movement of the virus through plasmodesmata. It has been demonstrated that BV1 is also involved in long

distance movement of the virus by allowing the spread of the virus through vascular system of the host (Lazarowitz, 1992, Hanley-Bowdoin *et al*., 1999). Fig. 10 below shows genome organization of a bipartite genome of four LYMVs infecting pulse crops (MYMIV, MYMV, HgYMV and DoYMV).

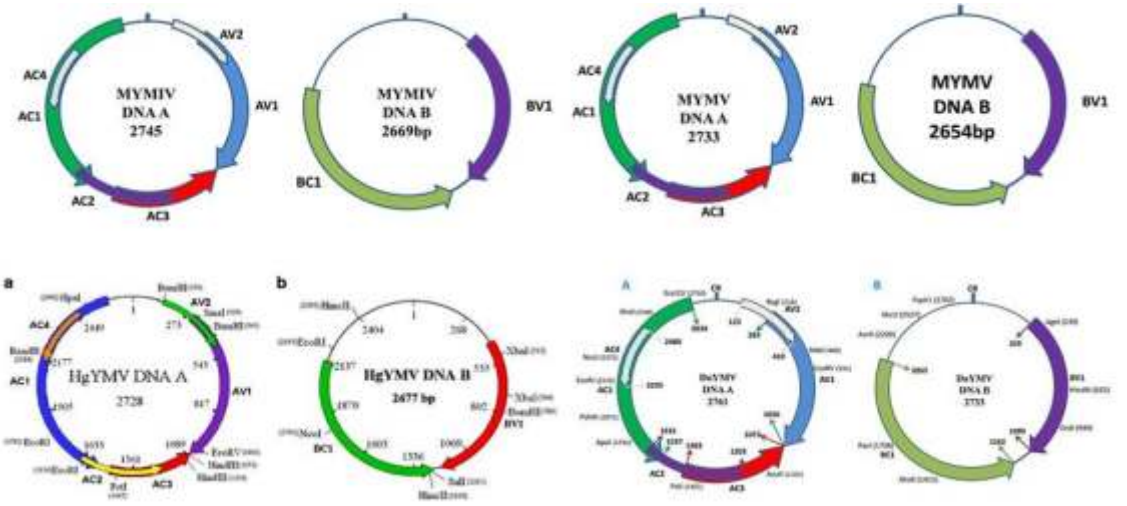


Figure 10. Genome organization of LYMVs (MYMIV-DNA A & DNA B, MYMV-DNA A & DNA B, HgYMV-DNA A & DNA B and DoYMV-DNA A & DNA B).

Molecular characterization

The genome of Thailand isolate of MYMV was cloned, sequenced and shown to be bipartite (Morinaga *et al*., 1990, 1993). In India, Varma *et al*. (1991) described the full genome (DNA-A and DNA-B) sequence of a blackgram isolate (Bg3) of MYMIV and found it to differ from Thailand isolate in the restriction pattern. A major breakthrough was made when Mandal *et al*. (1997) established the Koch's postulates using the cloned components of Bg3 isolate by agro-inoculation and for the first time successfully demonstrated the whitefly transmission of progeny virus from agro-inoculated plants. Last two decades have seen characterization of LYMVs isolates infecting different leguminous crops in different parts of India, *viz*. urdbean, mothbean, urbean, pigeonpea, soybean, dolichos, horsegram (Varma *et al*. 1991, Mandal *et al*. 1998, Roy, 2001, Malathi *et al*., 2005, Maruthi *et al.,* 2006, Karthikeyan *et al*., 2004, Girish and Usha 2005, Barnabas *et al*., 2010, Singh *et al.,* 2006).

Recently characterized LYMVs

Till 2007, only four virus species *viz.*, MYMIV, MYMV, HgYMV and DoYMV of LYMVs were known. However, recent past has witnessed characterization of many new begomoviruses

from different leguminous plants *viz*., *Rhynchosia yellow mosaic virus* (RhYMV) and *Rhynchosia yellow mosaic India virus* (RhYMIV) infecting *Rhynchosia minima*, a weed belonging to the family *Fabaceae*, respectively, in Pakistan (Ilyas *et al.*, 2009) and India (Jyothsna *et al.*, 2011), *Velvet bean severe mosaic virus* (VbSMV) infecting velvet bean (*Mucuna pruriens*), a medicinal plant belonging to the family *Fabaceae* (Zaim *et al.*, 2011), *Kudzu mosaic virus* (KuMV) infecting kudzu (*Pueraria montana*) (Ha *et al.*, 2008) and *Soybean mild mottle virus* and *Soybean chlorotic blotch virus* infecting soybean in Nigeria (Alabi *et al*., 2010). In phylogenetic studies, all these viruses clustered with LYMVs (Qazi *et al.*, 2007) or 'Legumoviruses' (Ilyas *et al.*, 2009, Briddon *et al.*, 2010, Alabi *et al*., 2010).

Velvet bean severe mosaic virus

Velvet bean [*Mucuna pruriens* (L.) DC] is one of the most important medicinal plants. It is used to treat many ailments, especially for Parkinson's disease because of the presence of 3,4-dihydroxyphenylalanine (l-dopa) in it. A yellow mosaic disease of velvet bean has been shown to be caused by a whitefly transmitted begomovirus named *Velvet bean severe mosaic virus* (VbSMV). DNA A of VbSMV has a maximum similarity of 76% (much below the species demarcation level of 91% (Brown *et al*., 2015) with DNA A of an isolate of MYMIV (AY937195), indicating it to be a new species of begomoviuses. Its DNA B has a maximum similarity of 49% with an isolate of HgYMV (AM932426). In phylogenetic studies based on DNA A sequence, it clustered with isolates of LYMVs (Zaim *et al*., 2011).

Rhynchosia yellow mosaic India virus

A begomovirus associated with yellow mosaic disease in *Rhynchosia minima*, a common leguminous weed was cloned, sequenced and named *Rhynchosia yellow mosaic India virus*-RhYMIV (Jyothsna *et al*., 2011). The virus has a bipartite genome with a typical Old World bipartite begomovirus genome organization. Blast results of its full length DNA A showed it to have a mximum similarity of 84% with an isolate of *Velvet bean severe mosaic virus*-(India: Lucknow:2009) VbSMV-(IN:Luc:09) (GeneBank Accession No. FN543425) indicating it to be new species of begomovbiruses. It has less than 73% identity with other legumoviruses. In phylogenetic studies based on DNA A sequence, RhYMIV clustered with isolates of legumoviruses.

Rhychosia yellow mosaic virus

In Pakistan, a yellow mosaic disease of *Rhynchosia minima*, a common leguminous weed was shown to be caused by a new begomovirus species named *Rhynchosia yellow mosaic virus* (RhYMV) (Ilyas *et al.*, 2009). DNA-A of RhYMV has a maximum sequence identity (69.5%) with

an isolate of *Mungbean yellow mosaic virus*. Since this was the first legumovirus species characterized from weed the author tried to see whether this virus could cause the disease in cultivated legume crops and through Agrobacterium-mediated inoculation demonstrated that RhYMV can cause the disease in soybean.

Kudzu mosaic virus

Kudzu (*Pueraria montana*) is an edible leguminous plant also called Japanese arrowroot. It belongs to the the pea family Fabaceae and subfamily Faboideae. A mosaic disease of kudzu was found to be caused by a bipartite begomovirus in Vietnam. Sequence analysis revealed that its DNA A shared maximum nucleotide identity of 65% with HgYMV. It was therefore considered a new species of begomoviruses and as designated as *Kudzu mosaic virus* (KuMV). In phylogetic tree, KuMV grouped tightly with three Old World legume-infecting bipartite begomoviruses: HgYMV, MYMV and MYMIV (Ha *et al*., 2008) and therefore is considered as a new member of LYMVs or legumoviruses. This virus has also been reported from China (Zhang and Wu, 2013).

*Dolichos yellow mosaic virus (*bipartite isolate*)*

Till 2014, DoYMV was the only LYMVs or legumoviruses for which DNA-B was not known. In 2015, an isolate of DoYMV infecting Dolichos bean at Kanpur has been characterized and shown to have bipartite genome (KJ481204 and KJ481205) (Akram *et al*., 2015).

The DNA-A of DoYMV consists of 2761 nucleotides and DNA-B of 2733 nucleotides with a genome organization typical of Old World bipartite begomoviruses. Nucleotide identity of DNA-B (KJ481205) of DoYMV with DNA-B of other legumoviruses was 57.5–61.0%. Both components contain a nonanucleotide and conserved inverted repeat sequences with the potential to form a stem-loop (Fig.11). Nucleotide identity of common region of DoYMV was 90.3%, above the threshold nucleotide identity (>85%) for considering a DNA-B molecule as cognate of DNA-A of a begomovirus.

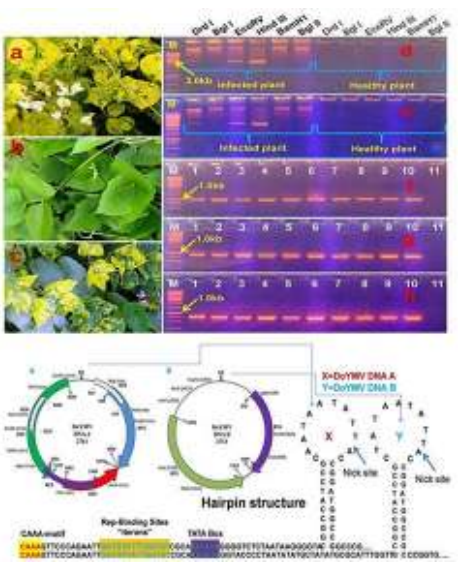


Fig. 11. Characterization of a bipartite isolate of DoYMV.

The multiple sequence alignment of the Ori region of the DNA-A and DNA-B (cognate) of DoYMV isolate (KJ481204 and KJ481205) and of other bipartite legumoviruses indicated presence of conserved rep binding motif (iterons), TATA box and stem-loop structure (Fig. 11, 12), the characteristic features of begomoviruses.

Table 1. Per cent nucleotide sequence identity between the components (DNA A, DNA B), common region (CR) and conserv region (CR) of cognate of DoYMV of this study and other selected legumoviruses and their replication-associated features

Virus	Accession No. DNA A/DNA B	PNI of CR of cognate	Predicted Iteron-related domain of Rep	Sequences surrounding the predicted Iteron showing sequence and spacing. The iterons a underlined.
*DoYMV	KJ481204/ KJ481205	90.3	MRAPGFRISA	GTC ATCGGTGTATCGGTGT.....(18)ATTGGTGTCTTTGGTGTCGCATATATA..(30)CAATAATATTAC GTC ATCGGTGTATCGGTGT..... (18)ATTGGTGTCATTGGTGTCGCATATATA..(46)CTATAATATTAC
#DoYMV	AY271891/ -	-	MRAPGFRISA	GTC ATCGGTGTATCGGTGT.....(18)ATTGGTGTCTTTGGTGTCGCATATATA..(30)CAATAATATTAC -
MYMV	JX244176/ JX244181	87.3	MPRLGRFAIN	TGTATCGGTGT.....(20) ATCGGTGTATCGGTGTCTTATTTATATG..(34)CAATAATATTAC GCAATCGGTGT .....(20) ATCGGTGTATCGGTGTCTTATTTATAGGT..(51)CTATAATATTAC
MYMIV	NC-004608/ NC-004609	88.3	MPREGRFAIN	TGT ATCGGTGT.....(25)ATCGGTGTATCGGTGTACTATATA.....(35)CTATAATATTAC CGAATTGGTGT..... (25) ATCGGTGTATTGGTGTATTATATA.....(25)ATATAATATTAC
HgYMV	NC_005635/ NC_005636	89.4	MPRERRFAIN	TGT ATCGGTTG..... (27) ATCGGTGAATGGTGACGCATATATA .....(32)CAATAATATTAC GCT ATTGGTGA..... (29)ATCGGTGAATGGTGACGCATATATA..... (33)TACTAATATTAC
RhYMIV	NC_014902/ NC_014903	93.2	MANSSRFKIN	TGT ATCGGTGT..... (26) ATTGGTGTATTGGAGTCCCATATATA..... (32)GAATAATATTAC CGAATCGGTGT..... (26) ATTGGTGTATTGGAGTCCCATATATA..... (31)GTATAATATTAC
KuMV	HQ162271/ HQ162272	78.3	MSRPKGFRVN	CCA ATTGGGGT.....(18) AGTATTAGTGTCGCATATATA .....(34)GAATAATATTAC CCGATTGGGGT..... (18) AGTATTAGTGTCGCATATATA..... (56)TTATAATATTAC
VbSMV	NC_013414/ NC_013415	98.1	MANASRFKIN	GCT ATCGGGGT.....(26) ATTGGTGTATTAGAGTCCCATATATA.....(32)GTATAATATTAC CGA ATCGGTGT..... (26) ATTGGTGTATTAGAGTCTCATATATA.....(31)GTATAATATTAC
RhYMV	AM999981/ AM999982	91.8	MPTPGRFCIN	GCTATCGGGTG .....(28) ATCGGTGTATTGGAGTCCTATATATA..... (26) GTATAATATTAC ACAATCGGAGT..... (26)ATCGGTGTATTGGAGTCCTATATA..... (27) GTATAATATTAC

* This study, PNI=Per cent nucleotide identity, CoR=Common region, #the cognate of this isolate was not characterized.
Yellow highlighted is TATA Box and green Nonanucleotides.

Fig. 12. Nucleotide characteristics of DNA-A and DNA-B of legumoviruses

Phylogenetic analysis of DNA-A sequences of begomoviruses indicated that DoYMV-[IN:Knp:14] grouped with legumoviruses separately from the begomoviruses infecting non-leguminous hosts. Four recombination events in DNA-A and two in DNA-B of DoYMV isolate were detected. *Mungbean yellow mosaic virus, Rhynchosia yellow mosaic virus* and *Horsegram yellow mosaic virus* were identified as probable parents.

Soybean mild mottle virus and *Soybean chlorotic blotch virus*

Two distinct virus species infecting soybean in Nigeria were recently characterized and and designated as Soybean mild mottle virus (SbMMV) and Soybean chlorotic blotch virus (SbCBV). SbCBV was found to have a bipartite genome, whereas SbMMV has only a DNA-A component. Sequence comparison with other begomoviruses available at databases revealed that these two species are new and grouped with other bipartite legumoviruses. Phylogenetic analysis of these viruses with those of other begomoviruses showed clustering of the two viruses within the 'legumovirus' clade. SbCBV isolate from *Centrosema pubescens* Benth. was found to be identical to those from soybean, indicating that leguminous wild species are potential alternative hosts for the virus. Since soybean is an introduced crop, the identification of two distinct begomoviruses naturally infecting soybean in Nigeria suggests the occurrence of 'legumoviruses' in plant species indigenous to Africa and underscores their potential threat to sustainable cultivation of soybean on the African continent (Alabi *et al*., 2010).

Virus species and pathogenic variability

The reports that the mungbean and urdbean genotypes vary in their reaction to YMD at different locations (Amin and Singh, 1989, Singh and Naimuddin, 2009) made one to believe that there exist strains of the virus (MYMV). The concerted research efforts invested in YMD causing viruses during last 25 years or so have resulted in the better understanding on the identity of virus species involved in the YMD of pulses. At present it is known that the virus involved in causing YMD in mungbean and urdbean in South and North India are two distinct species of the genus *Begomovirus*, but the issue of temporal and spatial variation in the disease reaction of a genotype across the country still remains. In fact the virus-vector-host interaction is a complex mechanism especially in case of geminiviruses as even a single-nucleotide difference between two isolates of a geminivirus (*Squash leaf curl virus)* has been shown to alter the host range (Ingham *et al*., 1995). Also, the role of insect vector in determining the natural host range of these viruses cannot be ruled out. Further other factors influencing epidemiology of a virus disease may also be responsible for difference in disease reaction of a genotype at different locations.

We collected YMD affected samples of different leguminous plants from around the Kanpur in North India and Coimbatore, Vamban in South India. Results of PCR tests using specific primers, indicated involvement of MYMIV in YMD affected samples of mungbean, urdbean, rajmash, cowpea, pigeonpea, soybean from North India and MYMV in samples of mungbean, urdbean, rajmash and HgYMV in horsegram from South India. However, some recent publications reporting occurrence of MYMIV in southern states of Tamil Nadu (Satya *et al*., 2015) and Andhra Pradesh (Reddy *et al*., 2015) and MYMV in North (Gautam *et al.,* 2014) have put a question mark on the existing understanding that MYMIV is present in north, east and central parts (Usharani *et al*., 2004) and MYMV in southern states of India (Karthikeyan *et al*., 2004). Also, presence of DNA beta satellite in MYMIV infected cowpea (Rouhibakhsh and Malathi, 2005, Ilyas *et al*., 2010, JX443646 and DQ118862) and in MYMV infected urdbean, mungbean (Satya and Alice, 2014) has been reported to exacerbate the disease symptoms. This is going to be a challenge to researchers involved in breeding for YMD resistant pulse varieties, particularly mungbean and urdbean. PCR based protocols using virus species specific primers can be employed routinely to specify the identity of the virus present in a location where the field screening of breeding materials and varietal development are taken up.

Host specificity and host adaptation

Briddon *et al*. (2010) considered DNA-A and DNA-B of begomoviruses to have very distinct evolutionary histories and that the component exchange played a greater role in diversification of begomoviruses. They also hypothesized that initially all the begomoviruses were monopartite and

DNA-B originated as a satellite, which was captured by monopartite progenitor, which later became the integral part of the genome.

Till 2007, only four viruses - MYMV, MYMIV, HYMV and DoYMV were known to cause YMD in many legumes across southern Asia (Qazi *et al*. 2007). Bipartite nature of DoYMV has been proved only very recently (Akram et al., 2015). During last decades few more begomoviruses characterized from leguminous hosts have been shown to have close phylogenetic relationship with viruses causing yellow mosaic disease in legumes. At present 11species can be said to be the members of LYMVs. All the LYMVs have their host ranges limited to plants of the family *Fabaceae.* The legume-infecting begomoviruses from the old world, the most unusual of the begomoviruses are distinct from the numerous legume-infecting begomoviruses that occur in the Americas. In phylogenetic analyses they segregate to form a separate cluster among the old world begomoviruses (Padidam *et al*., 1995; Fauquet *et al*., 2008). The number of begomoviruses with bipartite genome infecting legumes has gone up and a new term 'Legumoviruses' has been coined and is being used for them (Ilyas *et al*., 2009, Briddon *et al*., 2010).

A phylogenetic tree was constructed using representative sequences of all the legumoviruses, some other begomoviruses and an accession of MSV to out group the tree. The tree shows that all the legumoviruses cluster together, whereas other viruses formed a separate cluster (Fig. 13).

There are many reports of resistance sources in leguminous crops but the durability of the resistance has always been questioned probably due to genetic interaction between these begomoviruses within the legumes, in the form of both classical recombination and component exchange. There is however little evidence for interaction with viruses that infect other plants. This is indicative of genetic isolation, the viruses in legumes evolving independently of the begomoviruses in plant species of other families (Qazi *et al*., 2007).

Of the all LYMVs, the two which have been studied in somewhat detail- MYMV and MYMIV are unusual in having highly variable DNA-B components. An isolate of MYMV infecting blackgram (MYMV-[IN:Vig]) in Tamil Nadu was shown to have be associated with five different DNA-B components (KA21, KA22, KA27, KA28 and KA34). One of the five DNA-B components (KA27) showed 97% sequence identity to the DNA-B of MYMV isolate from Thailand, whereas rest four (KA21, KA22, KA28 and KA34) exhibited only 71–72% sequence identity to MYMV DNA-B. Co-existence of multiple DNA B components in field-infected *V. mungo* was proved by Southern and PCR analyses. Each of the five DNA-B components was infective together with the DNA A upon agroinoculation. Agro-inoculation with mixed cultures of *Agrobacterium* with partial dimers of DNA-A and all five DNA-Bs proved that all five DNA-B components can co-infect a single *V. mungo* plant (Karthikeyan *et al*., 2004). A variant of MYMIV causing golden mosaic of cowpea in northern India (MYMIV-[Cp]) was transmissible by whitefly (*Bemisia tabaci*) only to cowpea (*Vigna unguiculata*), yard long bean (*V. unguiculata* subsp.

sesquipedalis), and frenchbean (*Phaseolus vulgaris*) and not to blackgram (*V. mungo*), mungbean (*V. radiata*) and mothbean (*V. aconitifolia*). However, agro-inoculation of the cloned DNA-A and DNA-B components of MYMIV-[Cp] produced systemic symptoms in blackgram (*V. mungo*), mungbean (*V. radiata*), and mothbean (*V. aconitifolia*). Though the virus was transmissible to blackgram and produced severe symptoms, very low levels of viral DNA components were seen, indicating only limited adaptation to the host. It also indicated changes in pathogenicity following agroinoculation and passage to vector (Malathi *et al*., 2005). Nucleotide sequence comparison of MYMIV-CP DNA A (AF481865) indicated 1-6 % differences between MYMIV-CP DNA-A (AF481865) and other isolates of MYMIV. How these differences influence pathogenicity need to be studied, because even a single-nucleotide difference between two isolates of *Squash leaf curl virus* was shown to alter the host range (Ingham *et al*., 1995). Also, the role of insect vector in determining the natural host range of these viruses cannot be ruled out.

Furthermore, an isolate of MYMV from soybean (MYMV-[IN:Mad:Sb]) was shown to be associated with a DNA-B with high sequence identity (96%) to the DNA-B of HgYMV. DNA-B of HgYMV is the most distinct amongst the LYMV DNA-Bs, showing only 70–73% identity to the DNA-B components of MYMV and MYMIV. These findings indicate that component exchange (so called pseudo-recombination) is common-place for the LYMVs, both within and between species and is probably an adaptation allowing a change in host range (Qazi *et al*., 2007).

DNA-A and DNA-B components of bipartite begomoviruses share a region (~200 nucleotides) with high sequence identity. This region contains the origin (ori) of virion strand DNA replication. The ori consists of a conserved hairpin structure containing the nonanucleotide motif (TAATATTAC) (which is ubiquitous in geminiviruses) and repeated motifs ('iterons'). These iterons are sequence-specific recognition sequences for *Rep* protein (encoded by the ORF AC1 on the DNA-A) that nicks the nonanucleotide motif to initiate rolling circle DNA replication (Argüello-Astorga *et al*., 1994). The DNA-A and DNA-B components of bipartite begomoviruses including LYMVs possess the same iteron sequences, thereby ensuring that the DNA-A encoded *Rep* may initiate replication of both components; maintaining the integrity of the split genome (Shafiq *et al*., 2010). High level of specificity of the interaction between Rep and the iteron is responsible for preventing interaction between components of distinct begomovirus species (Chatterji *et al*., 2000; Fontes *et al*., 1992, 1994; Orozco *et al*., 1998). Thus, the integrity of the bipartite genomes of begomoviruses is maintained by each component having the same (or at least a closely related) ori containing the same iterons. MYMV, MYMIV and DoYMV have a similar iteron sequences (predicted to be GGTGT) whereas HgYMV has the predicted iteron motif GGTAT and would not be able to readily exchange components with the other LYMVs.

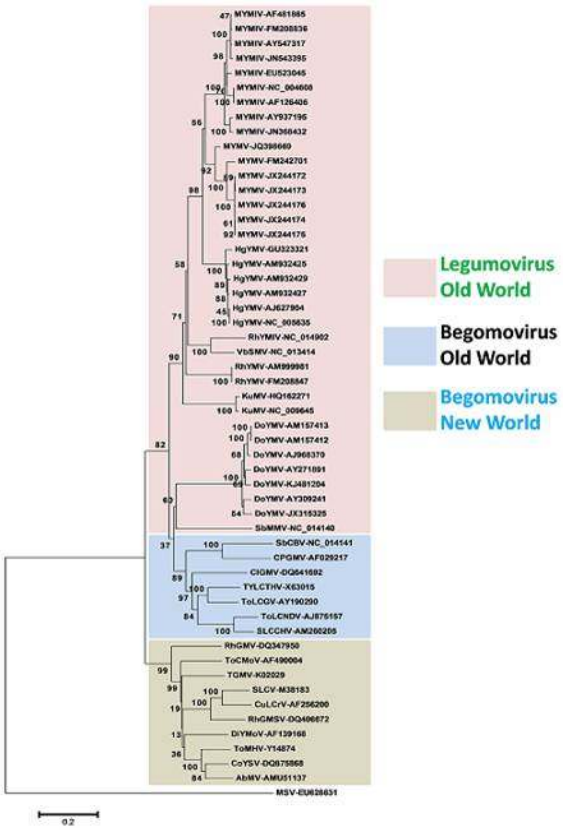


Fig. 13. Phylogenetic trees constructed from DNA-A components of legumoviruses and other selected begomoviruses. The analysis involved 54 nucleotide sequences of DNA-A. DoYMV, *Dolichos yellow mosaic virus*; AbMV, *Abutilon mosaic virus*; MYMV, *Mungbean yellow mosaic virus*; CoYSV, *Corchorus yellow spot virus*; MYMIV, *Mungbean yellow mosaic India virus*; SbMMV, *Soybean mild mottle virus*; CoGMV, *Corchorus golden mosaic virus*; HgYMV, *Horsegram yellow mosaic virus*; MSV, *Maize streak virus*; RhYMIV, *Rhynchosia yellow mosaic India virus*; CuLCrV, *Cucurbit leaf crumple virus*; KuMV, *Kudzu mosaic virus*; SLCV, *Squash leaf curl virus*; VbSMV, *Velvet bean severe mosaic virus*; RhGMSV, *Rhynchosia golden mosaic Sinalova virus*; RhYMV, *Rhynchosia yellow mosaic virus*; DiYMoV, *Dicliptera yellow mottle virus*; CIGMV, *Clerodendrum golden mosaic virus*; TGMV, *Tomato golden mosaic virus*; SLCCNV, *Squash leaf curl China virus*; ToCMoV, *Tomato chlorotic mottle virus*; ToLCNDV, *Tomato leaf curl New Delhi virus*; ToMHV, *Tomato mosaic Havana virus*; ToLCGV, *Tomato leaf curl Gujarat virus*; RhGMV, *Rhychosia golden mosaic virus*; TYLCTHV, *Tomato yellow leaf curl Thailand virus*; CPGMV, *Cowpea golden mosaic virus;* SbMMV, *Soybean mild mottle virus*; SbCBV, *Soybean chlorotic blotch virus*.

However, component capture between distinct species does occur if, by recombination, the ori of DNA B is replaced by that of the DNA-A (so-called 'origin donation'). This appears to be the case for the HgYMV-like DNA-B of MYMV-[IN:Mad:Sb], which contains the MYMV/MYMIV iteron motifs GGTGT (Girish and Usha, 2005). However, this isolate was also associated with a normal, MYMV DNA-B.

Diagnosis

Detection of LYMVs

A number of viruses are known to infect pulses or leguminous plants inducing a variety of symptoms that range from mosaic to necrosis to sterility. Yellow mosaics, sterility, stunt, crinkle, leaf curl, tip necrosis, mottling are the well known virus diseases in pulse crops. The causal viruses of these diseases are named based on symptoms. However, often similar types of symptoms are induced by different viruses and similar viruses may cause different symptoms in the host. For example, yellow mosaic disease in mungbean, urdbean, rajmash, dolichos, horsegram, pigeonpea, *etc*., is caused by at least four different viruses but with similar symptoms *viz*., yellow mosaic (Fig. 14). Identification of a virus based on the symptoms only may lead to an erroneous conclusion and hence is a challenge. This makes it imperative to have an accurate diagnosis of the viruses which is often the first step in disease management.

Fig. 14. YMD symptoms in four different pulse crops. In each crop the disease is caused by different virus species

Diagnostics, in definition are part of a separate discipline, which combines the development of wide range of traditional and newer techniques for sensitive and specific measurement of causative agent of disease in biological material. The traditional method used for diagnosis of virus disease is time consuming and are often considered obsolete in the era of state of the art diagnostics protocols. Current diagnosis techniques are broadly divided into serological and nucleic acid based procedures and combination of both. In serological procedure Enzyme linked immunoassay (ELISA), Tissue blot immunoassay (TIBA) and Quartz crystal microbalance (QCM) immune sensors are used. In nucleic acid based procedures, Reverse transcription – polymerase chain reaction (RT–PCR) and PCR, Multiplex RT-PCR, Fluorescence RT-PCR using Taqman TM technology, Competitive fluorescence PCR (CF-PCR), Immuno capture PCR (IC-PCR) and Nested PCR are used.

Use of polyclonal and monoclonal antibodies to *African cassava mosaic* (ACMV) and Indian cassava mosaic viruses (ICMV) has been employed to ascertain the identity of the YMD causing agents as begomoviruses (Harrison *et al*., 1991). Positive reaction with polyclonal and monoclonal antibodies to ACMV and ICMV established the etiological agents of YMD to be begomoviruses. Based on epitope profile, LYMVs isolates could be categorized broadly into two groups, one, comprising virus isolates causing yellow mosaic in dolichos and the second group

consisting of LYMVs isolates infecting other legumes (Malathi, 2007). Species specific identification of the virus isolates based on serological reaction has not been possible due to conserved coat protein gene. Once this was realized, nucleic acid based identification of begomoviruses developed fast.

In fact the use of nucleic acid-based diagnostic methods for the detection of plant pathogens started in late 70s of the last century when the first double-stranded RNA and dot-blot hybridization protocols were developed for the detection of viruses and viroids (Hull, 1986). Development of polymerase chain reaction (PCR) protocol (Saiki *et al*., 1985, 1988) in 90s of the last century was soon followed by its exploitation for the detection of plant pathogens (Puchta and Sanger, 1989). At present PCR is a widely accepted method for the detection of plant viruses.

Degenerate primers (Deng *et al*., 1994, Rojas *et al*., 1993) are commonly used to ascertain geminiviral nature of a virus. Use of degenerate primers however does not provide information about the species specific identity of the virus thereby limiting the utility of degenerate primers. PCR products from degenerate primers are required to be sequenced to identify the likely species of the *Geminiviridae*. Moreover, most of the geminivirus specific degenerate primers have been designed using genome sequences of New World Geminiviruses and their utility in detection of LYMVs has not been examined critically. There is however, limited information on specific primers for the detection and identification of pulse infecting begomoviruses wherein characterization of whole genome is targeted (Rouhibakhsh *et al*., 2008, Malathi and Johns, 2008).

Differences in the reaction of urdbean and mungbean genotypes against YMD across the country necessitated the need to develop an easy and efficient PCR based protocols for differentiating begomovirus species involved in YMD. The option of sequencing the whole genome is a time consuming and costly for routine tests. Therefore, at IIPR, Kanpur attempts were made to develop PCR based diagnostics for species specific identification of viruses known to cause YMD in different pulse crops. Species specific primers were designed (Table 4) and PCR protocols developed for detection of viruses in YMD affected samples of different pulse crops. These primers are designed in such a way that complete gene can be extracted from the direct sequencing of amplified product without cloning. Using these protocols infection of MYMIV, MYMV, HgYMV and DoYMV has been confirmed in mungbean, urdbean, soybean, pigeonpea, dolichos, rajmash, horsegram, many wild accessions of *Vigna* and weeds (Naimuddin *et al*., 2011a, b, c). We collected YMD affected samples of different leguminous plants from around Kanpur in North India and Coimbatore, Vamban in South India. Results of PCR tests using specific primers, indicated involvement of MYMIV in YMD affected samples of mungbean, urdbean, rajmash, cowpea, pigeonpea, soybean from North India and MYMV in samples of mungbean, urdbean, rajmash and HgYMV in horsegram from South India. Using primers (Table 4) MYMIV and DoYMV prevalent in and around Kanpur have been detected in different hosts. The protocols for the detection of these viruses are being described in following paragraphs.

Table 4. Details of the primers designed to get the complete coat protein gene and movement protein gene of four bogemoviruses known to infect different leguminous host in India and expected size of amplicons.

Primer ID	Primer sequence 5'……3'	AT (^{0}C)	~ size of DNA fragment to be amplified	Name of the virus to be detected/ DNA component
NM 1/AV1PF NM 2/AV1PR	GTA TTT GCA KCA WGT TCA AGA AGG DGT CAT TAG CTT AGC	54	1000bp	MYMIV/DNA A
HYMV-CP-F HYMV-CP-R	ATG CTT GCA ATT AAG TAC TTG CA TAG GCG TCA TTA GCA TAG GCA	56	1050 bp	HgYMV/DNA A
DYMV-CP-F DYMV-CP-R	CTG TGA AAT TTG TGC AGG TAC GCG GTT GCG AAT ATG TAT	54	900bp	DoYMV/DNA A
MYMV-CP-F MYMV-CP-R	ATG GG (T/G) TCC GTT GTA TGC TTG GGC GTC ATT AGC ATA GGC AAT	54	1000bp	MYMV/DNA A
HYMV-MPF HYMV-MPR	ATG GAG CAT TAT TCC GGT GCA TTA CA(G/A) GGT TTT GTT TAC AGT	64	900bp	HgYMV/ DNA B
MYMV-MPF MYMV-MPR	ATG GAG AAT TAT TCA GGC GCA TTA CAA CGC TTT GTT CAC ATT	58	900bp	MYMV/ DNA B
MYMIV-MPF MYMIV-MPR	ATG GAA AAT TAT TCA GGT GCA CTA CAA CGC TTT GTT CAC ATT	53	900bp	MYMIV/ DNA B
AC1PF AC1PR	AGT TGA TAT GGA TGT AATAGC3 ACA AAA ACG ACT TCA AATATG CCA A	49	1100bp	MYMIV/DNA A
AC2PF AC2PR	AGC TAA TGA CCC CTA AAT TAT GAG TA C TTG GAT GAA GAG AAC	49	480bp	MYMIV/DNA A
AC3PF AC3PR	TTA TGA TTC GAT ATT GAA TTA ATA CTG AAG TGTGGG TGT AGC TAT	48	450bp	MYMIV/DNA A
AC4PF AC4PR	CAA ATT ACAATT TAA GTT ATG ACT TCT AGCCTT GTC AAC ACC AG	48	390bp	MYMIV/DNA A

NM1/NM2=Used for CP gene amplification of *Mungbean yellow mosaic India virus*; MYMIV-MPF/MYMIV-MPR=used for movement protein gene amplification of *Mungbean yellow mosaic India virus* (DNA B component); HYMV-CPF/HYMV-CPR= Used for CP gene amplification of *Horse gram yellow mosaic virus*; HYMV-MPF/HYMV-MPR= Used for movement protein gene amplification of *Horse gram yellow mosaic virus*; MYMV-CP-F/MYMV-CP-R=Used for CP gene amplification of *Mungbean yellow mosaic virus*; MYMV-MPF/MYMV-MPR=Used for CP gene amplification of *Mungbean yellow mosaic virus*; DYMV-CP-F/DYMV-CP-R= Used for CP gene amplification of *Dolichos yellow mosaic virus;* AC1PF/AC1PR, AC2PF/AC2PR, AC3PF/AC3PR, AC4PF/AC4PR= Used for AC1, AC2, AC3 and AC4 gene amplification of MYMIV (Naimuddin *et al.*, 2011a, b, c).

MYMIV infection in Ageratum

A. conyzoides, a common weed seen all round the year throughout the country is often found affected with yellow vein disease (Fig. 15). In our attempt to identify alternate host of MYMIV we tested it for presence of MYMIV.

Fig. 15. Yellow vein symptoms in *A. conyzoides*

Results of the PCR tests using total DNA isolated from diseased

samples (n=40) of *A. conyzoides* as template with primers pairs, NM1/NM2 and MYMIV-AC1F/MYMIV-AC1R (Table 4) specifically designed to detect a part of DNA-A of MYMIV gave amplicons of expected size ~1000bp and ~1100bp, respectively in 52.5% samples (Fig. 16). This indicates that the samples were infected with MYMIV. Since all the LYMVs have bipartite genome, we also used primers specific to a segment of DNA B of these three viruses (Table 1). All the samples positive with MYMIV DNA-A also gave positive reaction to MYMIV DNA-B in PCR with primers pair, MYMIV-MPF/MYMIV-MPR (amplicon size ~900bp). However, none of the 40 samples gave any amplification with primers pairs MYMV-CPF/MYMV-CPR and HgYMV-CPF/HgYMV-CPF specific to DNA-A and HgYMV-MPF/HgYMV-MPR and MYMV-MPF/MYMV-MPR specific to DNA B of MYMV and HgYMV, respectively. All the disease free plant samples gave no amplification with any of the primer pairs used.

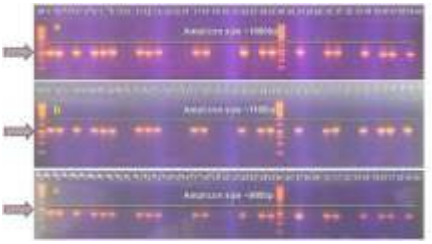


Fig. 16. Gel electrophoresis of PCR amplified products of MYMIV genome. a) Segments of DNA A with primer pairs NM1/NM2 and b) with primer pair MYMIV-AC1F/MYMIV-AC1R, segment of DNA B with primer pair MYMIV-MPF/MYMIV-MPR.

To further confirm the association of MYMIV with ageratum, the RCA technology was employed. RCA products of the four samples, positive to MYMIV in PCR tests, digested with restriction enzymes *Eco*RI and *Eco*RV that have only one cut site in DNA-A and *Bgl*I that has only one cut site in DNA-B of MYMIV produced a band of ~2.7kb indicating that both the components of MYMIV genome were present in the diseased samples of *A. conyzoides*. Presence of undigested high molecular weight DNA and bands <2.7kb in the gel, however, indicated possibility of another geminivirus also. Gel electrophoresis of RCA product of one of the samples negative to MYMIV in PCR tests digested with restriction enzymes *Eco*RV and *Bgl*I showed only high molecular weight DNA and no band of ~2.7kb; whereas restriction digestion with *Eco*RI released three DNA fragments with total size of ~2.7kb. This indicated that the MYMIV negative sample carried a geminivirus different than MYMIV (Fig. 17). This weed is found all round the year throughout the country and hence presence of MYMIV in this weed has importance in the carrying the diseases from one season to other. Intensive studies are required to study the diversity in the geminiviruses infecting weeds so as the epidemiology of yellow mosaic disease is clearly understood (Naimuddin *et al*., 2014).

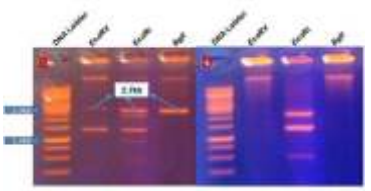


Fig. 17. Gel electrophoresis of restriction digested RCA products (a) of MYMIV positive in PCR (b) of MYMIV negative in PCR.

Mixed infection of MYMIV and DoYMV in Cowpea

Cowpea, grown for green pods used as vegetable and also for green fodder in North Indian plains was found to be affected by yellow mosaic disease during *kharif* 2009 in and around Kanpur. Yellow mosaic affected cowpea plants showed typical golden mosaic symptoms on leaf lamina.

Symptoms initially started as small yellow flecks/spots on leaf lamina and progressed to appear as golden yellow coloured spots in the lamina. As the infection grew older, some degree of malformation and reduction in the width of lamina were also noticed. The disease has been referred as golden mosaic disease of cowpea. In India, cowpea golden mosaic was once thought to be caused by a virus different than the viruses causing yellow mosaic disease in crops like mungbean and urdbean (Varma and Reddy, 1984). Malathi *et al.* (2005) found golden mosaic disease of cowpea in Delhi to be caused by an isolate of MYMIV. Recently, John *et al.* (2008) also reported involvement of MYMIV in causing golden mosaic disease of cowpea in western India. There is, however, no information on the virus causing golden mosaic in other parts of the country. In an endeavor to get the information on the variability in viruses causing YMD in pulse crops, we attempted to identify the virus involved in golden mosaic disease of cowpea at Kanpur. Primer pairs designed to get a fragment of DNA-A containing coat protein gene of four begomoviruses *viz*., MYMIV, MYMV, HgYMV and DoYMV known to infect various leguminous crops in India were used in PCR tests to detect virus(es) associated with golden mosaic disease of cowpea in and around Kanpur (Table 4).

Results obtained by these primer pairs (Table 4) indicated involvement of MYMIV in all the ten samples, whereas three samples yielded mixed infection with MYMIV and DoYMV. In PCR all the samples yielded amplified DNA fragment of expected sizes *viz.,* ~1000bp and ~900 bp with primer pairs specific to respectively, coat protein gene (located on DNA-A) and movement protein gene (located on DNA-B) of MYMIV. No amplification was obtained with primers specific to coat protein gene of HgYMV and MYMV. However, in three samples, amplification of DNA fragment of approximately 1000bp was observed with primer pair specific to coat protein gene of DoYMV. Sequencing of amplicons obtained with MYMIV and DoYMV specific primers yielded complete coat protein gene of MYMIV and DoYMV, respectively.

The sequence of amplified coat protein gene of MYMIV contained a single open reading frame with 774 nucleotides and 257 amino acids. The sequence data was submitted to GenBank (GU591171) and the isolate was designated as MYMIV-[CpKn]. In case of DoYMV also the coat protein gene contained a single open reading frame with 774 nucleotides and 257 amino acids and the isolate was designated as DoYMV-[CpKn]. The sequence data was submitted to GenBank (GU591170). Nucleotide comparison of coat protein gene sequences of MYMIV-[CpKn] isolate with that of other begomoviruses infecting leguminous hosts indicated that MYMIV [CpKn] under study had 95-99 % similarity with isolates of MYMIV described from different places. DoYMV-[CpKn] had 95-96 % similarity with other isolates of DoYMV described from India and Bangladesh. This is the first report of mixed infection of cowpea with two LYMVs (MYMIV and DoYMV) (Naimuddin and Akram, 2010).

MYMIV and MYMV in wild accessions of *Vigna*

To confirm as to which of the LYMVs infect wild accessions of *Vigna* grown at Kanpur, PCR based tests were employed using the primers mentioned in Table 4 . During 2009-2010, field infected *V. mungo* var. *silvestris* (accession no. IPUW-07) plants showed yellow mosaic symptoms typical of begomoviruses responsible for yellow mosaic diseases in leguminous plants. Initially

only few yellow patches appeared in the inter-veinal areas and by the time crop reaches maturity more than 80% of the leaf area became yellow. Disease incidence was 100 per cent. PCR tests yielded amplified DNA fragment of expected sizes *viz.,* ~950 bp. direct sequencing of purified PCR products was used to obtain the *AV1* gene sequence. The sequences of amplified coat protein gene contained a single open reading frame with 774 nucleotides and 257 amino acids. The sequence data was submitted to GenBank under accession number FJ821189. Comparison of coat protein gene sequences of begomovirus infecting *Vigna mung* var. *silvestris* with that of other begomoviruses infecting leguminous host including various isolates of *Mungbean yellow mosaic India virus* (MYMIV) indicated that begomovirus under study had maximum similarity (95-97 %) with isolates of MYMIV described from different legume host *viz*. MYMIV [Mungbean] (97 %), MYMIV [Soyabean] (97 %), MYMIV [Pakistan] (96 %), MYMIV [Nepal] (96 %), MYMIV [Akola] (96 %), MYMIV [Bangladesh] (95 %) and MYMIV (95 %) at nucleotide level. Nucleotide similarity with other begomovirus causing yellow mosaic in leguminous hosts was 84 % with MYMV [Soybean] and HYMV, and 77 % with DoYMV. PCR tests with primers specific to AC1, AC2, AC3 and AC4 genes of MYMIV also gave positive reaction and the sequences of these genes (FJ663015) had maximum similarity with the corresponding genes of other isolates of MYMIV. This study indicated that these primers can be used to identify MYMIV infection in YMD affected plants (Naimuddin *et al*., 2011a).

During the rainy season of 2010, accessions of wild species of *Vigna hainiana* (IC-331615, National Bureau of Plant Genetic Resources, New Delhi, India) and *V. trilobata* (IC-331436), grown at Indian Institute of Pulses Research, Kanpur, India, showed symptoms like yellowing of inter-veinal tissue and bright yellow spots in the leaves (Fig. 18). All plants of *V. hainiana* (8 plants) and *V. trilobata* (15 plants) were affected. The disease severity measured in terms of percentage foliage yellowing was 80% in the former and 30% in the latter. The association of a begomovirus was confirmed by PCR using primer pairs specific to MYMIV and MYMV (Table 4). In PCR, all the four samples drawn from each of the two accessions showing yellow mosaic symptoms gave positive results with MYMIV specific primer pairs NM1/NM2 and MYMIV-MPF/ MYMIV-MPR and yielded amplicons of ~1000bp and ~900bp respectively, indicating presence of both DNA-A and DNA-B components. Results with MYMV specific primers were negative (Naimuddin *et al*., 2011b).

Fig.18. Yellow mosaic symptoms on wild *Vigna. V. umbellata* and *V. glabrescens* are free from yellow mosaic symptoms.

In another experiment, leaf samples were collected from wild accessions of *Vigna* (3 of *V. hainiana,* 6 of *V. trilobata,* 4 of *V. mungo* var. *silvestris*, 1 of *V. radiata* var. *radiata*) grown at main

research farm of IIPR, Kanpur during 2010, which showed yellow mosaic symptoms and from one accession of each of *V. umbellata* and *V. glabrescens* that were free from yellow mosaic disease (Table 5).

Table 5. Identification of the causal virus of yellow mosaic disease of wild *Vigna* accessions grown at main research farm of IIPR, Kanpur during 2010

S. No.	Accession	Species/sub-species	Collection/ Source	Detection of MYMIV	
				DNA A	DNA B
1	IC-331448	*Vigna hainiana*	[1]NBPGR, T	+	+
2	IC-331450	*Vigna hainiana*	NBPGR, T	+	+
3	IC-251381	*Vigna hainiana*	NBPGR, T	+	+
4	IC-349701	*Vigna trilobata*	NBPGR, T	+	+
5	IC-331436	*Vigna trilobata*	NBPGR, T	+	+
6	IC-331454	*Vigna trilobata*	NBPGR, T	+	+
7	[#]JAP/10-7	*Vigna trilobata*	[2]WG	+	+
8	IC-251446	*Vigna umbellate*	NBPGR, T	-	-
9	[#]JAP/10-5	*Vigna trilobata*	WG	+	+
10	[#]JAP/10-9	*Vigna trilobata*	WG	+	+
11	IC-251372	*Vigna glabrescens*	NBPGR, T	-	-
12	IC-277031	*Vigna mung* var. *silvestris*	NBPGR, T	+	+
13	IC-277021	*Vigna mung* var. *silvestris*	NBPGR, T	+	+
14	IC-539798	*Vigna mung* var. *silvestris*	NBPGR, T	+	+
15	IC-277014	*Vigna mung* var. *silvestris*	NBPGR, T	+	+
16	IC-251426	*Vigna radiata* var. *radiata*	NBPGR, T	+	+

= accession number yet to be allotted, *= per cent foliage showing yellow mosaic [1]NBPGR,T= NBPGR Regional Station, Thrissur, [2]WG= Western Ghats, +=presence and -=absence of MYMIV.

The causal virus was identified by PCR using specific primers designed to amplify a segment of DNA A that contained CP gene of four begomoviruses. In PCR, all the samples drawn from accessions showing yellow mosaic symptoms gave positive results with MYMIV specific primer pairs NM1/NM2 and MYMIV-MPF/ MYMIV- MPR and yielded amplicons of ~ 950bp and ~ 900bp, respectively (Table 5). This clearly indicated that the yellow mosaic disease of wild species/subspecies of *Vigna* grown at Kanpur is caused by *Mungbean yellow mosaic India virus*. PCR results with primers specific to MYMV, HgYMV and DoYMV were negative indicating that these accessions at Kanpur are affected by YMD caused by MYMIV only. Accessions of *V. umbellata* and *V. glabrescens* that were free from yellow mosaic symptoms gave negative result not only with MYMIV specific primers but also with primers specific to MYMV, HgYMV and DoYMV. These primers were also used to detect LYMVs in wild accession of *Vigna* grown at NBPGR, New Delhi. YMD in most of the accessions was due to MYMIV infection whereas MYMV infection was confirmed in one accession of *V. hainiana* (IC331450) (Gautam *et al.*, 2014).

Since these species/subspecies are exploited for creating variability for different characters including disease resistance in cultivated species, it is essential to accurately identify the causal virus of yellow mosaic disease of wild accessions before using them as parents.

PCR diagnostic kit

Fig. 19 : LYMVs diagnostic kit

Information generated from our work on detection of YMD causing viruses in leguminous hosts and weeds described above was used to develop PCR kits (Fig. 19) for the accurate identification of the viruses causing yellow mosaic disease in pulses (MYMIV, MYMV, HgYMV, DoYMV). A PCR based detection kit has been developed and named as "LYMVs PCR Diagnostic Kit". This kit consists of a working protocol, four pairs of primers (AV1F/AV1R or MYMIV-CPF/MYMIV-CPR, YMV-FA/MYMV-R11, HgYMVcp14-F/ HgYMVcp14-F and DYMVcp14-F/ DYMVcp14-R specific for the amplification of target virus in the samples, DNA of all the four target viruses to be used as "Positive Control", master mix (2x) and nuclease free water.

Procedure for detection of LYMVs using kit

- Add 2x master mix (12.5 µl) in a PCR tube.
- Add 2 µl total DNA (10-20ng DNA) extracted from fresh tissue (leaf/stem/root) of mungbean, urdbean, cowpea, rajmash, soybean, horsegram and dolichos using any commercial Plant DNA extraction kit.
- For positive control, add 2 µl of target virus DNA as template in a separate tube.
- Add 1µl primer (25pmole/µl) each, AV1F/AV1R or MYMIV-CPF/MYMIV-CPR for the detection of MYMIV, YMV-FA/MYMV-R11 for the detection of MYMV, HgYMVcp14-F/ HgYMVcp14-F for the detection of HgYMV, DYMVcp14-F/ DYMVcp14-R for the detection of DoYMV in respective tubes.
- Add nuclease free water to make up the volume 25 µl.
- Give short vortex by machine or by pippeting, centrifuge briefly and place the tubes in thermal cycler.
- Thermal conditions-One step of initial denaturation at 94 ^{0}C for 2-3 minutes (temperature and duration may vary depending on the brand of the *Taq* polymerase used), 30-40 cycles involving denaturation at 94 ^{0}C for 1 minute, annealing at 54^{0}C for 1 minute and extension at 72 ^{0}C for 1 minute followed by one step of final extension at 72 ^{0}C for 10 minutes.
- Analyze the PCR products by loading 5-10 µl in 1% agarose gel.

- If the sample (es) is infected with LYMVs, the amplicon (DNA band) of ~950-1000 bp will be visible under UV light. It is possible to get expected size DNA bands (of target viruses) from a sample with different primers pair indicating the mixed infection of LYMVs.

  This kit was validated using the number of samples mentioned below:

Crop	Number of sample tested	Virus detected			
		MYMIV	MYMV	HgYMV	DoYMV
Mungbean	23	20	3	-	1
Cowpea	2	2	-	-	-
Dolichos	10	-	-	-	10
Rajmash	5	5	-	-	-
Ageratum	8	4	-	-	-
Pigeonpea	5	4	-	-	-
Total	**53**	**35**	**3**	**-**	**11**

Out of 53 samples tested 35 were found infected with MYMIV, 3 with MYMV and 11 with DoYMV.

For simultaneous detection of LYMVs (MYMIV, MYMV, HgYMV and DoYMV) also a PCR based kit has been developed. The time and cost is reduced to 1/4th, if this is being used for the detection of LYMVs. The PCR is performed in single tube for the detection of four viruses. This kit comprises of one forward primer (common for all four viruses) and four reverse primers (specific for each virus), positive DNA of all four target viruses, master mix (2X) and nuclease free water. This kit is named as "LYMVsMplex". Each primer amplifies the target virus, which is distinguished based on the size of amplicons. This protocol is in process for patenting.

Management

Management of YMD in mungbean and urdbean has been attempted by many workers. Approached adopted for the management of YMD are described here.

Nutrients

Soil application of boron at 3 kg/ha and molybdenum at 2 kg/ha reduced incidence of YMD and increased grain yield in mungbean in Bangladesh (Jalaluddin *et al*., 2006). Application of nitrogenous and phosphorous fertilizers was found to encourage the build-up of whitefly population as well as incidence of the disease in mungbean (Yein and Singh, 1982). On the other hand, in a pot experiment, Sekhar and Chand (2001) found highest reduction in yellow mosaic disease (53.34%) when 20 kgN+40 kg P+50 kg K/ha were applied. K and P at 40 kg/ha were also effective against YMD. However, increase in N rate with 40 kg P/ha increased the severity of YMD.

Intercrops

Intercrops of safflower, sunflower and maize were effective in delaying the onset and subsequent development of yellow mosaic disease in urdbean (Thakur and Agrawal, 1998). Borah *et al* (1996) found that use of cotton as a trap crop, sown one month ahead between the green gram rows with a single spray of dimethoate 0.03% at 15 days after germination of mungbean, effectively controlled both *B. tabaci* (11.83/10 plants) and yellow mosaic virus (7.81%). Even the intervarietal intercropping of mungbean and urdbean has been reported effective in reducing the incidence of YMD in both mungbean and urdbean (Dasgupta and Chowdhury, 1985).

Sowing dates

Adjusting sowing dates has also been advocated to reduce losses caused by YMD in mungbean and urdbean. Sowing early or late may depend on the agroecological zone. In North India, early sown crops of mungbean and urdbean in *kharif* season get more YMD than the crops sown late. Dubey and Singh (2006) found that under Delhi conditions, incidence and severity of YMD was more in crops sown early (1 July) than crops sown late (15, 30 July, 15 Aug). 15 July was the best sowing date as the crop gave significantly higher grain yield and got moderate disease. Disease was reduced in crops sown on 30 July and later, but the yield was also declined. In Orissa also, mungbean crop sown early (1 November) got less YMD than in crops sown late (16 November) (Nayak and Patra, 2000). In northern Telangana, sowing of *rabi* mungbean and urdbean early (25 September) results in higher incidence of YMD than in crops sown late (26 October) (Reddy *et al*., 1996). In Assam, Nath (1994) reported that mungbean crop sown between 1-15 February got less population of whitefly and incidence of YMD than the crops sown later.

Rouging

Rouging of infected plants helped reduce incidence of yellow mosaic disease and increased yield of mungbean (Jalaluddin *et al*., 2006, Islam *et al.*, 2002).

Botanicals

A number of plant extracts have also been tried for management of yellow mosaic disease in mungbean and urdbean. Of them, neem products are most commonly used plant products. Singh *et al*. (2004) found that six weekly foliar sprays of aqueous root extract of *Boerhaavia diffusa* starting from the seedling stage were most effective in delaying the symptom appearance, reducing the disease severity and decreasing the disease incidence by 80-90%. Treatments also increased root nodulation, plant height, primary and secondary branches, pod formation and grain yield. Gupta and Pathak (2009) studied many admixtures of neem products and insectides for their efficiency against incidence of YMD and its vector whitefly in urdbean in Bundelkhand zone of Madhya Pradesh and found that admixture of neem seed kernel extract (NSKE) (in cow urine) @ 3%+dimethoate @ 0.03% and of neem oil @ 0.5%+dimethoate @ 0.03% to be effective in reducing the incidence not only of whitefly and yellow mosaic but also of pod borer. These treatments also increased grain yield and resulted in net profit of Rs 3934 and Rs 3320/ha with incremental cost benefit ratio of 11.2 and 10.9, respectively. Eight sprays of extracts of *Clerodendrum aculeatum*, root extract of *Boerhaavia diffusa* and neem leaf extract reduced the YMD incidence by 66.70, 60.27, 42.43%, respectively in mungbean, and by 63.65, 58.20, 42.93%, respectively in urdbean. All these treatments also increased plant growth and nodulation in both the crops (Singh and Awasthi, 2009). Sethuraman *et al.* (2001) found foliar sprays of different formulations of neem oil @ 3%, neem seed kernel extract @5% at 35 and 55 days after sowing to be as effective as monocrotophos in reducing the YMD disease in urdbean. Foliar sprays of 5% leaf extracts of pungam, nochi and *Bougainvillea* were however not found effective in reducing the disease incidence. Hossain *et al.* (2010) reported 18.67% decline in YMD incidence and 17.65% increase in grain yield of mungbean by foliar spray of neem leaf extract.

Seed priming

Seed priming for 8 h with water before sowing reduced incidence of YMD in mungbean by about 70%. Seed priming also reduced YMD severity. The differences between priming treatments in the incidence and severity of disease were reflected in increased yield (Rashid *et al*., 2004).

Insecticides for the control of whiteflies

A disease can be managed by targeting the cause of the disease. The fungal and bacterial diseases are managed by applying fungicides and bactericides, but in case of diseases caused by viruses chemicals are not used to target the virus in the plant. However, managing the vector of a virus (disease) helps reduce spread of the virus (disease). A number of publications are available on the use of insecticides in managing the YMD in mungbean and urdbean.

Seed treatment, foliar spray and soil application with insecticides

Seed treatment with thiomethoxam 25 WG @ 0.0035% and imidacloprid 70 WS @ 0.0035% though protected the crop from whiteflies up to 25 days after sowing but later resulted in more vector population (6.00 & 7.33/5 plants) and high MYMV incidence (19.8% & 24.3%) than the other treatments (Panduranga *et al*., 2011). Studies conducted by Agrawal *et al.* (1979) at Hardoi in Uttar Pradesh revealed efficacy of 2 applications (a fortnight apart) of dichlorvos, monocrotophos or phosphamidon in reducing the YMD in both mung and urd. A combination of Aureofungin (0.003%) and Dimecron (0.25 kg a.i./ha) decreased incidence of mungbean yellow mosaic virus in mungbean and increased yield (Ahmad and Gaur, 1982). In Himachal Pradesh, application of Octanol + demeton-S-methyl (as Metasystox) gave the best control of YMD and resulted in harvesting higher yields in blackgram (Singh and Sirohi, 1997). Ghosh (2008) found imidacloprid at 12.5 kg/ha, applied at 15- and 30-days stage of greengram crop grown as direct-sown under residual soil moisture conditions after the harvest of *kharif* rice (rainfed lowland) effective in reducing YMD and increasing grain yield. In further studies, Ghosh *et al*. (2009) found Imidacloprid (Confidor @ 0.25 ml/l) effective in reducing the YMD incidence in both pre- and post- *kharif* season by more than 40%. They advocated imidacloprid as an alternative to monocrotophos. No significant difference was found between Thiomethoxam was as effective as monocrotophos in reducing the disease and was therefore, also a potential replacement for Monocrotophos. Two sprays of imidacloprid @ 0.24 ml/l along with imidacloprid @ 5 g/kg seed treatment and 2 sprays of imidacloprid @ 0.24 ml/l alone recorded least mean disease incidence of 30.2 and 30.3% with least vector populations of 1.87 and 1.73 whiteflies leaf. (Salam et al. 2009). Ganapathy and Karuppiah (2004) also found the application of thiamethoxam @ 0.2 g /litre at 15 DAS effective in decreasing the whitefly population and YMD incidence and increased yield. Panduranga *et al.* (2011) evaluated inecticides for managing the YMD and found foliar spray of thiomethoxam 25 WS @ 0.005%, spirotetramat 150 OD @ 90 g a.i./ha and acetamprid 20% SP @ 0.002% to be the most effective treatments in reducing the whitefly population and YMD incidence.

Granular insecticides have also been used in managing the whitefly and YMD in mungbean and urdbean. Four granular insecticides *viz*., aldicarb 10G, carbofuran 3G, disulfoton 5G and phorate 10G applied by broadcasting before sowing, in furrows alongwith seed, in furrows below the seed and broadcasting at the time of hoeing applied at 1.5 kg a.i./ha significantly reduced not only YMD but also galerucid beetle (*Madurasia obscurella*), stemfly (*Melanagromyza phaseoli* [*Ophiomyia phaseoli*]), pod borer (*Euchrysops cnejus*) in mungbean (Pandey *et al*., 2007). Sharma and Varma (1982) found aldicarb, phorate or disulfoton granules at 2 kg a.i./ha or a combination of disulfoton granules and paraffin oil as most effective in controlling the spread of the YMD and consequently in increasing yield. Application of aldicarb at a rate of 1.5 kg a.i./ha in the furrow before sowing gave best control of *B. tabaci* populations (66-67% at 5 weeks after sowing) and also incidence of YMD by 31-39% in mungbean (Yein and Singh, 1982). Soil application of Solvirex at sowing followed by 3 weekly foliar sprays of Metasystox beginning after 20 days after

sowing lowered the spread of YMD in mungbean crop and increased grain yield (Singh and Varma, 1977).

Based on two years' experimentation a treatment involving seed treatment with imidacloprid 17.8SL @ 5ml/kg seeds and two foliar sprays with the insecticide Nurelle D505 (50% chlorpyriphos and 5% cypermethrin) @0.1% at 15 and 45 days after sowing was found best in significantly increasing the grain yield in mungbean cv. T44 at Kanpur. None of the treatments used significantly reduced the yellow mosaic disease (Akram and Naimuddin, 2016).

Resistant varieties

Like other virus diseases, YMD is difficult to manage through chemicals. In fact mungbean and urdbean are relatively short duration crops and are grown mostly under rain fed conditions and therefore application of insecticides is not very practical. Efficacy of the insecticidal sprays may not last up to the desired period in rainy season. Though the insecticides kill the whitefly, the vector of the YMD and also the other insect pest on these crops, but whitefly being polyphagous and highly active moves to other fields/hosts and as soon as effect of insecticides diminishes, they again build up in these crops. Therefore, use of resistant varieties offer a cheap and best mean of avoiding losses caused by YMD. A number of YMD resistant varieties of mungbean and urdbean have been developed and released for cultivation in different parts of the country. Some of the YMD resistant varieties developed under All India Coordinated Research Programme on MULLaRP (mungbean, urdbean, lentil, lathyrus, rajmash and pea) are given in Table 6.

Table 6. YMD resistant varieties of mungbean and urdbean recommended for cultivation in different zones

Zone	YMD resistant varieties	
	Mungbean	Urdbean
NEPZ	Pant Mung 4, Meha, Samrat, HUM 16, HUM 12, TMB 37	Uttara, Azad Urd 1, WBU109
NWPZ	Ganga-8, IPM 02-3, MH 2-15 (Sattaya)	IPU 94-1 (Uttara), KU 300, KUG 479
CZ	HUM 1, PKV AKM 4	RBU 38, KU 96-3, NUL 7
SZ	CO 6, OUM 11-5, COGG 912, IPM 02-14 (for spring/summer), PKV AKM 4, HUM 1	WBG 26, TU 94-2 (for rabi), KU 301 (for rabi), TU 40 (for rabi), LU 391, IPU 02-43, IPU 07-3, VBG 04-008
NHZ	Pant Mung 6, KM 2241 , Pusa 0672	NDU 99-2 (Varieties Pant U 31 and Pant U 40 are suitable for lower hills and plains of Uttarakhand and Himachal Pradesh)

Transgenic approach for YMD management

Management of YMD is primarily through the control of its vector, whiteflies by application of insecticides. It involves the possibility of emergence of pesticide-resistant forms of the vector besides environmental concerns. Many YMD resistant varieties have been released for cultivation in different parts of the country but the durability of the resistance has been questioned. This makes a case for exploiting genetically engineered resistance mostly pathogen (virus) derived against YMD.

Genetic engineering can be used to modify the genetic makeup of the existing varieties in such a way that the engineered plant does not allow multiplication of the target virus thereby rendering it resistant the latter. The idea that the resistance can be incorporated in to a susceptible crop by introducing a gene or part of gene of the pathogen in to the plant genome (Sanford and Jhonston, 1985) was translated in to a reality by Powell-Abel *et al.* (1986) when they demonstrated coat protein (CP) gene (of *Tobacco mosaic virus*, TMV) mediated resistance in tobacco. This was followed by a number of reports on coat protein mediated resistance (CPMR) targeted against RNA viruses of major groups (Beechey, 1997, Lecoq, 1997).

CPMR has also been exploited in case of begomoviruses. It works in case of monopartite geminiviruses (Kunik *et al*., 1994), but it is assumed that a CP-mediated strategy against bipartite geminiviruses will not produce a high level of resistance (Azzam *et al*., 1996, Frischmuth and Stanley, 1998) because nuclear shuttle protein (NSP) (gene located on DNA B) can substitute for function of CP in transport. However, CPs may have the potential for transgenic interference as geminivirus CPs control specific interactions with the virus vector (Briddon *et al*., 1990; Azzam *et al*., 1994; Hofer *et al*., 1997; Noris *et al*., 1998; Morin *et al*., 2000, Haq *et al*., 2011) and a mutated or non-functional CP may therefore impede the virus spread among its vectors in geminivirus infected fields.

Replication-associated protein gene (*Rep*) of geminiviruses is known to interact with various host factors to regulate the virus replication (Kong *et al*., 2000; Kong and Hanley-Bowdoin, 2002, Hanley-Bowdoin *et al*., 2004, Morilla *et al*., 2006) and with the geminivirus REn protein (Settlage *et al*., 2005), which induces the host genes required for geminivirus DNA accumulation (Selth *et al*., 2005). The crucial role *Rep* gene plays in the replication and accumulation of geminivirus in host makes *Rep* gene an excellent target for interference by the expression of mutant proteins. As at least some of these functions are virus non-specific, the targeting of *Rep* gene may also provide broader resistance against different geminiviruses (Hong and Stanley, 1995; Brunetti *et al*., 2001, Hong and Stanley, 1996; Noris *et al*., 1996; Lucioli *et al*., 2003; Chatterji *et al*., 2001).

Also, modified genes like *AC1* coding for *Rep* protein has also been shown to confer broad resistance in transgenic plants against different geminiviruses. *AC2* gene that encodes transcriptional activator protein (TrAP) and *AC3* gene codes replication enhancer (*REn*) proteins and *AC4* gene that plays role in suppressing the gene silencing mechanism of the host have also been used as transgene to confer resistance against geminiviruses. The subject of engineering resistance to geminiviruses has been nicely reviewed by Vanderschuren *et al.* (2007). Pathogen derived resistance has been demonstrated in transgenic tobacco having MYMV-Vig genes for coat

protein (*CP*), replication-associated protein (*Rep*)-sense, Rep-antisense, truncated *Rep* (*T-Rep*), nuclear shuttle protein (*NSP*) and movement protein (*MP*) (Shivaprasad *et al*., 2006; Sunitha *et al*., 2011, 2013). Virus (MYMV-Vig) accumulation was inhibited in transgenic tobacco plants harbouring the Rep-sense and T-*Rep* gene of MYMV Vig agroinoculated with partial dimers of MYMV-Vig (Shivaprasad *et al*., 2006). siRNA targeted against *TrAp* gene of *Mungbean yellow mosaic virus* has been shown to reduce virus multiplication in transgenic tobacco (Shanmugapriya *et al.,* 2015).

So far the results of genetic engineering or biotechnological approached have been encouraging in modifying the native immune system of the plant (transgenic) in a way that plants are protected against the geminivirus infection and it is very likely to lead researchers to use novel tools that can be exploited to stabilize the crop production in general and pulses in particular.

Integrated management

Varied approaches for the management of YMD have been used by different workers. It is however agreed that these strategies or approaches if integrated in suitable and need based manner to evolve an integrated management schedule for YMD may yield better results. An integrated approach that is envisaged here includes-

- Use of disease resistant varieties
- Seed treatment with insecticides
- Foliar spray of insecticides at 30 and 45 days after sowing in mungbean. Second spray can be delayed to coincide with flowering and podding. Further sprays may be given considering the pest population
- If possible, borders of crops like sorghum, pearl millet or maize may be planted around mungbean/urdbean crop
- Removal of weeds from in and around the crop field
- Removal of YMD affected plants particularly at early stage of crop.

Future Line of Research

Since more than one virus are involved in causing YMD, mapping of these viruses in the country is essentially required. Any change in the known reaction (to YMD) of a genotype needs to monitored and investigated. Mapping of YMD causing viruses in different seasons in different agro-ecological zones of the country will provide information on the spatial and temporal diversity in legumoviruses population structure. Once it is done, deploying appropriate resistance gene will be the best strategy for combating YMD and in mitigating the yield losses caused by this disease.

The genomes of mungbean and adzuki bean have been sequenced and their genomic resources need to be exploited more to identify markers linked to YMD resistance in pulse crops. Programme for exploiting YMD resistance genes in the wild relatives of pulse crops needs to be strengthened further. Genetic engineering using viral genes of legumoviruses has potential to develop transgenic pulses with broader resistance to YMD. This has been briefly described elsewhere in this publication under transgenic approaches for management of YMD.

Of late, many publications have emerged on characterization of legumoviuses in pulses including soybean and cowpea which have indicated complexity in the viral population. Though four distinct virus species are known to cause YMD in pulses, cases of multiple infections and association of DNA-B component of other species instead of cognate DNA-B present a complex problem that can be understood only through well planned studies. Also, association of DNA Beta satellite (β-DNA) with YMD causing viruses in pulses reported to exacerbate the disease symptoms in host is another area that needs to be investigated and taken care of especially in cases where there is change from the known host reaction against YMD under field conditions. There is need to have differential hosts for differentiation of virus species and strains. This will help in indicating that the variety developed for a particular zone is resistant to which virus species/strain and help avoid embarrassing situation when a YMD resistant genotype at one location turns out to be susceptible at other location. All these notwithstanding, efforts to identify sources of resistance against YMD should continue. Resistance of a genotype should be put to temporal and spatial tests before claiming it to be resistant. A policy is also required to restrict the movement of a variety in zones for which it has not been released for cultivation. But if it is necessary, it has to be only after proper testing against diseases and pests.

References

1. Acharya S, Gupta SK, Jamwal BS. 1993. Evaluation of local germplasm of urdbean from Jammu. *Indian Journal of Pulses Research* 6:198-199.
2. Agrawal HS, Gupta NK, Prasad VK and Vishwakarma S. 1979. Chemical control of yellow mosaic of moong. *Pesticides* 13: 44-47.
3. Ahmad M and Harwood RF. 1973. Studies on whitefly transmitted yellow mosaic of urdbean (*Phaseolus mungo*). *Plant Disease Reporter* 57:800-802.
4. Ahmad M. 1975. Screening of mungbean (*Vigna radiata*) and urdbean (*V. mungo*) germplasms for resistance to yellow mosaic virus. *Journal of Agricultural Research* 13: 349-354.
5. Ahmad Q. 1991. Growth attributes and grain yield of mungbean plants affected by mungbean yellow mosaic virus in field. *Indian Phytopathology* 43: 559-560.
6. Ahmed SR and Gaur RB. 1982. Chemotherapeutic approach to check yellow mosaic virus of moong (*Phaseolus aureus* L.). *Hindustan Antibiotics Bulletin* 24: 18-20.
7. Akhtar KP, Sarwar G, Abbas G, Asghar MJ, Nighat S and Shah TM. 2011. Screening of mungbean germplasm against mungbean yellow mosaic India virus and its vector *Bemisia tabaci*. *Crop Protection* 30: 1202-1209.
8. Akram M and Naimuddin. 2016. Management of mungbean yellow mosaic disease and effect on grain yield. *Indian Journal of Plant Protection* 44:127-131
9. Akram M, Naimuddin, Agnihotri AK, Gupta S and Singh NP. 2015. Characterization of full genome of *Dolichos yellow mosaic virus* based on sequence comparison, genetic recombination and phylogenetic relationship. *Annals of Applied Biology* 67:354-363.
10. Alabi OJ, Kumar PL, Mgbechi-Ezeri JU and Naidu RA. 2010. Two new 'legumoviruses' (genus Begomovirus) naturally infecting soybean in Nigeria. *Archives of Virology* 155: 643-656.
11. Amin KS and Singh RA. 1989. Search for stable resistance to yellow mosaic virus in mungbean. National Symposium on New Frontiers in Pulses Research and Development, 10-12 November, 1989. Indian Society for Pulses and Development, DPR, Kanpur, India, pp. 122-123.
12. Ammavasai S, Phogat DS and Solanki IS. 2004. Inheritance of resistance to mungbean yellow mosaic virus (MYMV) in green gram [*Vigna radiata* (L.) Wilczek]. *Indian Journal of Genetics and Plant Breeding* 64:146.

13. Argüello-Astorga GR, Guevara-González LR, Herrera-Estrella LR and Rivera-Bustamante RF. 1994, Geminivirus replication origins have a group-specific organization of iterative elements: a model for replication. *Virology* 203: 90–100.

14. Arutkani KE, Ayyanathan and Chandrasekaran A. 1999. Evaluation of greengram genotypes for resistance to yellow mosaic disease. *Madras Agricultural Journal* 86: 527-528.

15. Aski M, Dikshit HK, Singh D, Singh A and Prapti P. 2015. Inheritance of resistance to Mungbean Yellow Mosaic India Virus (MYMIV) in mungbean (*Vigna radiata*). *Indian Journal of Agricultural Sciences* 85: 1144-1147.

16. Azzam O, Diaz O, Beaver JS, Gilbertson RL, Russell DR and Maxwell DP. 1996. Transgenic beans with the bean golden mosaic geminivirus coat protein gene are susceptible to virus infection. *Annual report of the Bean Improvement Cooperative* 39: 276–277.

17. Azzam O, Frazer J, de la Rosa D, Beaver JS, Ahlquist P and Maxwell DP. 1994. Whitefly transmission and efficient ssDNA accumulation of bean golden mosaic geminivirus require functional coat protein. *Virology* 204: 289–296.

18. Bag MK, Gautam NK, Prasad TV, Pandey S, Dutta M and Roy A. 2014. Evaluation of an Indian collection of black gram germplasm and identification of resistance sources to *Mungbean yellow mosaic virus*. *Crop Protection* 61: 92-101.

19. Banks GK, Colvin J, Chowda Reddy RV, Maruthi MN, Muniyappa V, Venkatesh HM, Kiran Kumar M, Padmaja AS, Beitia FJ and Seal SE. 2001. First report of the *Bemisia tabaci* B Biotype in India and an associated *Tomato leaf curl virus* disease epidemic. *Plant Disease* 85: 231.

20. Barnabas AD, Radhakrishnan GK and Ramakrishnan U. 2010. Characterization of a begomovirus causing horsegram yellow mosaic disease in India. *European Journal of Plant Pathology* 127:41-51.

21. Basandrai AK, Gartan SL, Basandrai D and Kalia V. 1999. Blackgram (*Phaseolus mungo*) germplasm evaluation against different diseases. *Indian Journal of Agricultural Sciences* 69: 506-508.

22. D, Basandrai AK, Singh I and Kalia V. 2003. Multiple disease resistance against anthracnose, cercospora leaf spot, powdery mildew and mungbean yellow mosaic virus in blackgram (*Vigna mungo*). *Journal of Mycology and Plant Pathology* 33:56-58.

23. Bashir M and Zubair M. 1992. Identification of resistance in urdbean (*Vigna mungo*) against two different viral diseases. *Pakistan Journal of Botany* 34: 49-51.

24. Bashir M, Jamali AR and Ahmad Z. 2006. Genetic resistance in mungbean and mashbean germplasm against mungbean yellow mosaic begomovirus. *Mycopathology* 4: 1-4.

25. Beachy RN. 1997. Mechanisms and applications of pathogen-derived resistance in transgenic plants. *Current Opinion in Biotechnology* 8: 215-220.

26. Biswas KK and Varma A. 2000. Identification of variants of mungbean yellow mosaic geminivirus by host reaction and nucleic acid spot hybridization. *Indian Phytopathology* 53:134-141.

27. Biswas KK and Varma A. 2001. Evaluation of resistance in blackgram (Phaseolus mungo) to variants of mungbean yellow mosaic geminivirus. *Indian Journal of Agricultural Sciences* 71: 215-218.

28. Biswas KK, Malathi VG and Varma A. 2005. Resistance in mungbean against five variants of Mungbean yellow mosaic virus. *Indian Journal of Virology* 16: 27-31.

29. Borah RK, Nath PD and Deka N. 1996. Effect of insecticides and crop trap on the incidence of white fly *Bemisia tabaci* (Genn.) and yellow mosaic virus in greengram *Vigna radiata* (L.) Wilczek. *Indian Journal of Virology* 12:75-77.

30. Briddon RW, Patil BL, Basavaraj B, Nawaz-ul-Rehman MS and Fauquet CM. 2010. Distinct evolutionary histories of the DNA-A and DNA-B components of bipartite begomoviruses. *BMC Evolutionary Biology* 10:97. DOI: 10.1186/1471-2148-10-97

31. Briddon RW, Pinner MS, Stanley J and Markham PG. 1990. Geminivirus coat protein gene replacement alters insect specificity. *Virology* 177: 85–94.

32. Brown JK. 2000. Molecular markers for the identification and global tracking of whitefly vector-*Begomovirus* complexes. *Virus Research* 71: 233-260.

33. Brown JK. 2007. The *Bemisia tabaci* Complex: Genetic and Phenotypic Variability Drives Begomovirus Spread and Virus Diversification. Online. APS*net* Features. doi: 10.1094/APSnetFeature-2007-0107.

34. Brunetti A, Tavazza R, Noris E, Lucioli A, Accotto GP and Tavazza M. 2001. Transgenically expressed T-Rep of tomato yellow leaf curl Sardinia virus acts as a trans-dominant-negative mutant, inhibiting viral transcription and replication. *Journal of Virology* 75:10573–10581.

35. Capoor SP and Varma PM. 1950. A new virus disease of *Dolichos lablab*. *Current Science* 19: 248–249.

36. Capoor SP and Varma PM. 1948. Yellow mosaic of *Phaseolus lunatus*. *Current Science* 17:152–153.

37. Cayalvizhi B, Nagarajan P, Raveeendran M, Rabindran R, Selvam NJ, Bapu JRK and Senthil N. 2015. Unravelling the responses of mungbean (*Vigna radiata*) to mungbean yellow mosaic virus through 2D-protein expression. *Physiological and Molecular Plant Pathology*, 90:65-77.

38. Chand P and Varma JP. 1980. Some characteristics of mungbean and urdbean varieties resistant and susceptible to yellow mosaic virus. *Indian Phytopathology* 33: 48-53.

39. Chand P and Varma JP. 1983. Effect of yellow mosaic on growth components and yield of mungbean and urdbean. *Haryana Agricultural University Journal of Research* 13:98-102.

40. Chatterji A, Beachy RN and Fauquet CM. 2001. Expression of the oligomerization domain of the replication-associated protein (Rep) of tomato leaf curl New Delhi virus interferes with DNA accumulation of heterologous geminiviruses. *Journal* of *Biological Chemistry* 276: 631–25 638.

41. Chatterji A, Chatterji U, Beachy RN and Fauquet CM. 2000. Sequence parameters that determine specificity of binding of the replication associated protein to its cognate site in two strains of Tomato leaf curl virus-New Delhi. *Virology* 273: 341–350.

42. Chaudhary, GG, Mathur AK and Tyagi RNS. 1981. Reaction of moong cultivars to yellow mosaic virus of moong (*Phaseolus aureus*) in Rajasthan. *Indian Journal of Mycology and Plant Pathology* 11: 273-274.

43. Chenulu VV, Venkateshwarlu V and Rangaraju R. 1979. Studies on yellow mosaic diseases of mungbean. *Indian Phytopathology* 32:230-235.

44. Chhabra KS and Kooner BS. 1981. Field resistance in black gram, *Vigna mungo* L. against insect-pests complex and yellow mosaic virus. *Indian Journal of Entomology* 43:288-293.

45. Chhabra KS and Kooner BS. 1994. Reaction of some promising mungbean genotypes towards whitefly, jassid and yellow mosaic virus. *Pest Management and Economic Zoology* 2:11-14.

46. Chhabra KS, Kooner BS and Singh G. 1979. *Journal of Research* 16:385-388. PAU, Ludhiana.

47. Chu H-chi, Buckner JS, Karut K, Freeman TP, Nelson DR, Hennebeny TJ. 2003. A survey of sizes and weights of *Bemisia tabaci* (Homoptera: Aleyrodidae) B biotype life stages from field grown cotton and cantaloupes. *Insect Science* 10:121–129.

48. Dasgupta B and Chowdhury AK. 1985. Use of intervarietal intercropping to minimize the yellow mosaic virus of urd and mung beans. *Indian Journal of Plant Pathology* 3: 100-101.

49. Deng D, Mcgrath PF, Robinson DJ and Harrison BD. 1994. Detection and differentiation of whitefly-transmitted geminiviruses in plants and vector insects by the polymerase chain reaction with degenerate primers. *Annals of Applied Biology* 125: 327–336.

50. Dhole VJ and Reddy KS. 2012. Genetic analysis of resistance to mungbean yellow mosaic virus in mungbean (*Vigna radiata*). *Plant Breeding* 131: 414-417.

51. Dhole VJ and Reddy KS. 2013. Development of a SCAR marker linked with a MYMV resistance gene in mungbean (*Vigna radiata* L. Wilczek). *Plant Breeding* 132:127-132.

52. Dubey SC and Singh B. 2006. Integrated management of cercospora leaf spots and yellow mosaic of urdbean (*Vigna mungo*). *Indian Journal of Agricultural Sciences* 76:485-489.

53. Equbal MJ, Sahni S, Singh D and Kumar B. 2015. Exploitation of urdbean germplasms for *Mungbean yellow mosaic virus* (MYMV) management and improved seed yield. *International Journal of Agriculture Sciences* 7: 536-539.

54. Fauquet CM, Briddon RW, Brown JK, Moriones E, Stanley J, Zerbini FM and Zhou X. 2008. Geminivirus strain demarcation and nomenclature. *Archives of Virology* 153:783–821.

55. Fontes EPB, Eagle PA, Sipe PS, Luckow VA and Hanley-Bowdoin L. 1994. Interaction between a geminivirus replication protein and origin DNA is essential for viral replication. *Journal of Biological Chemistry* 269:8459–8465.

56. Fontes EPB, Luckow VA and Hanley-Bowdoin L. 1992. A geminivirus replication protein is a sequence-specific DNA binding protein. *Plant Cell* 4: 597–608.

57. Frischmuth S and Stanley J. 1998. Recombination between viral DNA and the transgenic coat protein gene of African cassava mosaic geminivirus. *Journal of General Virology* 79: 1265–1271.

58. Ganapathy T and Karuppiah R. 2004. Evaluation of new insecticides for the management of whitefly (*Bemisia tabaci* Genn.), Mungbean yellow mosaic virus (MYMV) and urdbean leaf crinkle virus (ULCV) diseases in mungbean (*Vigna radiata* (L.) Wilezek). *Indian Journal of Plant Protection* 32:35-38.

59. Gautam, NK, Akram M, Akhtar J, Khan Z, Dwivedi NK, Latha M and Ram B. 2014. Responses of wild Vigna species/sub-species to yellow mosaic disease viruses, detected by a PCR-based method. *Phytopathologia Mediterrenea* 53:428-437.

60. Ghosh A. 2008. Management of yellow mosaic virus by chemical control of its vector, Whitefly (*Bemisia tabaci*) and its impact on performance of green gram (*Phaseolus aureus*) under rainfed lowland rice fallow. *Archives of Phytopathology and Plant Protection* 41: 75-78.

61. Ghosh D, Laha SK and Biswas NK. 2009. Effect of different pesticides on incidence of mungbean yellow mosaic virus incidence. *International Journal of Plant Protection* 2: 67-70.

62. Gill CK and Singh L. 1999. Effect of mungbean yellow mosaic virus on yield components in mungbean cultivar. *Insect Environment* 5: 112-113.

63. Girish KR and Usha R. 2005. Molecular characterization of two soybean-infecting begomoviruses from India and evidence for recombination among legume-infecting begomoviruses from South-East Asia. *Virus Research* 108:167-176.

64. Gopalaswamy SVS, Ramana MV and Krishna YR. 2012. Screening of black gram entries for resistance to insect pests and diseases under rice fallows. *Annals of Plant Protection Sciences* 20: 237-238.

65. Govindan K, Nagarajan P and Angappan K. 2014. Molecular studies on transmission of mung bean yellow mosaic virus (MYMV) by *Bemisia tabaci* Genn. in mungbean. *African Journal of Agricultural Research* 9:2874-2879.

66. Greathead AH. 1986. Host Plants. In: *Bemisia tabaci* - a literature survey on the cotton whitefly with an annotated bibliography (Ed. M.J.W. Cock). CAB International Institute of Biological Control, Ascot, UK. Pp 7-25.

67. Gupta MP and Pathak RK. 2009. Bioefficacy of neem products and insecticides against the incidence of whitefly, yellow mosaic virus and pod borer in Black gram. *Natural Product Radiance* 8:133-136.

68. Gupta O and Mishra M. 2014. Field resistance in mungbean and urdbean genotypes against yellow mosaic disease. *Journal of Food Legumes* 27:80-81

69. Gupta O. 2003. Resistance to Mungbean yellow mosaic virus, phenotypic characters and yield components in urdbean. *Indian Phytopathology* 56: 110-111.

70. Gupta S, Kumar S, Singh RA and Chandra S. 2005. Identification of a single dominant gene for resistance to Mungbean Yellow Mosaic Virus in blackgram (*Vigna mungo* (L.) Hepper). *SABRAO Journal of Breeding and Genetics* 37: 85-89.

71. Gupta S, Sen Gupta D, Anjum TK, Pratap A and Kumar J. 2013. Inheritance and molecular tagging of MYMIV resistance gene in blackgram (*Vigna mungo* L. Hepper). *Euphytica* 193:27-37.

72. Gurha SN, Misra DP and Kamthan KP. 1982. Studies on some aspects of yellow mosaic disease of black gram (*Vigna mungo* (L.) Hepper). *Madras Agricultural Journal* 69: 435-438.

73. Gurha SN, Misra DP and Kamthan KP. 1982. Studies on some aspects of yellow mosaic disease of black gram (*Vigna mungo* (L.) Hepper). *Madras Agricultural Journal* 69: 435-438.

74. Ha C, Coombs S, Revill P, Harding R, Vu M and Dale J. 2008. Molecular characterization of begomoviruses and DNA satellites from Vietnam: additional evidence that the New World geminiviruses were present in the Old World prior to continental separation. *Journal of General Virology* 89:312–326.

75. Hamilton WDO, Bisaro DM, Coutts RHA and Buck KW. 1983. Demonstration of the bipartite nature of the genome of a single-stranded DNA plant virus by infection with the cloned DNA components. *Nucleic Acids Research* 11: 7387-7396.

76. Hanley-Bowdoin L, Settlage SB and Robertson D. 2004. Reprogramming plant gene expression: a prerequisite to geminivirus DNA replication. *Molecular Plant Pathology* 5: 149–156.

77. Hanley-Bowdoin L, Settlage SB, Orozco BM, Nagar S and Robertson D. 1999. Geminiviruses: models for plant DNA replication, transcription, and cell cycle regulation. *Critical Reviews in Plant Sciences* 18:71-106.

78. Haq QMI, Jyothsna P, Ali A and Malathi VG. 2011. Coat protein deletion mutation of *Mungbean yellow mosaic India virus* (MYMIV). *Journal of Plant Biochemistry and Biotechnology* 20: 182-189.

79. Harrison BD, Muniyappa V, Swanson MM, Roberts IM and Robinson DJ. 1991. Recognition and differentiation of seven whitefly-transmitted geminiviruses from India, and their relationship to African cassava mosaic and Thailand mungbean yellow mosaic viruses. *Annals of Applied Biology* 118:299-308.

80. Hofer P, Bedford ID, Markham PG, Jeske H and Frischmuth T. 1997. Coat protein gene replacement results in whitefly transmission of an insect nontransmissible geminivirus isolate. *Virology* 236: 288–295.

81. Honda Y, Iwaki M, Saito Y, Thongmeearkom P, Kittisak K and Deema N. 1983. Mechanical Transmission, Purification, and Some Properties of Whitefly-Borne Mung Bean Yellow Mosaic Virus in Thailand. *Plant Disease* 67:801-804.

82. Hong Y and Stanley J. 1995. Regulation of African cassava mosaic virus complementary-sense gene expression by N-terminal sequences of the replication-associated protein AC1. *Journal of General Virology* 76: 2415–2422.

83. Hong Y and Stanley J. 1996. Virus resistance in *Nicotiana benthamiana* conferred by African cassava mosaic virus replication-associated protein (AC1) transgene. *Molecular Plant-Microbe Interactions* 9: 219–225.

84. Hossain ASMA, Akhter K, Akhtar N, Muqit A and Islam SMAS. 2010. Effect of some management practices on the incidence of mungbean yellow mosaic and on grain yield. *Bangladesh Journal of Plant Pathology* 26: 9-13.

85. Hull R. 1986. The potential for using dot-blot hybridisation in the detection of plant viruses. In: Jones RAC and Torrance L (eds.) Developments in Applied Biology 1: Developments and Applications in Virus Testing (pp. 3–12) Association of Applied Biologists, Wellesbourne, UK.

86. Ilyas M, Qazi J, Mansoor S and Briddon RW. 2009. Molecular characterisation and infectivity of a "Legumovirus" (genus *Begomovirus*: family *Geminiviridae*) infecting the leguminous weed *Rhynchosia minima* in Pakistan. *Virus Research* 145:279-284.

87. Ilyas M, Qazi J, Mansoor S and Briddon RW. 2010. Genetic diversity and phylogeography of begomoviruses infecting legumes in Pakistan. *Journal of General Virology* 91:2091-2101.

88. Ilyas MB, Chaudhry MA and Ghazanfar MU. 2007. Screening of mungbean germplasm for the sources of resistance against mung bean yellow mosaic virus infection. *Pakistan Journal of Phytopathology* 19:101-104.

89. Ingham DJ, Pascal E and Lazarowitz SG. 1995. Both bipartite geminivirus movement proteins define viral host range, but only BL1 determines viral pathogenicity. *Virology* 207: 191-204.

90. Iqbal U, Iqbal SM, Rukhsana A, Jamal A, Farooq MA and Zahid A. 2011. Screening of mungbean germplasm against mungbean yellow mosaic virus (MYMV) under field conditions. *Pakistan Journal of Phytopathology* 23: 48-51.

91. Islam MR, Ali MA, Islam MS and Hossain AFMGF. 2002. Effect of nutrients and weeding on the incidence of mungbean mosaic. *Pakistan Journal of Plant Pathology* 1:48-50

92. Islam MN, Sony SK and Borna RS. 2012. Molecular characterization of Mungbean yellow mosaic disease and coat protein gene in mungbean varieties of Bangladesh. *Journal of Plant Tissue Culture and Biotechnology* 22:73-81

93. Jain R, Roopa LG, Ashok RP and Suresh BG. 2013. Genetic inheritance of yellow mosaic virus resistance in mungbean [*Vigna radiata* (L.) Wilczek]. *Trends in Biosciences* 6: 305-306.

94. Jalaluddin M, Khalil MI and Kashem MA. 2006. Management of yellow mosaic of summer mungbean with boron, molybdenum, rouging and insecticide. *Bangladesh Journal of Plant Pathology* 22:17-20.

95. John P, Sivalingam PN, Haq QMI, Kumar N, Mishra A, Briddon R W and Malathi VG. 2008. Cowpea golden mosaic disease in Gujarat is caused by a *Mungbean yellow mosaic India virus* isolate with a DNA B variant. *Archives of Virology* 153:1359-1365.

96. Jyothsna P, Rawar R, Malathi VG. 2011. Molecular characterization of a new begomovirus infecting a leguminous weed, *Rhynchosia minima* in India. *Virus Genes* 42:407-414.

97. Karim Z, Hossain MS and Islam MM. 2010. Screening of mungbean lines against mungbean yellow mosaic virus (MYMV) under field condition. *Bangladesh Journal of Plant Pathology* 26:79-80.

98. Karthikeyan A, Shobhana VG, Sudha M, Raveendran M, Senthil N, Pandiyan M, Nagarajan P. 2014. Mungbean yellow mosaic virus (MYMV): a threat to green gram (*Vigna radiata*) production in Asia. *International Journal of Pest Management* 60:314-324.

99. Karthikeyan AS, Vanitharani R, Balaji V, Anuradha S, Thillaichidambaram P, Shivaprasad PV, Parameswari C, Balamani V, Saminathan M and Veluthambi K. 2004. Analysis of an isolate of *Mungbean yellow mosaic virus* (MYMV) with a highly variable DNA B component. *Archives of Virology* 149:1643-1652.

100. Kaur L, Chadha ML, Kuo G and Gowda CLL. 2007. Multiple disease resistant sources of mungbean. *Acta Horticulturae* 752: 423-426.

101. Kaushal RP and Singh BM. 1988. Inheritance of disease resistance in blackgram (*Vigna mungo*) to mungbean yellow mosaic virus. *Indian Journal of Agricultural Sciences* 58: 123-124.

102. Khan MG, Ahmad W, Khattak GSS, Siraj-ud-Din and Ahmad H. 2007. Mode of inheritance of resistance to Mungbean Yellow Mosaic Virus (MYMV) in mungbean (*Vigna radiata* (L.) Wilczek). 23:1071-1074.

103. Khattak GSS, Haq MA, Rana SA, Abass G and Irfan M. 2000. Effect of mungbean yellow mosaic virus (MYMV) on yield and yield components of mungbean (*Vigna radiata* (L.) Wilczek). *Kasetsart Journal, Natural Sciences* 34: 12-16.

104. Khattak GSS, Haq MA, Rana SA, Srinives P and Ashraf M. 1999. Inheritance of resistance to mungbean yellow mosaic virus (MYMV) in mungbean (*Vigna radiata* (L.) Wilczek). *Thai Journal of Agricultural Science* 32: 49-54.

105. Khedar OP, Singh RV, Shrimali M and Majumdar VL. 2006. Management of yellow mosaic virus by locating resistant source in mungbean. *Journal of Arid Legumes* 3:75.

106. Kong LJ and Hanley-Bowdoin L. 2002. A geminivirus replication protein interacts with a protein kinase and a motor protein that display different expression patterns during plant development and infection. *Plant Cell* 14:1817–1832.

107. Kong LJ, Orozco BM, Roe JL, Nagar S, Ou S, Feiler HS, Durfee T, Miller AB, Gruissem W, Robertson D and Hanley-Bowdoin L. 2000. A geminivirus replication protein interacts with the retinoblastoma protein through a novel domain to determine symptoms and tissue specificity of infection in plants. *EMBO Journal* 19: 3485–3495.

108. Kothandaraman SV, Devadason A and Ganesan MV. 2016. Seed-borne nature of a begomovirus, *Mung bean yellow mosaic virus* in black gram. *Applied Microbiology and Biotechnology* doi:10.1007/s00253-015-7188-7.

109. Kumar A and Bal RS. 2012. Screening of urdbean germplasm against MYMV and Cercospora leaf spot diseases. *Trends in Biosciences* 5: 127-128.

110. Kumar R, Shamshad Ali and Rizvi SMA. 2006. Screening of mungbean genotypes for resistance against whitefly, Bemisiatabaci and mungbean yellow mosaic virus. *Indian Journal of Pulses Research* 19:135-136.

111. Kundu A, Patel A, Paul S and Pal A. 2015. Transcript dynamics at early stages of molecular interactions of MYMIV with resistant and susceptible genotypes of the leguminous host, *Vigna mungo*. *PLoS ONE*, 10:4 pp.e0124687.

112. Kunik T, Salomon R, Zamir D, Navot N, Zeidan M, Michelson I, Gafni Y and Czosnek H. 1994. Transgenic tomato plants expressing the tomato yellow leaf curl virus capsid protein are resistant to the virus. *Biotechnology* 12: 500–504.

113. Wu LC, Rogers SG and Elmer JS. 1992. Sequence-specific interaction with the viral AL1 protein identifies a geminivirus DNA replication origin. *Plant Cell* 4:799-809.

114. Lecoq H. 1997. Molecular biology and genetic engineering: new prospects for control of viral diseases of plants. *Comptes Rendus de l'Académie d'Agriculture de France* 83:153-160.

115. Lisha VS, Antony B, Palaniswami MS and Henneberry TJ. 2003. *Bemisia tabaci* (Homoptera: Aleyrodidae) Biotypes in India. *Journal of Economic Entomology* 96: 322-327.

116. Lucioli A, Noris E, Brunetti A, Tavazza R, Ruzza V, Castillo AG, Bejarano ER, Accotto GP and Tavazza M. 2003. Tomato yellow leaf curl Sardinia virus rep-derived resistance to homologous and heterologous geminiviruses occurs by different mechanisms and is overcome if virus-mediated transgene silencing is activated. *Journal of Virology* 77: 6785–6798.

117. Malathi VG. 2007. Genetic identity of yellow mosaic viruses infecting legumes and their phylogenetic relationship. *Indian Phytopathology* 60:143-155

118. Malathi VG and John P. 2008. Geminiviruses infecting legumes. In: Characterization, Diagnosis & Management of Plant Viruses, Volume 3: Vegetables and Pulse Crops (G.P. Rao, P. Lava Kumar and Ramon J. Holguin-Pena, Ed.), Stadium Press LLC, Texas, USA. Pp 97-123.

119. Malathi VG, Surendranath B, Naghma A and Roy A. 2005. Adaptation to new hosts shown by the cloned components of *Mungbean yellow mosaic India virus* causing cowpea golden mosaic in northern India. *Canadian Journal of Plant Pathology* 27: 439-447.

120. Mandal B, Varma A and Malathi VG. 1997. Systemic infection of *Vigna mungo* using the cloned DNAs of the blackgram isolate of mungbean yellow mosaic geminivirus through agroinoculation and transmission of the progeny virus by whiteflies. *Journal of Phytopathology* 145:505–510.

121. Mandal B, Varma A and Malathi VG. 1998. Some biological and genomic properties of pigeonpea isolate of mungbean yellow mosaic geminivirus. *Indian Phytopathology* 51:121-129.

122. Manivannan N, Sethuraman K and Natarajan S. 2001. Screening of greengram (*Vigna radiata* (L.) Wilczek) germplasm for yellow mosaic resistance. *Legume Research* 24: 268-271.

123. Marimuthu T, Subramanian CL and Mohan R. 1981. Assessment of yield loss due to yellow mosaic infection in mungbean. *Pulse Crops Newsletter* 1:104.

124. Maruthi MN, Rekha AR, Govindappa MR, Colvin J and Muniyappa V. 2006. A distinct begomovirus causes Indian dolichos yellow mosaic disease. *Plant Pathology* 55: 290.

125. Mayee CD and Datar VV.1986. Phytopathometry. Technical Bulletin-1, MAU, Prabhani, India, p.146.

126. McAuslane HJ. 2009. Featured creatures. Sweet potato whitefly B biotype or silver leaf whitefly *Bemisia tabaci* (Gennadius) or *Bemisia argentifolii* Bellows & Perring (Insecta: Hemiptera: Aleyrodidae) Publication Number: EENY-129 April 2009. University of Florida, USA, an online publication.

127. Mishra DK. 2003. Inheritance of yellow mosaic virus in mung bean. *JNKVV Research Journal* 37: 40-45.

128. Morilla G, Castillo AG, Preiss W, Jeske H and Bejarano ET. 2006. A versatile transreplication-based system to identify cellular proteins involved in geminivirus replication. *Journal of Virology* 80: 3624–3633.

129. Morin S, Ghanim M, Sobol I and Czosnek H. 2000. The GroEL protein of the whitefly Bemisia tabaci interacts with the coat protein of transmissible and non-transmissible begomoviruses in the yeast two-hybrid system. *Virology* 276: 404–416.

130. Morinaga T, Ikegami M and Miura K. 1990. Physical mapping and molecular cloning of mung bean yellow mosaic virus DNA. *Intervirology* 31:50-56.

131. Morinaga T, Ikegami M and Miura K. 1993. The nucleotide sequence and genome structure of mungbean yellow mosaic geminivirus. *Microbiology and Immunology* 37:471-476.

132. Mound LA and Halsey SH. 1978. *Bemisia tabaci* (Gennadius). In: Whitefly of the World- A Systematic Catalog of the Aleyrodidae (Homoptera) with Host Plant and Natural Enemy Data. British Museum (Natural History) and John Wiley & Sons, Chichester, New York, Brisbane, Toronto. pp. 118-124.

133. Munawwar MH, Ali A and Malik SR. 2014. Identification of resistance in mungbean and mashbean germplasm against mungbean yellow mosaic virus. *Pakistan Journal of Agricultural Research* 27:129-135

134. Muniyappa V, Reddy HR and Shivashankar G. 1975. Yellow mosaic disease *Dolichos biflorus* Linn. (horsegram). *Current Research* 4: 176.

135. Muniyappa V, Rajeshwari R, Bharathan N, Reddy DVR and Nolt BL. 1987. Isolation and characterization of a geminivirus causing yellow mosaic disease of horsegram (*Macrotyloma uniflorum* (Lam.) Verdc.) in India. *Journal of Phytopathology* 119: 81-87.

136. Murugesan S and Chelliah S. 1977. Transmission of green gram yellow mosaic virus by whitefly, *Bemisai tabaci*. *Madras Agricultural Journal* 64: 437-448.

137. Nadeem S, Mughal SM, Farooq K and Bashir M. 2006. Evaluation of mungbean germplasm for resistance against mungbean yellow mosaic begomovirus. *Pakistan Journal of Botany* 38: 449-457.

138. Naimuddin and Akram M. 2010. Detection of mixed infection of begomoviruses and their molecular characterization based on CP gene sequences. *Journal of Food Legumes* 23:191-195.

139. Naimuddin, Akram M and Gupta S. 2011a. Identification of *Mungbean yellow mosaic India virus* infecting *Vigna mungo* var. *silvestris* L. *Phytopathologia Mediterranea* 50: 94−100.

140. Naimuddin, Akram M and Pratap A. 2011b. First report of natural infection of *Mungbean yellow mosaic India virus* in two wild species of *Vigna*. *New Disease Reports* 23:21.

141. Naimuddin, Akram M, Gupta S and Agnihotri AK. 2014. *Ageratum conyzoides* harbours *Mungbean yellow mosaic India virus*. *Plant Pathology Journal* 13: 59-64.

142. Naimuddin, Akram M, Pratap A, Chaubey BK and Joseph KJ. 2011c. PCR based identification of the virus causing yellow mosaic disease in wild *Vigna* accessions. *Journal of Food Legumes* 24:14-17.

143. Nair NG and Nene YL. 1974. Studies on yellow mosaic of urdbean (*Phaseolus mungo* L.) caused by mungbean yellow mosaic virus. Factors influencing transmission and symptom expression. *Indian Journal of Farm Sciences* 2:42-47.

144. Nair NG. 1971. Studies on the yellow mosaic of urd bean (*Phaseolus mungo* L.) caused by mung bean yellow mosaic virus. Ph.D. Thesis, G.B. Pant Univ. Agriculture and Technology, Pantnagar, UP, India. Pp. 1-66.

145. Nariani TK. 1960. Yellow mosaic of mung (*Phaseolus aureus* L.). *Indian Phytopathology* 13:24-29.

146. Nath PD. 1994. Effect of sowing time on the incidence of yellow mosaic virus disease and whitefly population on greengram. *Annals of Agricultural Research* 15: 174-177.

147. Nayak BC and Patra AK. 2000. Response of greengram (Phaseolus radiatus) cultivars to dates of sowing during winter. *Indian Journal of Agricultural Sciences* 70: 320-322.

148. Nene YL. 1972. A survey of viral diseases of pulse crops in Uttar Pradesh. G.B. Pant Univ. Agriculture and Technology, Pantnagar, UP, India. pp.191.

149. Nene YL. 1973. Viral diseases of warm weather pulse crops in India. *Plant Disease Reporter* 57: 463–467.

150. Neupane RK, Darai R, Sah RP, Pokhrel DN and Shanmugasundaram S. 2006. Development of mungbean (*Vigna radiata* (L.) Wilczek) varieties for rice and maize-based systems in Nepal. In: AVRDC, Shanhua, Taiwan, Improving income and nutrition by incorporating mungbean in cereal fallows in the Indo-Gangetic Plains of South Asia DFID Mungbean Project for 2002-2004, proceedings of the final workshop and planning meeting, Punjab Agricultural University, Ludhiana, Punjab, India, 27-31 May 2004. 268-282pp.

151. Noris E, Accotto GP, Tavazza R, Brunetti A, Crespi S and Tavazza M. 1996. Resistance to tomato yellow leaf curl geminivirus in *Nicotiana benthamiana* plants transformed with a truncated viral C1 gene. *Virology* 224: 130–138.

152. Noris E, Vaira AM, Caciagli P, Masenga V, Gronenborn B and Accotto GP. 1998. Amino acids in the capsid protein of tomato yellow leaf curl virus that are crucial for systemic infetion, particle formation, and insect transmission. *Journal of Virology* 72: 10 050–10 057.

153. Obaiah S, Reddy BVB, Reddy NPE, Prasad YS. 2013. Screening of some blackgram Vigna mungo (L.) Hepper) genotypes for resistance to yellow mosaic virus. *Current Biotica* 7: 96-100.

154. Orozco BM, Gladfelter HJ, Settlage SB, Eagle PA, Gentry RN and Hanley-Bowdoin L. 1998. Multiple cis elements contribute to geminivirus origin function. *Virology* 242: 346–356.

155. Padidam M, Beachy RN and Fauquet CM. 1995. Classification and identification of geminiviruses using sequence comparisons. *Journal of General Virology* 76: 249–263.

156. Pal SS, Dhaliwal HS and Bains SS. 1991. Inheritance of resistance to yellow mosaic virus in some *Vigna* species. *Plant Breeding* 106: 168-171.

157. Pandey A, Pandey A and Srivastava SP. 2007. Management of pest complex of summer mung by granular insecticides. *Annals of Plant Protection Sciences* 15: 376-380.

158. Panduranga GS, Vijayalakshmi K and Reddy KL. 2011. Evaluation of insecticides for management of *Bemisia tabaci* and MYMV disease in mungbean [*Vigna radiata* (L.) Wilczek]. *Annals of Plant Protection Sciences* 19: 295-298.

159. Panigrahi KK and Baisakh B. 2013. Reaction of local and mutant lines of black gram (Vignamungo [L] Hepper) to yellow mosaic virus and powdery mildew. *Journal of Plant Protection and Environment* 10: 57-60.

160. Patel MB and Srivastava KP. 1990. Field screening of some high yielding genotypes of mungbean, *Vigna radiata* (Linnaeus) Wilczek to whitefly *Bemisia tabaci* (Gennadius) and yellow mosaic virus (YMV). *Indian Journal of Entomology* 52: 547-551.

161. Pathak AK and Jhamaria SL. 2004. Evaluation of mungbean (Vignaradiata L.) varieties to yellow mosaic virus. *Journal of Mycology and Plant Pathology* 34:64-65.

162. Perring TM. 2001. The *Bemisia tabaci* species complex. *Crop Protection* 20: 725-737.

163. Powell-Abel P, Nelson RS, De B, Hoffmann N, Rogers SG, Fraley RT and Beachy RN. 1986. Delay of disease development in transgenic plants that express the tobacco mosaic virus coat protein gene. *Science* 232: 738–743.

164. Prasad AVSD, Murugan E and Vanniarajan C. 2015. Inheritance of resistance of mungbean yellow mosaic virus in urdbean (*Vigna mungo* (L.) Hepper). Current Biotica 8: 413-417.

165. Prasanna HC, Kanakala S, Archana K, Jyothsna P, Varma RK and Malathi VG. 2015. Cryptic species composition and genetic diversity within *Bemisia tabaci* complex in soybean in India revealed by *mtCOI* DNA sequence. *Journal of Integrative Agriculture 14: 1786–1795*

166. Puchta H and Sanger HL. 1989. Sequence analysis of minute amounts of viroid RNA using the polymerase chain reaction (PCR). *Archives of Virology* 106: 335–340.

167. Qazi J, Ilyas M, Mansoor S and Briddon RW. 2007. Legume yellow mosaic viruses: genetically isolated begomoviruses. *Molecular Plant Pathology* 8:343–348.

168. Qazi J, Mansoor S, Amin I, Awan MY, Briddon RW and Zafar Y. 2006. First report of Mungbean yellow mosaic India virus on mothbean in Pakistan. *Plant Pathology* 55:818.

169. Raj SK, Aslam M, Srivastava KM and Singh BP. 1989. Association of geminivirus-like particles with yellow mosaic disease of *Dolichos lablab* L. *Current Science* 58: 813-815.

170. Rashid A, Harris D, Hollington P and Shamsher Ali. 2004. On-farm seed priming reduces yield losses of mungbean (*Vigna radiata*) associated with mungbean yellow mosaic virus in the North West Frontier Province of Pakistan. *Crop Protection* 23: 1119-1124.

171. Rathi YPS. 1972. Mungbean yellow mosaic virus: Host range and relationship with the vector, *Bemisia tabaci* Genn. Ph.D. Thesis, G.B. Pant Univ. Agriculture and Technology, Pantnagar, UP, India. Pp. 1-93.

172. Raychaudhari SP, Dhingra KL and Verma JP. 1977. Virus diseases of pulse crops in India. *Seed and Farms* 3:7-17.

173. Reddy BVB, Obaiah S, Prasanthi L, Sivaprasad Y, Sujitha A and Krishna TG. 2015. Mungbean yellow mosaic India virus is associated with yellow mosaic disease of blackgram (Vigna mungo L.) in Andhra Pradesh, India. *Archives of Phytopathology and Plant Protection* 48: 345-353.

174. Reddy KR and Singh DP. 1993. Inheritance of resistance to mungbean yellow mosaic virus. *Madras Agricultural Journal* 80: 199-201.

175. Reddy MD, Krishna A and Rao PS. 1996. Performance of promising greengram and blackgram varieties during *rabi* in red chalka soils of Northern Telangana. *Journal of Research ANGRAU* 24: 98-100.

176. Rogers SG, Bisaro DM, Horsch RB, Fraley RT, Hoffmann NL, Brand L, Elmer JS and Lloyd AM. 1986. Tomato golden mosaic virus A component DNA replicates autonomously in transgenic plants. *Cell* 45: 593-600.

177. Rojas MR, Gilbertson RL, Russell DR and Maxwell DP. 1993. Use of degenerate primers in the polymerase chain reaction to detect whitefly transmitted geminiviruses. *Plant Disease* 77:340-347.

178. Rouhibakhsh A and Malathi VG. 2005. Severe leaf curl disease of cowpea – a new disease of cowpea in northern India caused by Mungbean yellow mosaic India virus and a satellite DNA β. Plant Pathology 54:259.

179. Rouhibakhsh A, Choudhury NR, Mukherjee SK and Malathi VG. 2012. Enhanced nicking activity of Rep in presence of pre-coat protein of Mungbean yellow mosaic India virus. *Virus Genes* 44:356-361

180. Rouhibakhsh A, Haq QMI and Malathi VG. 2011. Mutagenesis in ORF AV2 affects viral replication in Mungbean yellow mosaic India virus. *Journal of Biosciences* 36:329-340.

181. Rouhibakhsh A, Priya J and Perisamy A. 2008. An improved DNA isolation method and PCR protocol for efficient detection of multicomponents of begomovirus in legumes. *Journal of Virological Methods* 147: 37-42.

182. Roy A and Malathi VG. 2001. Molecular cloning of cowpea golden mosaic geminivirus and its relationship with mungbean yellow mosaic geminivirus. *Tropical Agricultural Research* 13:341-352.

183. Saiki RK, Gelfand DH, Stoffel S, Scharf SJ, Higuchi R, Horn GT, Mullis KB and Ehrlich HA. 1988. Primer directed enzymic amplification of DNA with a thermostable polymerase. *Science* 239: 487–491.

184. Saiki RK, Scharf S, Faloona F, Mullis KB, Horn GT, Erlich HA and Arnheim N. 1985. Enzymatic amplification of Beta-globin genomic sequences and restriction site analysis for diagnosis of sickle cell anaemia. *Science* 230: 1350–1354.

185. Salam SA, Patil MS and Byadgi AS. 2009. IDM of mung bean yellow mosaic disease. *Annals of Plant Protection Sciences* 17: 157-160.

186. Saleem M, Haris WAA and Malik IA. 1998. Inheritance of yellow mosaic virus in mungbean (*Vigna radiata* L. Wilczek). *Pakistan Journal of Phytopathology* 10: 30-32.

187. Sanford JC and Johnston SA. 1985. The concept of parasite-derived resistance - deriving resistance genes from the parasite's own genome. *Journal of Theoretical Biology* 113: 395-405.

188. Satya VK and Alice D. 2014. Molecular detection of *Mungbean yellow mosaic virus* and their associated satellite DNA from field samples of Tamil Nadu. *Madras Agricultural Journal* 101:146-150

189. Satya VK, Alice D, Malathi VG and Velazhahan R. 2015. Molecular identification and genetic diversity of yellow mosaic virus in blackgram. *Biochemical and Cellular Archives* 15: 475-479.

190. Sekhar NR and Chand H. 2001. Effect of host nutrition on mung bean yellow mosaic virus, anthracnose, root rot and leaf crinkle of mungbean [*Vigna radiata* (L.) Wilczek]. *Indian Journal of Plant Protection* 29:88-91.

191. Selth LA, Dogra SC, Rasheed MS, Healy H, Randles JW and Rezaian MA. 2005. A NAC domain protein interacts with tomato leaf curl virus replication accessory protein and enhances viral replication. *Plant Cell* 17: 311–325.

192. Sethuraman K, Manivannan N and Natarajan S. 2001. Management of yellow mosaic disease of urdbean using neem products. *Legume Research* 24: 197-199.

193. Settlage SB, See RG and Hanley-Bowdoin L. 2005. Geminivirus C3 protein: replication enhancement and protein interactions. *Journal of Virology* 79: 9885–9895.

194. Shafiq M, Shaheen A, Zafar Y, Briddon RW and Mansoor S. 2010. Pepper leaf curl Lahore virus requires the DNA B component of Tomato leaf curl New Delhi virus to cause leaf curl symptoms. *Virology Journal* 7:367.

195. Shanmugapriya G, Das SS and Veluthambi K. 2015. Transgenic tobacco plants expressing siRNA targeted against the Mungbean yellow mosaic virus transcriptional activator protein gene efficiently block the viral DNA accumulation. *Virus Disease* 26: 55-61.

196. Sharma ML, Nayak MK, Bhadouria SS and Shams A. 2004. Screening of newly developed blackgram varieties against whitefly and yellow mosaic virus. *Shashpa* 11: 71-74.

197. Sharma SR and Varma A. 1982. Control of yellow mosaic of mungbean through insecticides and oil. *Journal of Entomological Research* 6: 130-136.

198. Shivaprasad PV, Thillaichidambaram P, Balaji V and Veluthambi K. 2006. Expression of full-length and truncated Rep genes from Mungbean yellow mosaic virus-Vigna inhibits viral replication in transgenic tobacco. *Virus Genes* 33: 365-374.

199. Shukla GP, Pandya BP and Singh DP. 1978. Inheritance of resistance to yellow mosaic in mungbean. *Indian Journal of Genetics and Plant Breeding* 38: 357-360.

200. Singh A and Sirohi A. 1997. Effect of various insecticides for the control of yellow mosaic of black gram. Plant Disease Research 12:37-38.

201. Singh SK, , Singh AK and Pandey PK. 2006. Cloning, restriction mapping and phylogenetic relationship of genomic components of MYMIV from *Lablab purpureus*. *Bioresource Technology* 97:1807-1814.

202. Singh A, Sirohi A and Panwar KS. 1998. Inheritance of mung bean yellow mosaic virus resistance in urdbean (*Vigna mungo* (L.) Hepper). *Indian Journal of Virology* 14: 89-90.

203. Singh BR, Singh M, Yadav MD and Dingar SM. 1982. Yield loss in mungbean due to yellow mosaic. *Science and Culture* 48: 435-436.

204. Singh DP. 1980. Inheritance of resistance to yellow mosaic virus in blackgram (Vigna mungo (L.) Hepper). *Theoretical and Applied Genetics* 57:233-235.

205. Singh G, Kapoor S, Verma MM, Gill KS, Khehra AS and Bains KS. 1987. Urdbean lines resistant to yellow mosaic virus. (Abstract: pp. 103-104). First symposium on crop improvement, 23-27 February, 1987. Crop Improvement Society of India, Ludhiana, India.

206. Singh G, Singh S and Sheoran OP. 2013a. Inheritance of Mungbean Yellow Mosaic Virus (MYMV) resistance in mungbean [*Vigna radiata* (L.) Wilczek]. *Legume Research* 36:131-137.

207. Singh JP and Varma JP. 1977. Effect of some insecticides and oils on incidence of yellow mosaic and grain yield of mungbean cv. Varsha. *Proceedings of the National Academy of Sciences, India* 47: 219-225.

208. Singh K, Singh S and Gumber RK. 1996. Resistance to mungbean yellow mosaic virus in mung bean. *Indian Journal of Pulses Research* 9: 90.

209. Singh RA and Naimuddin. 2009. 25 years of Pulses Research at IIPR: 1984-2009. Indian Institute of Pulses Research, Kanpur, Publication No. 2/2009; pp. 110-113.

210. Singh RA, Sahambhi HS and Gurha SN.1982. Investigations on yellow mosaic of urdbean (*Vigna mungo*) with particular reference to yield losses and sources of resistance. *Indian Journal of Mycology and Plant Pathology* 12:115.

211. Singh RN. 1979. Natural infection of bean (*Phaseolus vulgaris*) by mungbean yellow mosaic virus. *Indian Journal of Mycology and Plant Pathology* 9:124–126.

212. Singh S and Awasthi LP. 2004. Varietal screening of urd bean against mungbean yellow mosaic virus under field conditions. *Annals of Plant Protection Sciences* 12: 225-226.

213. Singh S and Awasthi LP. 2009. Screening of mungbean [*Vigna radiata* (L.) Wilczek] germplasm against natural infection of plant viruses. *International Journal of Plant Protection* 2: 93-94.

214. Singh S, Awasthi LP and Verma HN. 2004. Prevention and control of yellow mosaic disease of mungbean by application of aqueous root extract of *Boerhaavia diffusa*. *Indian Phytopathology* 57: 303-307.

215. Singh S, Bisht IS and Dabas BS. 1987. Screening of urd bean (*Vigna mungo*) germplasm for field resistance to yellow mosaic virus. *Indian Journal of Mycology and Plant Pathology* 17: 216-217.

216. Singh SK, Chakraborty S, Singh AK and Pandey PK. 2006. Cloning, restriction mapping and phylogenetic relationship of genomic components of MYMIV from *Lablab purpureus*. *Bioresource Technology* 97: 1807-1814

217. Singh VB, Haq QMR and Malathi VG. 2013b. Antisense RNA approach targeting Rep gene of Mungbean yellow mosaic India virus to develop resistance in soybean. *Archives of Phytopathology and Plant Protection* 46:2191-2207

218. Singh, RA and Gurha SN. 1994. Influence of cropping seasons on the incidence of yellow mosaic disease in mungbean genotypes. *Indian Journal of Pulses Research* 7: 206-208.

219. Sirohi A, Singh A and Bishnoi SS. 2002. Inheritance of mungbean yellow mosaic virus resistance in blackgram (*Vigna mungo*). *Crop Improvement* 29: 173-176.

220. Srivastava SK. 1989. Further studies on viruses of cowpea (*Vigna unguiculta* ssp. *unguiculta* (L.) Walp.) Ph.D. Thesis, IARI, New Delhi. 199p.

221. Stanley J. 1983. Infectivity of the cloned geminivirus genome requires sequences from both DNAs. *Nature* 305: 643-645.

222. Sudha M, Karthikeyan A, Anusuya P, Ganesh NM, Pandiyan M, Senthil N, Raveendran M, Nagarajan P and Angappan K. 2013a. Inheritance of resistance to *Mungbean yellow mosaic virus* (MYMV) in inters and intra specific crosses of mungbean (*Vigna radiata*). *American Journal of Plant Sciences* 4: 1924-1927.

223. Sudha M, Karthikeyan A, Nagarajan P, Raveendran M, Senthil N, Pandiyan M, Angappan K, Ramalingam J, Bharathi M, Rabindran R, Veluthambi K and Balasubramanian P. 2013b. Screening of mungbean (*Vigna radiata)* germplasm for resistance to Mungbean yellow mosaic virus using agroinoculation. *Canadian Journal of Plant Pathology* 35: 424-430.

224. Sudha M, Karthikeyan A, Shobhana VG, Nagarajan P, Raveendran M, Senthil N, Pandiyan M, Angappan K, Balasubramanian P, Rabindran R and Bharathi M. 2015. Search for Vigna species conferring resistance to Mungbean yellow mosaic virus in mungbean. *Plant Genetic Resources: Characterization and Utilization* 13:162-167

225. Suman S, Sharma VK, Harsh Kumar and Shahi VK. 2015. Screening of mungbean [*Vigna radiata* (L.) Wilczek] genotypes for resistance to mungbean yellow mosaic virus (MYMV). *Environment and Ecology* 33:855-859.

226. Sunitha S, Marian D, Hohn B and Veluthambi K. 2011. Antibegomoviral activity of the agrobacterial virulence protein VirE2. *Virus Genes* 43: 445-453.

227. Sunitha S, Shanmugapriya G, V and Veluthambi K. 2013. *Mungbean yellow mosaic virus* (MYMV) *AC4* suppresses post-transcriptional gene silencing and an *AC4* hairpin RNA gene reduces MYMV DNA accumulation in transgenic tobacco. *Virus Genes* 46:496-504.

228. Sunitha S, Mahajan N and Veluthambi K. 2012. The TrAP/REn monodirectional promoter of Mungbean yellow mosaic geminivirus (MYMV) displays root-specific expression in transgenic tobacco. *Plant Cell, Tissue and Organ Culture*, 109:535-545.

229. Sunter *G,* Gardiner WE, Rushing AE, Rogers SG and Bisaro DM. 1987. Independent encapsidation of tomato golden mosaic virus, a component DNA in transgenic plants. *Plant Molecular Biology* 8: 477-484.

230. Suyal G, Mukherjee SK and Choudhury NR. 2013a. The host factor RAD51 is involved in mungbean yellow mosaic India virus (MYMIV) DNA replication. *Archives of Virology* 158:1931-1941.

231. Suyal G, Mukherjee SK, Srivastava PS and Choudhury NR. 2013b. Arabidopsis thaliana MCM2 plays role(s) in mungbean yellow mosaic India virus (MYMIV) DNA replication. *Archives of Virology* 158:981-992.

232. Suyal G, Rana VS, Mukherjee SK, Saima Wajid and Choudhury NR. 2014. Arabidopsis thaliana NAC083 protein interacts with Mungbean yellow mosaic India virus (MYMIV) Rep protein. *Virus Genes* 48:486-493.

233. Thakur MP and Agrawal KC. 1991. Appraisal of losses due to yellow mosaic of mungbean from Chhattisgarh region. International Conference on Virology in the Tropics, NBRI, Lucknow, India, 2-6 December, 1991, p. 31.

234. Thakur MP and Agrawal KC. 1998. Management of yellow mosaic disease of urdbean in Chhattisgarh region. *Indian Journal of Virology* 14: 85-87.

235. Townsend R, Watts J and Stanley J. 1986. Synthesis of viral DNA forms in *Nicotiana plumbaginafolia* protoplasts inoculated with cassava latent virus (CLV), evidence for independent replication of one component of the CLV genome. *Nucleic Acids Research* 14: 1253-1265.

236. Vanderschuren H, Stupak M, Fütterer J, Gruissem W and Zhang P. 2007. Engineering resistance to Geminiviruses-review and Perspectives. *Plant Biotechnology Journal* 5:207-220

237. Varma A and Reddy DRR. 1984. Golden and green mosaic-two new virus diseases of cowpea in northern India. *Indian Phytopathology* 37:409

238. Usharani KS, Surendranath B and Haq QMR. 2004. Yellow mosaic virus in north India is distinct from the species infecting southern and western India. *Current Science* 86:845-850.

239. Varma A and Malathi B. 2003. Emerging Geminivirus problems: A serious threat to crop production. *Annals of Applied Biology* 142:145–164.

240. Varma A, Dhar AK and Malathi VG. 1991. Cloning and restriction analysis of mungbean yellow mosaic virus, p 114. In: *International Conference on Virology in the Tropics.* Lucknow, India.

241. Varma A, Dhar AK and Mandal B. 1992. MYMV transmission and control in India. In: *Mungbean Yellow Mosaic Disease* (S.K. Green, D. Kim, ed.), Asian Vegetable Research and Development Centre, Taipei, Taiwan, 8–27.

242. Vasudeva RS. 1942. A mosaic disease of cowpea in India. *Indian Journal of Science* 12:281-283.

243. Verma RPS and Singh DP. 1988. Inheritance of resistance to mungbean yellow mosaic virus in greengram. *Annals of Agricultural Research* 9: 98-100.

244. Verma RPS and Singh DP. 1989. Inheritance of resistance to mungbean yellow mosaic virus in blackgram. *Indian Journal of Genetics & Plant Breeding* 49: 321-324.

245. Vinoth R and Jayamani P. 2014. Genetic inheritance of resistance to Yellow Mosaic disease in inter sub-specific cross of blackgram (*Vigna mungo* (L.) Hepper). *Journal of Food Legumes* 27: 9-12.

246. Vohra K and Beniwal SPS. 1979. Effect of mungbean yellow mosaic virus on yield and seed quality of urdbean (*Vigna mungo*). *Seed Research* 7:168-174.

247. Williams FJ, Grewal JS and Amin KS. 1968. Serious and new diseases of pulse crops in India in 1966. *Plant Disease Reporter 52: 300-304.*

248. Yadav MS and Brar KS. 2010. Assessment of yield losses due to mungbean yellow mosaic India virus and evaluation of mungbean genotypes for resistance in South-West Punjab. *Indian Phytopathology* 63: 318-320.

249. Yein BR and Singh H. 1982. Effect of pesticides and fertilizers on the population of whitefly and incidence of yellow-mosaic virus in greengram. *Indian Journal of Agricultural Sciences* 52: 852-855.

250. Zaim M, Kumar Y, Hallan V and Zaidi AA. 2011. Velvet bean severe mosaic virus: a distinct begomovirus species causing severe mosaic in *Mucuna pruriens* (L.) DC. *Virus Genes* 43: 138-146.

251. Zhang J and Wu ZJ. 2013. First Report of *Kudzu mosaic virus* on *Pueraria montana* (Kudzu) in China. *Plant Disease* 97:148

252. Zhimo VY, Panja BN, Saha J and Nath R. 2013. Evaluation of mungbean genotypes for resistance against Cercospora leaf spot and Yellow Mosaic diseases under field condition. *Journal of Mycopathological Research* 51: 273-278.

maheshwari & sons
maheshwaripress@gmail.com
Lucknow (0522) 4071816