JOURNAL OF AGRICULTURE AND BASIC SCIENCES
ISSN Online: 2518-4210
http://www.jabsjournal.com/

Full Length Review Article

Mungbean Yellow Mosaic Disease and its Management

Waseem Akbar[*1], Muhammad Arslan Akhtar[2], Ghulam Murtaza[1], Aamir Hussain[1], Hafiz Muthar Javed[1], Muhammad Arshad[1] and Muhammad Amir Maqbool[2]
[1]Maize & Millets Research Institute, Yusafwala Sahiwal,
[2]Department of Plant Breeding & Genetics, University of Agriculture, Faisalabad
Corresponding author: wahussaini@yahoo.com

Abstract

Mungbean, an important pulse crop, is widely grown in various regions of the world with Indian sub-continent as center of origin. The crop is vulnerable to different biotic and abiotic stresses but Mungbean Yellow Mosaic Disease, transmitted by whitefly, is most damaging in all of its growing areas. Mungbean yellow mosaic disease severely affects the plants causing up to 100% yield losses. Disease management strategies are being applied at various levels ranging from seed treatment to various breeding strategies and genetic engineering techniques. Enormous efforts have also been devoted to find out resistance sources as well to determine genetics of disease resistance. This review summarizes the available information on all aspects of this disease.

Keywords: Mungbean, MYMV, MYMD, virus, disease management

1. Introduction

Mungbean (*Vigna radiata* v radiata), known as green gram, golden gram, chickasaw pea, oregon pea, moong, mung and mungo, is an important pulse crop. It belongs to family Febaceae and genus Vigna. It is an annual, erect to sub-erect, deep rooted, 25 to 100 cm tall herbaceous plant. Stem is hollow, branched, furrowed, squarish, covered with small hairs and sometime pigmented purple. Compound leaves arise on the stem alternately and are tri-foliate or pent-foliate. Leaflets are round at base and pointed at apex, broadly ovate, medium to dark green, containing hairs and sometimes lobes. These are palmately three-veined, cuneate at the base and acuminate at the distal end. Small and greenish to bright yellow flowers are born on indeterminate inflorescence known as raceme. Flower of mungbean is a typical papilionaceous, that is, contains one wing, two standards and two keels. It contains 10 stamens arranged diadelphosly and one gynoecium. Gynoecium contains monocarpic, superior ovary with marginal placentation, twisted style and hairy stigma. Usually 10 to 15 seeds are formed in long hairy pods known as legumes. Seed color varies, may be green or olive green, yellow, brown or blackish.

Indian subcontinent is center of origin of mungbean and also considered as a region of first domestication (Vavilov, 1926; Zukovskij, 1962) due to presence of a diverse collection of cultivated and wild types (Baudoin and Maréchal, 1985). Its progenitors are abundantly found in the forms of weeds growing in other crops in India (Chandel *et al*., 1984; Lawn and Cottell, 1988), and Australia (Lawn and Cottell, 1988). Later on it spread from Asia to Middle East, East Africa, Australia and finally to America. It is widely grown in tropical and sub-tropical regions of world. Almost 90% of mungbean is contributed from Asia. Major contributors in mungbean production are India, Pakistan, Thailand, Bangladesh and Philippine (Bakar, 1981; Malik, 1991; Malik and Bashir, 1992).

Mungbean is vulnerable to different biotic and abiotic stresses. Among biotic stresses, mungbean yellow mosaic disease (MYMD) is the most damaging one and is prevalent in all growing areas (Sudha *et al*., 2013a; Mohan *et al*., 2014). This disease is transmitted by whitefly (vector) and has a wide range of hosts. MYMD is characterized by small irregular yellow specks and spots along the veins which enlarge until leaves completely become yellow. Affected plants show stunted growth and produce a fewer flowers and pods with smaller and shriveled seeds (Habib *et al*., 2007; Sudha *et al*., 2013a). Breakdown of chlorophyll contents is caused by the mungbean yellow mosaic disease which results in yellowing of leaves. In addition to yellowing of leaves, destruction of chlorophyll content is responsible for reduced photo assimilation and subsequently plant yield reduces. It can ultimately cause up to 100% yield losses in severe epidemics (Sudha *et al*., 2013a; Mohan *et al*., 2014).

2. History of disease

The disease had been a serious threat to mungbean production since a long time in all growing areas. It was first reported in India on lima bean in 1940 (Capoor and Varma, 1948), in Dolichos (Capoor and Varma, 1950) and in mungbean (Nariani, 1960). In Pakistan (before partition e) the disease was first detected in field of chickpea during 1942 near Lyallpur (Vasudeva, 1942). Different researchers have reported the disease in their own regions, e.g. Bangladesh (Jalaluddin and Shaikh, 1981), Sri Lanka and

Thailand (Thongmeearkom *et al*., 1981). Nariani was first to report that the disease is caused by virus and the virus was named as mungbean yellow mosaic virus by Nene (1968). Viral particles were observed by Thongmeearkom *et al*. (19 81) and purified latter on by Honda *et al*., (1983). First complete sequence of yellow mosaic disease virus was isolated from mungbean originating from India (Morinaga *et al.,* 1993).

3. Viral description

3.1. Family and genus

Viruses cause many plant diseases and huge yield losses in agriculture. Another peculiar feature of plant viral diseases is that these cannot be managed chemically and hence are very damaging one. All agricultural crops like cotton, tobacco, potato, papaya, rice, pulses etc. are affected by viral diseases. Plant viruses are grouped into different families. Most important viral family, responsible for plant diseases is geminiviridae. It is a group of single stranded plant viruses with a specific twinned, quasi-isometric morphology (Bock *et al.,* 1974; Mumford, 1974). Due to this morphology it was given name geminiviruses; gemeni means sign of zodiac symbolized by twins (Harrison *et al.*, 1977). The geminivirus group was named in 1979 (Matthews, 1979) and upgraded to the family *Geminiviridae* in 1995 (Murphy, 1995). Geminiviradae is further divided into four genra depending upon host, viral molecular structure, vector etc. These include mastreviruses, topocoviruses, begomoviruses and curtoviruses. Mungbean yellow mosaic virus belongs to begomoviruses. The word begomo is actually abbreviation of bean golden mosaic virus (van Regenmortel *et al*., 1997). All begomoviruses cause the disease in dicots and are transmitted by whitefly. These are subdivided into two groups; monopartite and bipartite viruses. Monopartite viruses consist of a single DNA molecule (DNA-A) while bipartite viruses consist of two DNA molecules namely DNA-A and DNA-B. MYMV is a single stranded DNA bipatrtite begomovirus (Varma and Malathi, 2003; Balaji *et al*., 2004; Girish and Usha, 2005; Malathi and John, 2008) with approximately 2.8 kb DNA-A length (Hull, 2004). Like other begomoviruses its viral particles are isometric and geminate having 18 to 30 nm size with two single stranded DNA molecules (DNA-A & DNA-B) of 2726 and 2775 nucleotides, respectively (Bos, 1999; Hull, 2004).

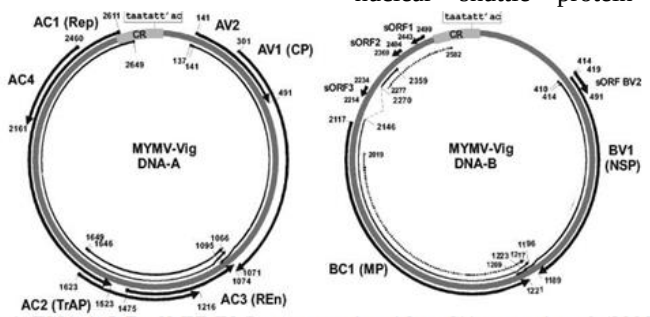

Fig. 1. DNA A & B of MYMV (Image reproduced from Shivaprasad *et al.* (2005))

3.2. Molecular description

DNA-A component encodes four genes namely AC1, AC2, AC3 and AC4 on complementary sense strand while two genes AV1 and AV2 on virion sense strand. The replication associated protein (Rep), the transcriptional activator protein (TrAP) and the replication enhancer protein (REn) are encoded by AC1, AC2 and AC3 respectively while AC4 is concerned with host determination, severity of symptoms and viral translocation (Jupin *et al.*, 1994; Laufs *et al.*, 1995; Wartig *et al*., 1997). Coat protein of the virus is encoded by AV1while AV2 is supposed to be involved in movement. On second DNA component, that is, DNA-B, two genes are present one on complementary strand (BC1) and another on virion strand (BV1). A protein known as nuclear shuttle protein (NSP), involved in transport of virus between nucleus and cytoplasm, is encoded by BV1 while BC1 encodes another most important protein known as movement protein (MP). This protein is involved in translocation of viral particles throughout the plant.

3.3. Replication

DNA viruses are further divided into two subcategories either single stranded or double stranded. These viruses replicate their genomes through one of the two mechanisms, that is, sigma replication or theta replication. MYMV is single stranded DNA begomovirus and replicate through sigma replication which is also known as rolling circle replication (RCR) (Gutierrez, 2000). The name given to this mode of replication explains its process details (rolling circle) along with molecular shape of DNA molecules (sigma) during replication. Replication takes place inside the nucleus of host where single stranded DNA is first converted to double stranded DNA. Replication starts at intergenic region (IR) of DNA molecules which does not encode any protein. This region plays role in transcription of viral genes. To start replication, Rep protein binds at the replication origin (*ori*) which is TAATATTAC (Stanely and Latham, 1992) in all geminiviruses. Initially there is a nick in this conserved site after TT as shown by slash (AATATT/AC) producing a protruding 5′ end. Due to this protruding end from circular molecule, DNA molecule looks like sigma (σ). 3′ end serves

as primer and is extended by polymerase which is moving over the circular single strand so named as rolling circle replication (RCR). As the full length of a single virus is replicated, it is cut from the extending molecule and is converted to circular form and is encapsulated to form virion.

4. Translocation in plant

Plant viruses have to move systematically from point of infection to other plant parts to spread in whole plant body. Viral movement within plant body can be divided into two type; short distance movement (cell to cell) and long distance movement. Short distance movement includes the viral movement from path of epidermal cell, mesophyll, bundle sheath cells, companion cells and finally phloem. This cell to cell route of viral movement is symplastic in nature that is, connected through plasmodesmata (Ding, 1998; Oparka, 2004). Plasmodesmata contain endoplasmic reticulum cylinders known as desmotubules. These channels have very small size as compared to viral particles and therefore pose a barrier to viral passage. Viruses have a protein known as movement protein which interact with pectin methyl transferase (protein found at plasmodesmata) to widen the plasmodesmata. In this way the viral particles move across the cells (Kumar *et al*., 2015).

Existence of actin and myosin at plasmodesmata has been reported through immunolabelling (Overall and Blackman, 1996; Radford and White, 1998). Viral movement protein recognizes the actin cytoskeleton resulting in structural changes of plasmodesmata and increase in size exclusion limit (SEL). Callose is another SEL regulator and its deposition at plasmodesmata affects its permeability. It has been reported that increase in callose deposition is required to decrease the SEL which restricts the viral movement (Radford *et al*., 1998; Sivaguru *et al*., 2000; Radford and White, 2001). This idea is further supported by fact that with the expression of β-1, 3-glucanase, which is hydrolyzing agent, the viral spread increases (Bucher *et al*., 2001).

5. Transmission of disease

Transmission of viruses from one plant to other plants is very crucial for their survival and is pivotal in disease spread. Viruses are either transmitted by vectors or by mechanical means. Plant viruses are mostly transmitted by different insects or nematodes which feed on host plants. Viruses can be divided into several types based on the mode of transmission. Some viruses can be transmitted by vector just after they feed on infected plants, that is, viruses attached to their mouthparts are ready for transmission. This type of viral transmission is called non-persistent. Some viruses are not transmitted immediately after feeding but require long time for transmission. These viruses reach to the gut of insects and then ultimately to the saliva. Such viruses can be transmitted taking long time after feeding so this transmission is called persistent transmission. If the persistently transmitted viruses only circulate in vector body they are called circulative while if they also replicate inside vector body they are called propagative.

MYMV is mainly transmitted by whitefly in circulative persistent manners (Ahmad and Harwood, 1973; Honda, 1983; Shad *et al*., 2005; Malathi and John, 2008; Paul *et al*., 2013; Mohan *et al*., 2014) and by grafting (Akhtar and Haq, 2003) but not vertically (seed transmission), sap and soil (Shad *et al*., 2005). Only Thailand strain of MYMV is reported to be transmitted mechanically (Honda *et al*., 1983). The disease is transmitted by whitefly (Bemisia tabaci) that delivers the virus through proboscis to phloem cells of the host plant. Acquisition feeding period requires 24 hours for transmission of the virus in mungbean (Nariani, I960). However, the minimum period of acquisition and inoculation, from and to mungbean is 15 minutes (Rathi and Nene, 1972). Starvation of the vector for four hours prior to acquisition and inoculation feeding increased the efficiency of transmission by 12 and three fold respectively over the non-starved flies (Nair and Nene, 1973). The minimum incubation period for virus in vector is four hours but 24 hours incubation period gave the maximum transmission (Nene, 1968). A single whitefly can transmit the virus, but for 100% transmission, 10 viruliferous flies per plant are required (Nair and Nene, 1973). Both male and female flies can transmit the virus but female adults are four times more efficient than males (Rathi, 1979); neither sex remains infective throughout its life (Costa, 1976). Pre-adult stages could also acquire and transmit the virus.

Vector, virus and host range are dependent on each other. Host range of virus is determined not only by the plant viral interaction but also by the vector of viruses. Virus can affect only that plant which supports its replication but there should be a vector which can transmit the virus to host plant. Absence of any of these factors escapes the plant from host range of virus. Virus-vector relation is governed by different proteins e.g. coat protein, helper component protein and cuticle lining of vector mouthparts. It has been reviewed that when specific domain of coat protein found in begomovirus was replaced with domain of curtovirus coat protein, the vector changed from whitefly to leafhopper (Briddon *et al*., 1990).

6. Alternate hosts

A single virus can affect a wide range of plants which are known as host plants / alternate

hosts of the virus. Alternate hosts are very important for virus survival over a long period of the time by assuring their presence within specific place round the year. Mostly the alternate hosts are close relative of each other because their similarity allows the same viruses and vectors to replicate and feed on them. For example begomoviruses are transmitted by whitefly and mostly affect dicots. MYMV infects different legumes including soybean, mothbean, frenchbean, cowpea and urdbean (mash) (Dhingra and Chenululu, 1985; Qazi *et al.*, 2007).

7. Resistance mechanisms of plants

Viruses differ from other pathogens in mode of disease development. Other pathogens depend upon the plants mainly for food while viruses have to just multiply themselves inside the host. So they require a living host instead of dead plant, hence they are called biotrophic pathogens. In some cases they don't develop any disease but in other cases they cause disease. Plants have evolved different mechanisms to combat viruses and neutralize their detrimental effects. Important defense mechanisms of plants against viruses include hypersensitivity response and gene silencing.

7.1 Hypersensitive and necrotic response (HR)

This mechanism is used by plants against all types of pathogens like bacteria, fungi and viruses. R genes are of prime importance for this response. These genes activate HR response and leads to changes in plant defense hormones such as salicylic acid and jasmonic acid. It also initiates the accumulation of reactive oxygen species such as superoxide and hydrogen peroxide (Culver and Padmanabhan, 2007; Carr *et al*., 2010; Pallas and García, 2011; Mandadi and Scholthof, 2012). It has been reported that HR affects calcium ion homeostasis and membrane permeability. HR activates some proteases which are involved in cell death (Mur *et al*., 2008). In this way cells in periphery of infection become dead to limit the viral spread to other plant parts in resistant genotypes.

7.2 RNA silencing

It is common observation that new leaves of infected plants are mostly disease free. Basically same or related viruses can not affect the newer plant leaves. This is due to a process known as "recovery". Recovery process is actually due to another plant defense system known as RNA silencing. RNA silencing is well known in all organisms; animals, plants, fungi and insects. RNA silencing is viewed as a primary antiviral defense system of plants (Chellappan *et al*., 2005). In this mechanism small RNAs degrade larger RNAs in sequence specific manner (Baulcombe, 2004; Carr *et al*., 2010). There are two types of small RNAs; micro RNAs and short interfering RNAs depending upon their source (Brodersen and Voinnet, 2006; Xie *et al*., 2004). Micro RNAs are synthesized by transcription of the non-protein coding DNA followed by some procedure. siRNAs are produced by cleavage of dsRNA (Xie *et al*. 2004; Brodersen and Voinnet, 2006). RNA silencing has two modes of actions depending upon the target. In first case, post transcriptional gene silencing, the messenger RNA of virus is targeted and degraded resulting in immunity against that virus. In another case cytosine residue of viral DNA or histone protein is methylated (Mathieu and Bender, 2004; Vanitharani *et al*., 2005; Akbergenov *et al*., 2006; Dogar, 2006; Daxinger *et al*., 2009) which makes the DNA compact and transcription is blocked. This is called transcriptional gene silencing. Although symptom recovery is not found in case of MYMD (Boulton, 2003; Varma and Malathi, 2003) but it has been reported that resistant genotypes degrade the RNAs of MYMIV more rapidly as compared to susceptible genotypes (Yadav *et al*., 2009). Transcriptional gene silencing in region of replication start site of MYMV has been reported in resistant genotypes (Yadav and Chattopadhyay, 2011).

8. Physiological changes associated with MYMD

Plant is a living entity so responds to any internal or external change accordingly. On exposure to any pathogen, plants try to adjust their physiology, morphology and anatomy to minimize the deleterious effects of pathogen and to maintain their own growth and development. Gene expression of a large number of genes is altered in response to pathogens. The response is different in resistant (incompatible reaction) and susceptible (compatible reaction) plants.

The first response of plant to any pathogen is production of reactive oxygen species (ROS). The production of ROS during plant pathogen interaction is harmful for the pathogen; ROS transmits signal to induce defense pathways in the host (Gara *et al*., 2003). During disease, ROS and total phenols increase in both resistant and susceptible plants but rate of change is much greater in resistant plants. To save the plants from deleterious effects of ROS, there is another plant system known as anti-oxidant system. Anti-oxidants help in keeping the ROS activity within limits. Superoxide dismutase (SOD) sharply increases in resistant plants as compared to susceptible plants and coverts superoxide to H_2O_2 (Kundu *et al*., 2013). This increased ROS production causes the rupturing of plastids making the chlorophyll free inside cell. This exposed

chlorophyll is destroyed by the enzyme named as chlorophyllase (Goodman *et al*., 1986). In susceptible genotypes there is significant reduction in Chlorophyll *a*, Chlorophyll *b* and carotenoid contents while no significant change has been observed in resistant genotypes. More the reduction of chlorophyll less is green color; increased yellowing of leaves is primary symptom of disease. This reduction in chlorophyll content results in reduction of photosynthesis in mungbean. Soluble and total carbohydrate contents increased significantly in resistant plants while slight change was found in susceptible plants. Insoluble carbohydrate showed slight increase in resistant genotypes but significantly decreased in susceptible genotypes (Kundu *et al*., 2013).

9. Genetics of disease resistance

Genetic resistance against diseases, insects or stresses is most economical and viable solution for crop improvements. Therefore a massive work has been done in searching the MYMD resistance. Different levels of disease resistance are found in mungbean germplasm. Disease resistance may be dominant or recessive over susceptibility. Mostly dominant resistance is found against bacterial and fungal pathogens (Soosaar *et al*., 2005) while it is reported that resistance against plant viruses is mostly controlled by recessive genes (Truniger and Aranda, 2009). This condition has its further important implications. In case of dominant resistance, the resistant genotype is of great importance because it has an important mechanism which does not support viral replication or its spread within plant body. While in case of recessive resistance, recessive plant does not have any resistance mechanism or process but it is deficient of any plant factor which is crucial for viral replication or spread (Diaz-Pendon *et al*., 2004). Therefore when such plant is crossed with susceptible plant, resulting F_1 is susceptible due to presence of that factor contributed by susceptible plant. It shows that the nature of disease resistance is very important for determination of mechanism underlying the resistance and resistance development. Secondly the numbers of genes which confer resistance are also very important in breeding resistance crops. Numbers of genes provide the relative ease or difficulty with which the trait can be transferred in any genetic background.

There is conflict among the scientists regarding the numbers of genes conferring resistance against this disease. Resistance to MYMD has been reported to be controlled by single recessive gene (Thakur *et al*.1977; Malik *et al*., 1988; Malik and Bashir, 1992; Reddy and Singh 1995; Saleem *et al*., 1998; Basak *et al*., 2004; Khan *et al.,* 2007; Sudha *et al.,* 2013b). These findings are based on different generations like F_2, BC_1 and BC_2 analyzed by chi square test. According to some other scientists, resistance is controlled by two recessive genes (Shukla and Pandya, 1985; Ammavasai *et al*., 2004). Chandra *et al*. (2010) has reported that the resistance to MYMD is controlled by three recessive genes. Sandhu *et al.* (1985) has claimed that resistance is controlled by single dominant gene. So it shows that there is no single accepted idea on numbers of genes and nature of resistance (recessive/ dominant). It has also been reported that there is no involvement of extra-nuclear genes in the resistance. It means that there is no difference in reciprocal crosses of resistant and susceptible parents (Shukla *et al*., 1978; Khattak *et al*., 2000; Khan *et al.,* 2007). Recently Akbar et al. (2018) has shown that the resistance is controlled by two genes showing additive gene action. Interestingly Akbar *et al*. (2018) has also reported monogenic resistance for the disease from the same data and concluded that conflict regarding number of resistant genes springs from disease severity scale and number of classes used by the researchers. This finding has given a new direction to the researchers to manage this conflict regarding nature of disease resistance.

Most accepted idea about MYMD resistance is that it is qualitative /oligogenic in nature but some scientists claim that it is rather quantitative. The idea of quantitative nature is supported by the fact that there are no clear cut classes of disease reaction. Plants show a continuous change in disease severity which implies that resistance is not discrete. One explanation to these continuous varying disease symptoms is that in addition to the major genes there are some modifying genes. These modifying genes alter the degree of expression of major genes and produce a continuous resistance (Khattak *et al.,* 2000). The idea of quantitative nature has also been supported by Akbar *et al*. (2018) who reported additive gene action for resistance.

10. Crop management / solutions

10.1 Insect control

MYMD is transmitted only through insect vector (whitefly) instead of mechanical transmission. So, one of the ways to stop the spread of disease is to control the vector. Whitefly is a polyphagous insect and it is found round the year in field providing the inoculation source. It can be controlled by management practices (botanicals and chemicals). Primary and effective way for controlling whitefly is chemical control. Acetamiprid, thion, imidachlorpid, triazophos are popular systemic chemical insecticides against whitefly. These provide control in both ways; they

kill the insect not only on contact, but are also absorbed by the plant. In latter way these provides protection against the insect for more than few weeks (Wang *et al*., 2009). Seed treatment with insecticides is also potential way to control the vector. Time of application, stages of vector to be controlled and the way of insecticide application are worth important. Younger stages of insects can be easily controlled as compared to adults. Similarly, crop season plays a vital role in determining the insect population. Generally whitefly population is very high during hot and dry season while less in humid and cool season. So the planting time of crop can be managed for controlling the insect vector. In this context mungbean planting during spring season is preferred with respect to MYMD.

Different botanicals are also used to control whitefly population in crops. Neem (*Azadirachta indica*) plant is famous for its biological products against pests. Neem seed extract and neem oil are effectively used against whitefly. It is especially effective at nymph stages of the insect. In this way the transformation of nymph to adult whitefly is hampered by this product, resulting in reduction of whitefly population (Dubey *et al*., 2011).

10.2. Breeding for MYMD resistance

Genetic resistance is most important and viable control against any disease. It is completely based on plant genetic architecture and hence no insecticide or any other strategy is required to be used. It is also durable, economic and environment friendly approach. It only requires the source of resistance, from where we can transfer the resistance to any genotype. Secondly the inheritance pattern of resistance is also critical to develop the strategy to transfer the trait of interest.

Identification of true resistant source is prime objective in breeding against any disease. In this regard first and most crucial step is to measure the extent or severity of disease (Horsfall and Cowling, 1978; Akhtar and Khan, 2002). A massive work has been conducted to search out the resistance source in Pakistan, India, Bangladesh, Thailand and other affected countries. Grouping of the genotypes as resistant and susceptible is done following different disease scoring scales. These scoring scales are either based on disease incidence percentage (percentage of diseased plants) or on disease severity. The scoring scale based on infection percentage has been used previously by almost all scientists (Ahmad, 1975; Bashir *et al*., 2005; Bashir *et al*., 2006; Khattak *et al*., 2008) but scale based on disease severity is now becoming popular among scientists. The scoring method based on disease severity was given by Akhtar *et al*. (2009) and is quantitative in nature. Disease severity based scale is better because it considers the severity of attack and differentiates the responses from slightly affected to severely affected plants. While in case of disease incidence percentage scales, there is no difference between slightly and severely infected plants. This scale just bothers about the percentage of infected plants.

Screening of germplasm following any disease scoring scale yields the information about resistance sources and their resistance potentials. So screening of germplasm or identification of resistance source is first step in breeding. In this context a massive work has been conducted to search out the resistance sources in mungbean by different scientist (Ahmed, 1975; Sahoo and Hota, 1991; Singh *et al*., 1996; Shad *et al*., 2006; Akbar *et al.,* 2017) with different levels of success. Resistance to MYMV has also been recognized in wild species (V. *umbellata* and V. *sublobata*) of mungbean and may consent the introduction of such resistance by means of interspecific hybridization (Monika *et al*., 2001; Bisht *et al*., 2005; Pandiyan *et al*., 2008; Sudha *et al*., 2013). Through the practices of intra and inter-specific hybridization, several promising lines which are not only tolerant / resistant to MYMV but are also high yielding have been developed and released for commercial cultivation (Reddy and Singh, 1995; Saleem *et al*., 1998). As mentioned earlier, inheritance pattern of MYMD resistance is controversial among the scientists with respect to number of genes governing the resistance. After source identification, the next step in breeding is to determine the genetic nature of the trait/resistance. Information regarding inheritance of trait is crucial in decision of breeding method to be used. Different researchers have struggled to determine the inheritance pattern of MYMV resistance (as discussed above).

10.3. Engineering the resistance

In most cases natural sources of resistance to viral diseases are scarce therefore engineering the resistance artificially is an effective alternate. There are different ways to introduce resistance through genetic engineering but all the procedures follow the same basic principle. Sanford and Johnston (1985) were first to propose mechanism of artificial disease resistance through use of proteins of pathogens and resulting resistance was named pathogen derived resistance (PDR). In this approach genes of pathogens encoding important proteins are transferred to plants and plants expressing these genes become resistance to the disease. Viral proteins including coat protein, movement protein, rep protein and replicase proteins have been successfully used in engineering resistance to different viruses (Abel *et al*., 1986; von Arnim and Stanley, 1992; Vanitharani *et al*., 2004).

In case of MYMV, Rep and AC4 genes have been successfully used to engineer resistance

in soybean, blackgram (mash) and tobacco. Rep protein occupies the pivotal position in viral replication inside the nucleus of plant. Rep, a multifunctional protein, is involved in transcription regulation and viral replication. It is also involved in activation and recruitment of proteins encoded by hosts for its DNA synthesis (Hanley-Bowdoin *et al*., 1999). The central portion of Rep has a role in oligomerization (Orozco *et al*., 1997). Shivaprasad *et al*. (2006) has evaluated the use of different MYMV genes for developing resistance in non-host plant i.e. tobacco. Coat protein, nuclear shuttle protein (NSP), movement protein and replication associated protein (Rep) were transferred to different tobacco plants. After transformation with viral genes, plants were agro-inoculated with partial dimmers of MYMV. No resistance was observed in case of transformation with CP, MP, NSP and Rep antisense. Resistance was only observed in plants transformed with Rep sense gene and T-Rep gene. Haq *et al*. (2010) has also engineered the resistance against MYMV using the sense Rep gene. Another protein coding gene of MYMV, used in engineering resistance is AC4. It is found in DNA-A genome nested inside Rep gene. The C4 gene of monopartite viruses manifests several functions. The C4 genes of beet curly top virus (Stanley and Latham, 1992), tomato leaf curl virus (Selth *et al.*, 2004) and tomato leaf curl virus-Australia (Saeed *et al.*, 2008) act as major symptom determinants. Sunitha *et al*., 2013 has exploited this gene for developing resistance against MYMD. It was transferred to plant in two form; sense and hairpin structure. Resistance was observed only in hairpin structure while no resistance was seen with AC4 sense configuration.

References

Abel, P. P., R. S. Nelson, B. De, N. Hoffmann, S. G. Rogers, R. T. Frale y and R. N. Beachy. 1986. Delay of disease development in transgenic plants that express the tobacco mosaic virus coat protein gene. Sci. 232: 738-743.

Ahmad, M. 1975. Screening of mungbean (*Vigna radiata*) and urdbean (*Vigna mungo*) germplasm for resistance to mungbean yellow mosaic. J. Agric. Res. 13: 349-354.

Ahmad, M. and R. F. Harwood. 1973. Studies on whitefly transmitted yellow mosaic disease of cowpea (*Vigna unguiculata*). Plant Dis. Rep. 62: 224-226.

Akbergenov, R., A. Si-Ammour, T. Blevins, I. Amin, C. Kutter, H. Vanderschuren, P. Zhang, W. Gruissem, F. J. Meins, T. Hohn and M. M. Pooggin. 2006. Molecular characterization of Gemini virus derived small RNAs in different plant species. Nucleic Acids Res. 34: 462- 471.

Akhtar, K. P. and M. S. I. Khan. 2002. Modified scale for the assessment of cotton leaf curl virus (CLCuV). Pak. J. Phytopathol. 14: 88-90.

Akhtar, K. P., R. Kitsanachandee, P. Srinives, G. Abbas, M. J. Asghar, T. M. Shah, B. M. Atta, O. Chatchawankanphanich, G. Sarwar, M. Ahmad and N. Sarwar. 2009. Field evaluation of mungbean recombinant inbred lines against mungbean yellow mosaic disease using new disease scale in Thailand. Plant Pathol. J. 422-428.

Ammavasai S., D. S. Phogat and I. S. Solanki. 2004. Inheritance of resistance to Mungbean Yellow Mosaic Virus (MYMV) in greengram (*Vigna radiata* L. Wilczek). Indi. J. Genet. 64: pp. 146.

Bakar, A. K. 1981. Pest and disease problems of mungbean in West Malaysia. Malaysian Agr. J. 53: 29-33.

Balaji, V., R. Vanitharani, A. S. Karthikeyan, S. Anbalagan and K. Veluthambi. 2004. Infectivity analysis of two variable DNA B components of Mungbean yellow mosaic virus-Vigna in *Vigna mungo* and *Vigna radiata*. J. Biosci. 29: 297-308.

Basak, J., S. Kundagrami, T. A. Ghose and A. Pal. 2004. Development of Yellow Mosaic Virus (YMV) resistance linked DNA marker in *Vigna mungo* from population segregating for YMV reaction. Mol. Breed. 14: 375-383.

Bashir, M., Z. Ahmad and Z. A. Ghafoor. 2005. Sources of genetic resistance in mungbean and black gram against urdbean leaf crinkle virus (ULCV) Pak. J. Bot. 37: 47-51.

Bashir, M., Z. Ahmad and S. Mansoor. 2006. Occurrence and distribution of viral disease of mungbean and mashbean in Punjab, Pakistan. Pak. J. Bot. 38: 1341-1351.

Baudoin, J. P. and R. Maréchal. 1985. Genetic diversity in Vigna. P. 3-9. *In*: Singh, S.R. and Rachie, K.O. (eds) Cowpea Research, Production and Utilization. John Wiley and Sons, Chichester, UK.

Baulcombe, D. 2004. RNA silencing in plants. Nature. 431: 356-363.

Bisht, I., K. Bhat, S. Lakhanpaul, M. Latha, P. Jayan, B. Biswas and A. Singh. 2005. Diversity and genetic resources of wild Vigna species in India. Genet. Resour. Crop Evo. 52: 53-68.

Briddon, R. W., M. S. Pinner, J. Stanley and P. G. Markham. 1990. Geminivirus coat protein replacement alters insect specificity. Virol. 177: 85-94.

Bock, K. R., E. J. Guthrie and R. D. Woods. 1974. Purification of maize streak virus and its relationship to viruses associated with streak diseases of sugar cane and Panicum maximum. Ann. App. Biol. 3: 289-296.

Boulton, M. I. 2003. Geminiviruses: Major threats to world agriculture. Ann. Appl. Biol. 4:142- 143.

Brodersen, P. and O. Voinnet. 2006. The diversity of RNA silencing pathways in plants. Trend. Genet. 22: 268-280.

Bucher, G. L., C. Tarina, M. Heinlein, F. D. Serio, F. J. Meins and V. A. Iglesias. 2001. Local expression of enzymatically active class I beta-1, 3-glucanase enhances symptoms of TMV infection in tobacco. Plant J. 28: 361-369.

Capoor, S. P. and P. M. Varma. 1948. Yellow mosaic of *Phaseolus lunatus*. Curr. Sci. 17: 152-153.

Capoor, S. P. and P. M. Varma. 1950. New virus disease of *Dolichos lablab*. Curr. Sci. 19: 242-249.

Carr, J. P., M. G. Lewsey and P. Palukaitis. 2010. Signaling in induced resistance. Adv. Virus Res. 76: 57-121.

Chandel, K. P. S., R. N. Lester and R. J. Starling. 1984. The wild ancestors of urd and mung beans (*Vigna mungo* (L.) Hepper and *V. radiata* (L.) Wilczek). Bot. J. Linn. Soc. 89: 85-96.

Chellappan P., R. Vanitharani, C. M. Fauquet. 2005. MicroRNA-binding viral protein interferes with Arabidopsis development. Proc. Natl. Acad. Sci.102: 10381-10386.

Costa, A. S. 1976. Whitefly transmitted diseases. Ann. Rev. Phytopath. 14: 429-449.

Culver J. N. and M. S. Padmanabhan. 2007. Virus-induced disease: Altering host physiology one interaction at a time. Annu. Rev. Phytopathol. 45: 221-243.

Daxinger, L., T. Kanno, E. Bucher, J. Widen, U. Naumann, A. J. M. Matzke and M.Matzke. 2009. A stepwise pathway for biogenesis of 24-nt secondary siRNAs and spreading of DNA methylation. Eur. Mol. Biol. Organ. J. 28: 48-57.

Dhingra, K. L. and V. V. Chenulu. 1985. Effect of yellow mosaic on yield and nodulation of soybean. Indian J. Phytopathol. 38: 248-251.

Ding, B.1998. Intercellular protein trafficking through plasmodesmata. Plant Mol. Biol. 38: 279-310.

Dogar, A. M. 2006. RNAi dependent epigenetic marks on a geminivirus promoter. Virol. J. 3:5.

Dubey, N. K., R. Shukla, A. Kumar, P. Singh and B. Prakash. 2011. Global scenario on the application of natural products in integrated pest management programmes. p. 120. *In*: Dubey N. K. (ed.) Natural products in plant pest management. Wallingford, CAB International, UK.

Gara, L. D., M. C. Pinto and F. Tommasi. 2003. The antioxidant systems vis-à-vis reactive oxygen species during plant–pathogen interaction. Plant Physiol Biochem. 41: 863-870.

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

Gutierrez, C. 2000. DNA replication and cell cycle in plants: learning from geminiviruses. Embo. J. 19: 792-799.

Habib, S., N. Shad, A. Javaid and U. Iqbal. 2007. Screening of mungbean germplasm for resistance/tolerance against yellow mosaic disease. 5: 89-94.

Hanley-Bowdoin, L., S. B. Settlage, B. M. Orozco, S. Nagar and D. Robertson. 1999. Geminiviruses: models for plant DNA replication, transcription and cell cycle regulation. Crit. Rev. Plant Sci. 18: 71-106.

Haq, Q. M. I., A. Ali and V. G. Malathi. 2010. Engineering resistance against mungbean yellow mosaic india virus using antisense RNA. Indian J. Virol. 21: 82-85.

Harrison, B. D., H. Barker, K. R. Bock, E. J. Guthrie, G. Meredith and M. Atkinson. 1977. Plant viruses with circular single-stranded DNA. Nature 270: 760-762.

Honda, Y., M. Iwaki, Y. Saito, P. Thongmeearkom, K. Kittisak and N. Deema. 1983. Mechanical transimission, purification and some properties of whitefly-borne mungbean yellow mosaic virus in Thailand. Plant Dis. 67: 801-804.

Horsfall, J. G. and E. B. Cowling. 1978. Pathometry. The measurement of plant disease. P. 119-136. *In*: J. G. Horsfall and E. B. Cowling, (eds.) Plant Disease: An Advance Treatise. Academic press, New York.

Hull, R. 2004. Mathew's Plant Virology. Elsevier Publishers, India.

Diaz-Pendon, J. A., V. Truniger, C. Nieto, J. Garcia-Mas, A. Bendahmane and M. A. Aranda. 2004. Advances in understanding recessive resistance to

plant viruses. Mol. Plant Pathol. 5: 223-233.

Jalaluddin, M. and M. A. Q. Shaikh. 1981. Evaluation of Mungbean (*Vigana radiata* L.) germplasm for resistance to mungbean yellow mosaic virus. SABRAO J. 13: 61-68.

Jupin, I., F. D. Kouchkovsky, F. Jouanneau and B. Gronenborn. 1994. Movement of tomato yellow leaf curl geminivirus (TYLCV): Involvement of the protein encoded by ORF C4. Virol. 204: 82-90.

Khan, M.G., W. Ahmad, G. S. S. Khattak, Siraj-ud-Din and H. Ahmad. 2007. Mode of inheritance of resistance to mungbean Yellow mosaic virus (mymv) in mungbean (vigna radiata (l.) Wilczek). Sarhad. J. Agric. 23: 1071-1074.

Khattak, G. S. S., M. A. Haq, S. A. Rana, G. Abass, and M. Irfag. 2000. Effect of mungbean yellow mosaic virus (MYMV) on yields and yield components of mungbean (*Vigna radiata* L. Wilczek). Kasetsart J. Nat. Sci. 34:12-16.

Khattak, G. S. S., I. Saeed and S. A. Shah. 2008. Breeding high yielding and disease resistant mungbean (*Vigna radiata* (L.) Wilczek) genotypes. Pak. J. Bot. 40:1411-1417.

Kumar, J., S. Gunapati, A. Alok, A. Lalit, R. Gadre and N. Sharm. 2015. Cotton leaf curl Burewala virus with intact or mutant transcriptional activator proteins: complexity of cotton leaf curl disease. Arch. Virol. 160: 1219-1228.

Kundu, S., D. Chakraborty, A. Kundu and A. Pal. 2013.Proteomics approach combined with biochemical attributes to elucidate compatible and incompatible plant-virus interactions between *Vigna mungo* and Mungbean Yellow Mosaic India Virus. Proteome Sci.11: 15-16.

Laufs, J., W. Traut, F. Heyraud, V. Matzeit, S. G. Rogers, J. Schell and B. Gronenborn. 1995. In vitro cleavage and joining at the viral origin of replication by the replication initiator protein of Tomato yellow leaf curl virus. Proc. Natl. Acad. Sci. 92: 3879-3883.

Lawn, R. J. and A. Cottell. 1988. Wild mungbean and its relative in Australia. Biologist. 35: 267-273.

Malathi, V. G. and P. John. 2008. Mungbean yellow mosaic viruses. p. 364-371. *In*: Mahy, B. W. J., M. H. V and Van Regenmortel. (eds.). Desk encyclopedia of plant and fungal virology in encyclopedia of virology. Amsterdam, Elsevier, Austarlia.

Malik, B. A. and M. Bashir. 1992. Major diseases of food legume crops of Islamic countries. P. 25-38. *In*: F. F. Jamil and S. H. M. Naqvi (eds.) Proc. Agro climatology pests and disease and their control Int. workshop, COMSTECH NIAB, Faisalabad.

Malik, I. A. 1991. Breeding for resistance to MYMV and its vector in Pakistan. p. 79. *In*: Green, S. K. and D. Kim (Eds.) Pro. Int. Symp. Mungbean yellow mosaic disease International Workshop. 2-3 July 1991. Bangkok, Thailand.

Malik, I. A., Y. Ali and M. Saleem. 1988. Incorporation of tolerance to Mungbean Yellow Mosaic Virus from local germplasm into exotic large seeded mungbean. p. 297 – 307. *In*: S. Shanmungasundaram (ed.). Proc. Second Int. Symp. Mungbean. 14-16 March 1998. Bangkok, Thailand.

Mandadi, K. K. and K. B. G. Scholthof. 2012. Characterization of a viral synergism in the monocot Brachypodium distachyon reveals distinctly altered host molecular processes associated with disease. Plant physiol. 160:1432-1452.

Mathieu, O. and Bender, J. 2004. RNA-directed DNA methylation. J. Cell Sci. 117: 4881-4888.

Matthews, R. E. F. 1979. Classification and nomenclature of viruses. Third Report of the International Committee on Taxonomy of Viruses. Intervirology. 12: 132- 296.

Mohan, S., A. Sheeba, E. Murugan and S. M. Ibrahim. 2014. Screening of mungbean germplasm for resistance to mungbean yellow mosaic virus under natural condition. Indian J. Sci. Tech. 7: 891-896.

Monika, K. P., T. Singh and P. K. Sareen. 2001. Cytogenetics studies in mungbean-rice bean Hybrids. J. Cytol. Genet. 2: 13-16.

Morinaga, T., M. Ikegami and K. I. Miura. 1993. The nucleotide sequence and genome structure of mungbean yellow mosaic geminivirus. Microbiol. Immun. 37: 471-476.

Mumford, D. L. 1974. Purification of curly top virus. Phytopathol. 64: 136-139.

Murphy, F. 1995. Virus taxonomy. Sixth report of the International Committee on Taxonomy of viruses. Arch. Virol. 10: 313-314.

Nair, N. G. and Y. L. Nene. 1973. Studies on the yellow mosaic ofurdbean (*Phaseolus mungo* L.) caused by mungbean yellow mosaic virus, virus-vector relationships. Indian J. Farm Sci. 1: 62-70.

Nariani, T. K. 1960. Yellow mosaic of mung (Phaseolus aureus L.). Indian Phytopathol 13: 24-29.

Nene, Y. L. 1968. A survey of viral diseases of pulse crops in Uttar Pradesh. Final Tech. Report. Res. Bull. No. 4, U.P. Agricultural University, Pantnagar, India. 1-91 pp.

Oparka, K. J. 2004. Getting the message across: how do plant cells exchange macromolecular complexes? Trend Plant Sci. 9: 33-41.

Overall, R. L. and L. M. Blackman. 1996. A model of the macromolecular structure of plasmodesmata. Trend Plant Sci. 1. 307-311.

Pallas, V. and J. A. García. 2011. How do plant viruses induce disease? Interactions and interference with host components. J. Gen. Virol. 92: 2691-2705.

Pandiyan, M., N. Ramamoorthi, S. Ganesh, S. Jebaraj, P. Pagarajan and P. Balasubramanian. 2008. Broadening the genetic base and introgression of MYMV resistance and yield improvement through unexplored genes from wild relatives in mungbean. Plant Mutat. Rep. 2: 33-38.

Qazi, J., M. Ilyas, S. Mansoor and R. W. Briddon. 2007. Legume yellow mosaic viruses: genetically isolated begomoviruses. Mole. Plant Pathol. 8: 343-348.

Radford, J. E. and R. G. White. 2001. Effects of tissue-preparation-induced callose synthesis on estimates of plasmodesmata size exclusion limits. Protoplasma. 216: 47-55.

Radford, J. E. and R. G. White. 1998. Localization of a myosin-like protein to plasmodesmata. Plant J. 14: 743-750.

Rathi, Y. P. S. and Y. L. Nene. 1972. Some aspects of relationship between mungbean yellow mosaic virus and its vector, Bemisia tabaci. Indian Phytopathol. 27: 459-462.

Reddy, K. R. and D. P. Singh. 1995. Inheritance of resistance to mungbean yellow mosaic virus. Madras Agric J. 88: 199-201.

Saeed, M., S. Mansoor, M. Rezaian, R. Briddon and J. Randles. 2008. Satellite DNA β overrides the pathogenicity phenotype of the C4 gene of tomato leaf curl virus but does not compensate for loss of function of the coat protein and V2 genes. Archi. Virol. 153: 1367-1372.

Sahoo, B. K. and A. K. Hota. 1991. Field screening of greengram germplasm against insect pest and disease complex. Madras Agri. J. 8: 84-86.

Saleem, M., A. Wasif, A. Haris and I. A. Malik. 1998. Inheritance of yellow mosaic virus in mungbean (*Vigna radiata* (L.) Wilczek). Pak .J. Phytopathol. 10 : 30-32.

Sandhu, T. S., J. S. Brar, S. S. Sandhu and M. M. Verma. 1985. Inheritance of Resistance to Mungbean Yellow Mosaic Virus in Greengram. J. Res. 22: 607-611.

Sanford, J. C. and S. A. Johnston. 1985. The concept of parasite-derived resistance: Deriving resistance genes from the parasite's own genome. J. Theor. Biol. 13: 395-405.

Selth, L. A., J. W. Randles and M. A. Rezaian. 2004. Host responses to transient expression of individual genes encoded by Tomato leaf curl virus. Mol. Plant Microbe In. 17: 27-33.

Shad, N., S. M. Mughal, K. Farooq and Bashir. 2006. Evaluation of mungbeen germplasm for resistance against mungbean yellow mosaic begomovirus. Pak. J. Bot. 38: 449-457.

Shad, N, S. M. Mughal and M. Bashir. 2005. Transmission of Mungbean Yellow Mosaic Begomovirus (MYMV). Pak. J. Phytopathol. 17: 141-143.

Shivaprasad, P. V., P. Thillaichidambaram, V. Balaji and K. Veluthambi. 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.

Shukla, G. P. and B. P. Pandya. 1985. Resistance to Yellow Mosaic in Greengram. SABRAO J. 17: 165-171.

Shukla, G. P., B. P. Pandya and D. P. Singh. 1978. Inheritance of resistance to yellow mosaic in mungbean. Ind. J. Genet. Plant Breed. 38: 357-360.

Singh, K., S. Singh and R. K. Gumber, 1996. Resistance to mungbean yellow mosaic virus in mungbean. Ind. J. Pulses Res. 9: 90-97.

Sivaguru, M., T. Fujiwara, J. Samaj, F. Baluska, Z. Yang, H. Osawa, T. Maeda, T. Mori, D. Volkmann and H. Matsumoto. 2000. Aluminum-induced 1–N3-beta-D-glucan inhibits cell-to-cell trafficking of molecules through plasmodesmata. A new mechanism of aluminum toxicity in plants. Plant Physiol. 124: 991-1006.

Soosaar, J. L., T. N. M. Burch-Smith and S. P. Dinesh-Kumar. 2005. Mechanisms of plant resistance to viruses. Nat. Rev. Microbiol. 3: 789-798.

Stanley, J. and J. R. Latham. 1992. A symptom variant of beet curly top geminivirus

produced by mutation of open reading frame C4. Virol. 190: 506-509.

Sudha, M., A. Karthikeyan, P. Anusuya, N. M. Ganesh, M. Pandiyan, N. Senthil, M. Raveendran, P. Nagarajan and K. Angappan. 2013a. Inheritance of Resistance to Mungbean Yellow Mosaic Virus (MYMV) in Inter and Intra Specific Crosses of Mungbean (*Vigna radiata*). Afr. J. Plant Sci. 4: 1924-1927.

Sudha, M., A. Karthikeyan, P. Nagarajan, M. Raveendran, N. Senthil, M. Pandiyan, K. Angappan, J. Ramalingam, M. Bharathi, and R. Rabindran. 2013b. Screening of mungbean (*Vigna radiata*) germplasm for resistance to Mungbean yellow mosaic virus using agroinoculation. J. of Plant Pathol. 3:424-430.

Sunitha, S., G. Shanmugapriya, V. Balamani and K. Veluthambi. 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.

Thakur, R. P., P. N. Patel and J. P. Verma. 1977. Genetical relationship between reaction to bacterial leaf spot, yellow mosaic and Cercospora leaf spot diseases in mungbean (*Vigna radiata* L. Wilczek). Euphytica. 26: 765-774.

Thongmeearkom, P., Y. Honda, Y. Saito and R. Syamananda. 1981. Nuclear ultrastructural changes and aggregates of virus-like particles in mungbean cells affected by mungbean yellow mosaic disease. Phytopathol. 7: 41-44.

Truniger, V. and M.A. Aranda. 2009. Recessive resistance to plant viruses. Adv. Virus. Res. 75: 119-159.

van Regenmortel, H. V., D. H. L. Bishop, C. M. Fauquet, M. A. Mayo, J. Maniloff and C. H. Calisher. 1997. Guidelines to the demarcation of virus species. Arch. Virol. 142: 1505-15018.

Vanitharani, R., P. Chellappan, J. S. Pita and C. Fauquet. 2004. Differential roles of AC2 and AC4 of cassava geminiviruses in mediating synergism and suppression of posttranscriptional gene silencing. Journal of Virology 78: 9487–9498.

Vanitharani, R., P. Chellappan and C. M. Fauquet. 2005. Geminiviruses and RNA silencing. Trend Plant Sci. 10: 144-151.

Varma, A. and V. Malathi. 2003. Emerging geminivirus problems: a serious threat to crop production. Annals of Applied Biology. 142: 145-164.

Vasudeva, R. S. 1942. A mosaic disease of cowpea in India. Indian J. Sci. 12: 281-283.

Vavilov, N. I. 1926. Studies on the Origin of Cultivated Plants. Leningrad, Leningrad, Saint Petersburg, Russia.

von Arnim, A. and Stanley, J. 1992. Determinants of Tomato golden mosaic virus symptom development located on DNA B. Virol. 186: 286-293.

Wang, Z. Y., M. D. Yao and Y. D. Wu. 2009. Cross-resistance, inheritance and biochemical mechanisms of imidacloprid resistance in B-biotype Bemisia tabaci. Pest Manag. Sci. 65: 1189-1194.

Akbar, W., M. Aslam, M. A. Maqbool, M. Ali and M. Arshad. 2018. Inheritance pattern of mungbean yellow mosaic disease resistance and gene action for different traits in mungbean (Vigna radiata (L.) Wilczek) under protected and unprotected field conditions. Plant breed. 137 (5): 763-772.

Akbar, W., M. Aslam, M. S. Nawaz-ul-Rehman and R. M. Atif. 2017. Exploration of durable resistance in mungbean against mungbean yellow mosaic disease. Pak. J. Agric. Sci. 54 (2): 415-422.

June 2017Pakistan Journal of Agricultural Sciences 54(2):415-422

Wartig, L., A. Kheyr-Pour, E. Noris, F. D. Kouchkovsky, F. Jouanneau, B. Gronenborn and I. Jupin. 1997. Genetic analysis of the monopartite tomato yellow leaf curl geminivirus: roles of V1, V2, and C2 ORFs in viral pathogenesis. Virol. 228: 132-140.

Xie, Z., L. K. Johansen, A. M. Gustafson, K. D. Kasschau, A. D. Lellis, D. Zilberman, S. E. Jacobsen and J. C. Carrington. 2004. Genetic and functional diversification of small RNA pathways in plants. PLoS Biol. 2: 642-652.

Yadav, R. K. and D. Chattopadhyay. 2011. Enhanced viral intergenic region-specific short interfering RNA accumulation and DNA methylation correlates with resistance against a geminivirus. Mol. Plant Microbe. In. 24: 1189-1197.

Yadav, R. K., R. K. Shukla and D. Chattopadhyay. 2009. Soybean cultivar resistant to Mungbean yellow mosaic India virus infection induces viral RNA degradation earlier than the susceptible cultivar. Virus Res. 144: 89-95.

Zukovskij, P. M. 1962. Cultivated Plants and Their Wild Relatives. Bureau, London.