VIRAL DISEASES OF LEGUMINOUS CROPS

Manisha Srivastava*, U.P. Gupta[1] and Asha Sinha**

Abstract

In this paper viral diseases of leguminours crops and symptomatology is reviewed. Among them Cucumber mosaic, Common mosaic, Peanut Mottle, Cowpea mild mottle, Golden mosaic, Alfalfa mosaic, Sunn-hemp mosaic, Dolichos enation mosaic, Peanut stunt, Dolichos yellow mosaic, Blackgram mottle, Urdbean leaf crinkle, Cowpea severe mosaic, Southern bean mosaic, Bean common mosaic, Mung bean mosaic, Bean yellow mosaic, Tobacco ringspot, Tomato spotted wilt, leaf curl, Necrotic mosaic, Mosaic, and Clover yellow vein mosaic diseases are responsible for loss of leguminous crops. Detection of plant viruses and disease management (management of virus diseases and integrated management) possible through improved resistance and genetic engineering. Some transgenic legumes are resistant to Bean dwarf mosaic virus, Pea seed borne mosaic potyvirus and Alfalfa mosaic viruses.

Introduction

Pulses are considered to be the important source of dietary protein to predominantly vegetarian population of India. However, over the three decades the production remained almost static i.e., between 10 to 14 m tones per annum. In recent years, much emphasis has been directed towards increased cultivation of legume crops. Since, intensive cultivation practices often create new and more severe plant disease problems, it is essential to know the various diseases of these crops and the ways to control them. *Leguminosae* (*Fabaceae*), a large family of dicotyledonous plants, commonly called the pea family, consists of approxt 18000 species. The fruit is typically a pod or legume. Many of the Papilionoideae are important food crops, e.g. *Phaseolus*, *Vicia*, *Pisum sativum* (Pea), *Lens culinaris* (Lentil) and *Arachis hypogea* (Peanut). Others such as *Trifolium* (Clovers) and *Medicago sativa* (Lucerne) are used for forage. Viral diseases are shown to be one of the many factors responsible for loss of leguminous crops **(Table.1)**. Natural infection by Tospovirus of cucurbitaceous and fabaceous vegetable crops in India has been studied by Jain *et al.* (2007). A distinct begomovirus causing dolichos yellow mosaic disease in India has been studied by Maruti *et al.* (2006), Dolichos yellow mosaic virus belongs to a distinct lineage of old world begomoviruses of which biological and molecular properties were described by Maruthi *et al.* (2006). Cloning restriction mapping and phylogenetic relationship of genome components of MYMIV from *Lablab purpureus* has been studied by Singh *et al.* (2005). Yellow mosaic virus infecting soybean in northern India is distinct from the species infecting soybean in southern and western India was studied by Usharani *et al.* (2004). Current status of begomoviruses in the Indian subcontinent has been studied by Narayan Rishi (2004). Development of a specific detection technique for *cowpea golden mosaic virus* was reported by Roy *et al.* (2004). Two newly described begomoviruses of *Macroptilium lathyroides* and common bean was studied by Idris *et al.* (2003). A quantitative method to screen common bean plants for resistance to bean *common mosaic necrosis virus* and genetic

*Department of Botany, Harish Chandra P.G. College, Varanasi-221001, U. P., India.
**Department of Mycology and Plant Pathology, Institute of Agriculture Sciences, B.H.U., Varanasi - 221005, India.

characterization of their differential reactions among host group (Three common bean cultivars to NL-3K strain of bean *common mosaic necrosis virus*) was studied by Strausbaugh *et al.* (2003). Response of urdbean genotypes to powdery mildews, leaf curl, and yellow mosaic disease was evaluated by Barhate *et al.* (2003) Molecular characterization of the rep (Replication initiator protein) protein encoded by black gram isolate of *Indian mungbean yellow mosaic virus* was studied by Pant *et al.* (2001). A dosage- dependent Allele from bean conferring hypersensitive resistance or spreading vascular necrosis in response to the *Potyvirus bean common mosaic virus* was studied by Collmer *et al.* (2000). Identification of variants of *mungbean yellow mosaic virus* by host reaction through nucleic acid spot hybridization was studied by Biswas and varma (2000) and sources of resistance to *bean common mosaic virus* in French bean was studied by Dhar and Gurha (1998).

Symptomatology

Bean yellow mosaic virus (BYMV), a member of the potyvirus group is a flexuous rod, about 750 x 12 nm. Its dilution end point is 10^{-3} to 10^{-4} and the thermal inactivation point varies between 50 and 62 °c depending on the strain. Longevity in vitro varies from 1 to 4 days at room temperature. BYMV and its diseases in Soyabean are found through out Asia, Brazil, and in certain areas of the USA and the former USSR. Early symptoms include vein clearing along the small, branching veins of young leaves. Later, a conspicuous yellowing mottling of the entire leaf develops. Rusty, necrotic spots appear in the yellow areas as the leaves mature. Some strains produce severe mottling and crinkling of the leaves. *Soyabean mosaic virus* (SbMV) and at least 14 closely related potyviruses are reported from soybean (Jain *et al.,* 1992, Qusus *et al.,* 1995). Symptom severity depends on host genotype, virus strain, plant age and environmental conditions. Seedlings arising from infected seeds are spindly, with rugose or crinkled unifoliolate leaves, which may be mottled or curl longitudinally downward. Subsequent leaflets are chlorotic, severely stunted, mottled and rugose. Plants infected early in the season are stunted, have shortened petioles and internodes and often show browning of stems and petioles. Leaves are reduced in size; the youngest show the most severe symptoms (Pacumbaba, 1995). Typically, infected plants mature conspicuously later than uninfected ones and remain green while most other plants have become defoliated and dried.

Bean golden mosaic is caused by *bean golden mosaic virus* (BGMV) belonging to Geminivirus group (Goodman and Bird, 1978). The virion was first purified by Galvez and Castano (1976) and the disease was first reported from southern Brazil in 1961. BGMV has been recorded widely from Mexico, Central America, the Caribbean, Venezuela, Colombia and Argentina (Galvez and Morales, 1989b, and CIAT, 1990). Bean golden mosaic is known variously as bean yellow mottle, bean golden yellow mosaic, bean double- yellow mosaic and 'mosaico dorado' (Galvez and Morales, 1989b). Under dense vector population susceptible genotypes of *P. vulgaris* develop a brilliant golden yellow coloration, starting in the veins of the first trifoliolate leaves within two weeks of sowing. Following exposure to viruliferous whiteflies (*Bemisia tabaci* Genn.), small yellow dots appear near leaf veins about four days later. Young leaves of diseased plants usually become rolled and cupped. Severely affected plants become stunted with bleached leaves, pods often exhibit blotching and seed may be discolored as well as reduced in size and number. Less susceptible cultivars develop less intense symptoms, with a tendency toward remission (Galvez and Morales,

1989b).

Blackeye cowpea mosaic virus (BICMV) and *cowpea aphid borne mosaic virus* (CABMV) are two potyviruses that are pathogenic to cowpea. BICMV was first reported in the USA by Anderson (1955) and CAMV was reported a decade later from Europe and Africa (Bock and conti, 1974). Other potyvirus including pea nut mottle (Demski *et al.,* 1983), cowpea rugose mosaic, cowpea green vein banding and *cowpea severe mottle viruses* have also been reported from naturally infected cowpeas (dos Santos *et al.,* 1981). CAMV is a closely related but distinct virus within the BCMV subgroup of potyviruses (Khan *et al.,* 1993; Mink *et al.,* 1994). Natural infection of cowpea with BICMV or CAMV produces various symptoms such as mottling, interveinal chlorosis, green vein banding, leaf distortion, blistering and stunting of plant (Bock and Conti, 1974). Alfalfa mosaic is caused by *alfalfa mosaic virus* (AMV) and is classified under alfamo virus group. Virus particles are bacilliform with three different lengths; they are readily transmitted both by sap inoculation and nonpersistently by aphids to a wide range of host plants (Bos and Jaspars, 1971). AMV has been noted by Hampton *et al.* (1978) as causing malformation of leaves and mosaic of both red and white clovers. Akita (1981b) described symptoms on red clover as yellow mosaic and leaf wrinkling, while some symptomless plants had a latent infection. Fletcher (1983) reported that leaves of infected subterranean clover plants in New Zealand were smaller than normal and displayed vein- banding and interveinal yellowing.

Bean leaf roll virus (BLRV) is a member of the large group of yellowing viruses, the luteoviruses. The luteovirus group appears to comprise a continuum of serologically related viruses (Waterhouse *et al.,* 1988) and other members of this group, which have been reported to cause yield losses in faba bean, include subterranean clover red leaf virus (SCRLV) in New Zealand (Wilson and Close, 1973) and Australia (Johnstone, 1978), beet western yellows virus (BWYV) in the USA (Duffus, 1964) and chickpea stunt virus (CpSv). BLRV on faba bean produces symptoms of upward leaf rolling and thickening, accompanied by interveinal chlorotic yellowing (Cockbain, 1983). Early infection can suppress flowering and pod set.

Pea enation mosaic virus (PEMV) is the only member in its group and shares no known serological relationship with any other plant virus. The infectious genome is packaged in two isometric spherical nucleoprotein particles. The coat protein molecular weight is 21 KDa. The genome is composed of two ssRNA species consisting of 570 nucleotides (RNA1) and 4253 nucleotides (RNA2) (Demler and de Zoeten, 1994). A third small RNA (RNA3) is occasionally observed and is considered to be satellite RNA. PEMV was first identified by Osborn (1953) from fababean. Diagnostic symptoms in pea include translucent flecks or 'windows', together with vein – clearing and malformation in leaves and stipules. Plants are usually severely stunted and distorted. Pods are typically deformed severely and produce characteristic out growths or proliferations on its surface. The virus causes death of plants in susceptible cultivars or when plants are infected at an early stage. PsbMV was first discovered in Europe (Musil, 1966). The virus was reported shortly after in the United States (Stevenson and Hagedorn, 1969) and described by Hampton (1969) as 'Pea fizzle top virus. The virus is seed borne in pea, lentil and faba bean. Common symptoms include epinasty or down ward leaf rolling, mild chlorosis, vein clearing, mosaic, a general stunting of the plant, terminal resetting (a result of the reduction in internodal

growth) and deformed pods that fail to set. Mid season pea cultivars typically display more severe resetting symptoms than the early cultivars (Hampton and Baggett, 1970). *Pea streak carlaviruses* (PeSV) virions are slightly flexuous rod –shaped. Examination of purified virions of the PeSV – Walla Walla strain however, revealed three distinct particle sizes of 640 nm, 140nm and 95 nm in length (Larsen *et al.,* 1993). Veerisetty and Brakke (1977) reported that PeSV and alfalfa latent virus (ALV) were two distinct viruses based on coat protein molecular weight and comparative sizes of their RNAs. The capsid protein of PeSV with a molecular weight of 28 KDa encapsidates ss RNA of 8.1 kb as resolved in glyoxal – denaturing gels (Larsen *et al.,* 1993). Pea streak carlavirus (PeSV) was first reported on peas in Virginia by Zaumeyer in 1938 and thereafter in Wisconsin pea fields by Hagedorn and Walker (1949a). Symptoms in peas are characterized by purple to brown necrotic streaks on stems and petioles, brown necrotic lesions on leaves, and wilting of the plant. Symptoms of affected pods include brown necrotic lesions often associated with sunken areas. Several strains of PeSV exist and have been described as PeSV- Walla Walla (Larsen *et al.,* 1993), PeSV – central Ferry (Kaiser *et al.,* 1993) and alfalfa latent virus (Veerisetty and Brakke 1978). Particles of Red clover vein mosaic carlavirus (RCVMV) are slightly flexuous rods (Varma, 1970). encapsidating a single stranded RNA species with a length of 7.05 Kilobases as determined by glyoxal denaturing agarose gels (Larsen *et al.,* 1996b). The virus coat protein has an apparent molecular weight of 32-33.5 KDa (Veerisetty and Brakke, 1977; Larsen *et al.,* 1996b).

RCVMV was first described in red clover by Osborn in 1937. Hagedorn and Walker (1949b) later described the virus in pea as 'Wisconsin pea stunt' by which it is still often referred. Symptoms include marked vein clearing accompanied by a mosaic in pea leaves. Diagnostic symptoms in field infected plants include severe stunting, pronounced shortening of internodes resulting in resetting of leaves, lack of apical dominance and proliferation of axillary buds. Pod formation is severely affected when plants are infected before flower set thus reducing yields, moreover plant death can occur if infected at an early stage of growth (Hagedorn, 1984).

Bean yellow mosaic virus (BYMV) is a member of the potyvirus group. Synonyms include bean virus 2, and pea mosaic (or pea common mosaic) virus (PMV), it infects a wide range of legumes including soybean, fababean, clovers and a number of non- legume species. In general, plants infected with BYMV have considerably reduced growth and a very few survive to produce viable seed. The virus is transmitted through seed and can survive in stored seed upto five years (Gladstones, 1970). Plants infected with BYMV initially show yellow mottling of leaves, followed by the formation of many small leaves near the top of the plant and curling over of the stem into the form of a Shepherd's crook (Gondran *et al.,* 1994).

Broad bean true mosaic (BBTMV) and Broad bean strain (BBSV) viruses are members of the comovirus group. The two viruses are unrelated serologically and ELISA can be used for their identification. Foliar symptoms on faba bean are very similar for both the viruses, with chlorotic mottling in patches on the leaves and some times leaf deformation, although some leaves on infected plant may appear normal (Gibbs *et al.,* 1968). Apical dieback may occur in cooler conditions BBTMV and BBSV have been reported from Europe, North Africa and Asia. Bos *et al.* (1988) regarded BBTMV and BBSV as economically important viruses of faba bean, with frequent seed transmission. Pea early browning, primarily a disease of pea is locally important

in Netherlands and England (Boulton, 1996). *Pea early browning tobravirus* (PEBV) can also infect faba bean. PEBV is almost always symptom less on faba bean (Cockbain *et al.,* 1983) although virus concentration in plants may be high. The broad bean yellow band virus (BBYMV) serotype of PEBV (Russo *et al.,* 1984), however, can produce yellow vein – banding, rings and line patterns on the pods, although symptom less infection with BBYBV can also occur.

Pea seed borne mosaic potyvirus (PSbMV) causes a disease of prime importance in pea but the virus can affect faba beans as well. Three pathotypes (strains) have been reported from pea: P-1, L-1 and P-4, all of these can infect faba bean, but pathotype L-1 is the only one, which can infect lentil (*Lens culinaris*). Foliar symptoms of PSbMV in faba bean are vein- clearing and mosaic, particularly on younger leaves and are quite similar to those of BYMV. Fagbola *et al.,* (1996) noted varying severity of symptoms, including stunting and severe leaf, flower and pod distortion. Sterility mosaic is the most important disease of Pigeon pea in India and Nepal (Reddy *et al.,* 1990b). Recent studies at the Scottish Crops Research Institute (SCRI), Invergowrie, UK, on the similar reversion disease of black currant have detected a virus as the causal agent (A. T. Jones, SCRI, Invergowrie, UK, 1996, Personal Communication). The disease was first reported from Pusa in the state of Bihar, India, more than 65 years ago by Alam (1931) who gave the first detailed description of the disease. In the field, sterility mosaic can be easily identified as patches of bushy, pale green plants without flowers or pods (Reddy *et al.,* 1990 b). The leaves of infected plants are small with light and dark green mosaic. Mosaic symptoms initially appear as vein – clearing on young leaves. Strains of sterility mosaic prevalent in Bihar state of India and in Nepal cause severe internodal shortening of the branches and clustering of leaves which some times become filiform, the disease is transmitted by mite *Aceria cajani*. Cucumber mosaic is caused by *Cucumber mosaic virus* (CMV), which is a member of the cucumovirus group and like BYMV, is transmitted by aphids. According to Jones and McLean (1989) the initial symptoms of aphid transmitted CMV are not unlike those of BYMV. Further, unlike BYMV infected plants, CMV infected plants set and produce seeds but majority of the seedlings arising from infected seed die shortly after emergence or within 6-8 weeks post emergence. Plants that survive are stunted and have down curled leaflets.

Clover yellow vein mosaic is caused by *Clover yellow mosaic virus* (CYVV) of the potyvirus group, it infects several species in the *Leguminosae*, particularly *Trifolium Spp.* Hampton *et al.,* (1978), in a survey, observed mottling or mosaic when CYVV was artificially inoculated into white clover, but none when inoculated on to red clover. White clover mosaic is caused by White clover mosaic virus (WCMV) of the potexvirus group. Symptoms of WCMV on white clover have been described by Gibbs *et al.,* (1966) as chlorotic rings, patches and flecks which later develops into brown necrotic flecking. Carr (1984) described the typical symptoms on white, red, alsike and crimson clovers in Britain as light green striping or flecking of the leaves between the veins, although sometimes the infected white clover remains asymptomatic. Johnstone and McLean (1987) observed that WCMV causes systemic chlorotic mottle and vein- clearing in subterranean clover. Red clover necrotic mosaic is caused by red clover necrotic mosaic virus (RCNMV), which is classified in the dianthovirus group. RCNMV has been identified from USA (Edwardson and Christie, 1986), Canada (Rao and Hiruki, 1985), Czehoslovakia, Poland and Sweden (Musil *et al.,* 1983) and Britain (Prame and Harkness, 1987), where it was first isolated only in 1971. In red

clover, RCNMV causes veinal chlorosis, often followed by severe necrosis and deformation. The plants become weakened, stunted and may even die (Gilmour and Pemberton, 1976; Bowen and Plumb, 1979), however, if it survive, the symptoms tend to fade over the summer months (Carr, 1984). Subterranean clover red leaf is caused by Soyabean dwarf luteovirus (SDV), which is transmitted through aphid. Symptoms on subterranean clover are observed as intense reddening of the leaflets that develops progressively from the leaflet margins (Johnstone and McLean, 1987). These symptoms resemble those attributable to manganese deficiency and indeed the plants have only about half the manganese content as compared to healthy plants.

Detection of Plant Viruses

Diagnosis remains difficult for plant virus diseases. It has long been far from simple to demonstrate the presence of viruses. Viruses cannot be seen with the naked eye nor with hand lens or light microscope. The tools commonly used by plant pathologists for examining diseased plant for plant infecting fungi and bacteria are:

1. Identification of plant species and genotype infected.
2. Study of symptomatology
   a. External
   b. Internal (Inclusion bodies).
3. Study of transmissible nature by Sap inoculation, Transmission by vectors such as insects, mites, nematodes etc. Tissue grafting.
4. Establishment of pure culture and determination of host range of virus (es).
5. Determine physical properties of the virus (es) Longevity in vitro, Dilution end point and Thermal inactivation point.
6. Electron microscopy to determine particle shape and size of virus (es).
7. A – Detection by serological methods (Precipitin test, gel diffusion test, ELISA, Western blotting, Dot immuno binding assay).
   B – Detection by molecular biological methods (PCR based detection, Nucleic acid spot hybridization).
8. Proof of pathogenicity (Koch's postulates) by inoculation with purified virus preparation. Manuals for identifying disease are produced by the international centers of the consultative group on International Agricultural Research (CGIAR), including Centro Internacional de Agricultura Tropical (CIAT), International Center for Agricultural Research in the Dry Areas (ICARDA), International Crops Research Institute for the Semi – Arid Tropics (ICRISAT) and International Institute of Tropical Agriculture (IITA), on common bean, tropical pasture legumes, lentil, faba bean, ground nut, chick pea, pigeon pea and cowpea, these are widely available especially in developing countries.

Management of Viral Diseases

There is no cure for virus diseased plants and viruses cannot usually be eliminated from plants once they infect them. Since, replication of the virus is intimately associated with host metabolism any chemical interference will affect host metabolism and likely be phytotoxic. Some

chemicals, such as benzimidazole compounds (Carbendazim) or cytokinin – like substance may reduce symptom severity or decrease virus multiplication but cannot eliminate virus from infected plants. Viral diseases must be prevented only with proper prophylactic measures. The aim is to opt for indirect virus control by interfering with virus ecology so as to stop or delay the onset of virus incidence and to decrease the rate of progress of viral disease. Control of viral disease is much more difficult than of those caused by other pathogens, because of the complex disease cycle (fig.1), efficient transmission and lack of viricides (Varma, 1993).

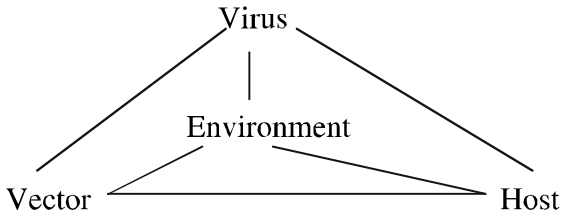

Fig. - 1 Virus disease cycle

The effective management of plant viral diseases demands integration of management practices, such as avoidance of sources of infection, host resistance, cultural practices (such as planting date, rouging infected plants early in the season) and minimal insecticide sprays to control the insect vector, have been effective in reducing virus incidence in legume crops. Measures for avoidance of infection with aphid borne viruses include growing crops at a distance away from known early sources of infection such as clover fields; even so, isolation may not always be effective where the vectors are particularly active (Bos *et al.,* 1988). It is difficult to isolate crops from infection by viruses such as BLRV and pea enation mosaic virus, which persist over long distances in their aphid vectors. It may be possible, especially for seed multiplication to grow crops in areas such as higher ground where the vectors may be less active or arrive at later stages of crop maturity. Sowing earlier in the spring when possible may allow the crop to be more mature when the most damaging period of vector activity occurs. PEBV may be avoided by not growing susceptible crops on land where the virus is known to be present.

Strict statutory seed certification schemes, such as that successfully used for many years in The Netherlands to control the spread of PEBV in pea (Boulton, 1996), and replicase mediate resistance to pea seed – borne mosaic virus (Jones *et al.,* 1998). Transformation of peas to produce agriculturally important insect resistance traits has also been reported (Chrispeels *et al.,* 1998; Charity *et al.,* 1999; Morton *et al.,* 2000). Madappattuparambil *et al.,* (2008) studied transgenic peanut (*Arachis hypogea* L.) plants expressing crylEc and Rice chitinase cDNA (Chi11) exihibit resistance against insect pest *Spodoptera litura* and fungal pathogen *Phaeoisariopsis personata.* Even so, the production and use of seed carrying little or no virus is desirable. Seed lots, or preferable sample plants grown from seed, which better indicate the actual rate of transmission can be screened for infection by ELISA. Those involved with distribution of seed for commercial, experimental or breeding purposes should also ensure that only healthy seed is exchanged or acquired. Production of clean seed is a principal objective in the management of many legume diseases and is often an integral part of disease management strategies. Management of seed borne pathogens of legumes is possibly more important than in other crops including cereals. The best way to avoid seed

borne infection is to identify locations or seasons for production of healthy seed, so avoiding high-risk areas or periods.

Table - 1 Viral Diseases of some important leguminous crops

Disease	Pathogen	Genome	Distribution/ Importance	References
Cucumber mosaic	Cucumber mosaic cucumovirus	ssRNA	USA	Harter (1938), Aderson (1955)
Common mosaic	Bean Common Mosaic Potyvirus	ssRNA	Wide spread	Morales and Bos (1988)
Peanut Mottle	Peanut Mottle Potyvirus	ssRNA	East Africa	Bock *et al.* (1978)
Cowpea mild mottle	Cowpea mild mottle carlavirus	ssRNA	Nigeria	Rossel and Thottappilly (1985)
Golden mosaic	Bean golden mosaic geminivirus	ssDNA	Latin America	Goodman and Bird (1978), Williams (1976)
	Lima bean golden mosaic geminivirus	ssDNA	Nigeria	Vetten and Allen (1983)
Alfalfa mosaic	Alfalfa mosaic alfamovirus	ssRNA	Sudan	Nour and Nour (1962)
Sunn-hemp mosaic, Dolichos enation mosaic	Sunn-hemp mosaic tobamovirus	ssRNA	India	Kassanis and Varma (1975)
Peanut stunt	Peanut stunt cucumovirus	ssRNA	Sudan	Ahmed and Mills (1985)
Dolichos yellow mosaic	Dolichos yellow mosaic geminivirus	ssDNA	India	Capoor and Varma (1950), Harrison *et al.* (1991)
Urdbean leaf crinkle	?	ssRNA	India	Williams *et al.* (1968), Beniwal *et al.* (1980)
Cowpea severe mosaic	Cowpea severe mosaic comovirus	ssRNA	Trinidad	Dale (1949)
Southern bean mosaic	Southern bean mosaic sobemovirus	ssRNA	India	Tremaine and Hamilton (1983)
Alfalfa mosaic	Alfalfa mosaic alfamovirus	ssRNA	Iran, probably widespread	Kaiser (1979), Jaspars and Bos (1980)
Cucumber mosaic	Cucumber mosaic cucumovirus	ssRNA	Widespread?	Purivirojkul and Poehlman (1977)
Bean common mosaic,? Mung bean mosaic	Bean common mosaic potyvirus	ssRNA	Iran and India, probably widespread	Kaiser and Mossahebi (1974)

Continue

Bean yellow mosaic	Bean yellow mosaic potyvirus	ssRNA	Indonesia	Iwaki and Auzay (1978)
Tobacco ringspot	Tobacco ringspot	ssRNA	Sri Lanka	Vignarajah (1978)
	nepovirus			
Tomato spotted wilt, leaf curl	Tomato spotted wilt tospovirus	ssRNA	India	Ghanekar *et al.* (1979)
Cowpea mild mottle	Cowpea mild mottle carlavirus	ssRNA	Tanzania	Mink and Keswani (1987)
Mungbean yellow mosaic	Mungbean yellow mosaic geminivirus	ssDNA	Widespread in South Asia	Honda *et al.* (1983), Harrison *et al.* (1991)
Peanut mottle	Peanut mottle potyvirus	ssRNA	East Africa	Bock *et al.* (1978)
Ringspot	Cucumber mosaic cucumovirus	ssRNA	West Africa, Fiji	Fauquet *et al.* (1979), Brunt and Phillips (1981), Rossel and Thottappilly (1985)
Necrotic mosaic	Cowpea mild mottle carlavirus	ssRNA	Ivory Coast	Fauquet *et al.* (1979), Rossel and Thottappilly (1985)
Mosaic	Cowpea mosaic comovirus, Cowpea severe mosaic comovirus	ssRNA	East Africa Brazil	Kitajima *et al.* (1979), Allen (1983)

Reference. The Pathology of food and pasture Legumes. Edited by D.J. Allen and J.M. Lenne, ICRISAT, International crops Research, CAB International, 1998.London, UK. ? Represents not available

Weeds are very important reservoirs of viruses belonging to groups like cucumo – poty, gemini and tospoviruses, which have a wide host range. Removal of weeds in an around field reduce the incidence of viral diseases. Insecticides are effective in preventing infection and spread with in a crop of luteoviruses such as BLRV since the vector is deterred or killed before virus is transmitted to the host phloem cells. They are less effective when viruses are transmitted in the non – persistent manner. Spread with in the crop of non – persistent viruses in epidemic situations, nevertheless, may be significantly limited by insecticide use. For most viral diseases, resistant lines have been developed by conventional breeding and along with judicious insecticide sprays to control the vector population which helps in management of the disease. Some examples include K-134 in groundnut against bud necrosis virus, for the white fly transmitted geminiviruses like TOLCV, CLCUV, ICMV and yellow mosaic virus in legumes.

In order to avoid development of insecticide resistance among the insect vectors and to reduce insecticide load on population and environment, biological control should be advocated. Unfortunately, biological control of vectors has not received the attention it deserves. Identification of proper and effective bioagent (Parasites and Predators) is a prerequisite for successful execution of biocontrol programme on vectors. The best and simplest approach for avoiding damage by viruses is the use of cultivars with genetic resistance if available, so that the crop becomes less vulnerable to virus. With the advances in the development of regeneration systems of legume

crops (eg. Pea, chickpea fababean, *Medicago* etc.), there is good potential for producing transgenic legumes **(Table 2)** through genetic engineering to reduce losses. Based on five seasons screening

Table - 2 Some examples of Transgenic Legume resistant to virus

Crop/ Virus	Sources of Transgene	Level of resistance	References
Bean dwarf mosaic virus (BDMV)	BV1 or of BDMV	Delayed infection	Yu Ming *et al.,* 2000
Pea seed borne mosaic potyvirus	N1b gene (Viral replicase gene)		Jones *et al.,* 1998
Alfalfa mosaic virus	Coat protein	Partial resistant	Grant *et al.,* 1998

ruder field and artificial conditions cowpea genotypes, Arka Garima, BC-244002, KLS-10, cowpea–263 have been identified as resistant sources to cowpea golden mosaic geminivirus (Chakraborty *et al.,* 1999). Azuki bean has a reproducible and efficient *Agrobacterium*-mediated transformation system (Yamada *et al.,* 2001; El-Shemy *et al.,* 2002). The majority of legume transformation studies have favored the use of *Agrobacterium tumefaciens* to generate transgenic soybeans (Hinchee *et al.,* 1988; Chee *et al.,* 1989), chickpeas (Fontana *et al.,* 1993) and pea (Puonti-Kaerlas *et al.,* 1990, 1992; De Kathen and Jacobsen 1990; Davies *et al.,* 1993; Schroeder *et al.,* 1993; Shade *et al.,* 1994; Zubko *et al.,* 1990). Breeding for resistance to faba bean viruses has been reviewed by Cockbain (1983), Bos *et al.,* (1988) and Makkouk *et al.,* (1993). However, there are few good sources of resistance and breeding programmes are not as advanced in faba bean as in other cool season crop legumes, particularly pea.

Advances in understanding and managing the major diseases of the most economically important crops including soybeans, groundnut and common bean have been relatively rapid. The availability of large germplasm collections and use of multilocational testing have contributed to progress in breeding for resistance. Molecular genetic and plant transformation technologies have made it possible to use novel approaches for developing plants resistant to specific viruses through transfer of alien genes, particularly of viral origin, commonly known as parasite derived resistance (Sanford and Johnson, 1985) includes use of novel viral genes viz. coat protein, replicase, movement, DNA copies of satellite RNAs that reduce symptom expression, defective interfering molecules, ribozymes or antisense RNA. Progress in transformation of large-seeded legumes has been extensively reviewed (Christou, 1997; Nagl *et al.,* 1997; Trick *et al.,* 1997), and more recent progress is presented in **(Table. 3).** There are several approaches that have been used to engineer plants resistant to viruses. The first method aims at introducing genes encoding viral coat protein (coat protein- mediated transgenic resistance to viruses such as TSWV, BPMV, BPMV and PEMV have been obtained in Peanut, Soybean, Bean and Pea crops respectively, The transgenic plants appears to block a receptor in the plant cell that is required for uncoating the viral nucleic acid, or to interfere with viral replication or expression of viral genes. Transformed Pea *(Pisum sativum* L.) lines were produced with two chimeric gene construct encoding the coat protein (CP) of Alfalfa mosaic virus (AMV) strain NZ1, where resistance is manifested by delay in symptom development or escape from infection altogether. A second approach to engineering virus resistance in plant has centered on satellite RNAs. The third approach aims at using antisense virus RNA to govern

Table - 3 Summary of legume transformation systems yielding transformed plants that transmitted the transgenic genotype to progeny reported since or in addition to Atkins and Smith (1997) and Babaoglu *et al.,* (2000).

Species, Genotype	**DNA Delivery**	**Explant**	**Selection**		**Citation**
			Marker	**Agent**	
Red clover (*Trifolium pratense*) NEWRC germplasm	At (EHA101, A208)	Petiole pieces (O)	*nptII*	Kan	Quesenberry *et al.* (1996)
Pigeon pea (*Cajanus cajan* L. Millsp.) N Hyderabad	At (GV2260)	Embryonic axis (O,C)	*nptII*	Kan	Lawrence and Koundal (2001)
	AT (EHA105)	Embryonic axes and cotyledonary nodes (O)	*nptII*	Kan	Satyavathi *et al.* (2003)
Chickpea (*Cicer arietinum*) PG1/PG12/Chafa/Turkey	At (C58C1/ EHA101)	Embryonic axis (O)	*pat*, *nptII*	PPT, Kan	Krishnamurthy *et al.* (2000)
Guar (*Cyamopsis tetragonoloba*) Lewis/ Santa Cruz	At (LBA4404)	Cotyledons (O)	*nptII*	Kan	Joersbo *et al.* (1999)
Jack	EHA105	Immature cotyledon (E)	hpt	Hyg	Yan *et al.* (2000)
BR-16/DokoPC/BR-19/Conquista	MB	Embryonic axis (O)	*ahas*	imazapyr	Aragão *et al.* (2000)
Bert	EHA101	Cotyledonary node (O)	*hph*	Hyg	Olhoft *et al.* (2003)

Continue

Lupin (*Lupinus angustifolius*) Unicrop/Merrit	At (AgL0)	Axillary shoot embryonic	*bar*	PPT	Pigeaire *et al.* (1997)
Lentil (*Lens culinaris* Medik) Laird/CDC599–23	MB	Cotyledonary node (O)	*als*	Chlorsulfuron	Gulati *et al.* (2002)
Bean (*Phaseolus vulgaris*) Olathe/Carioca	MB	Embryonic axes (O)	*bar*	PPT	Aragão *et al.* (2002)
Pea (*Pisum sativum*) 94-A26/ Bolero/Hadlee/ Crown/ Courier/89T46.UK	At (AGL1)	Immature cotyledons (O)	*nptII*	Kan	Grant *et al.* (1998)
Laser, Heiga	At (EHA105; C58C1/LBA4404)	Cotyledons (O)	*nptII, bar*	Kan, PPT	Nadolska-Orczyk and Orczyk (2002)
Mung bean (*Vigna radiata* L. Wilczek) K-851	At (LBA4404)	Cotyledonary node (O)	*nptII*	Kan	Jaiwal *et al.* (2001)
Fava bean (*Vicia faba*) Mythos	At (EHA101 and 105)	Epicotyls (O,C) Internodal stem	*nptII*	Kan	Böttinger *et al.* (2001)
Azuki bean (*Vigna angularis* Willd. Ohwi/Ohashi) Beni-dainagon	At (EHA105)	Elongated epicotyls (O,C)	*nptII*	Kan	Yamada *et al.* (2001)

Genotype, DNA delivery system, explant, selectable marker gene and agent, and citation are presented. N, Not identified; At, A. tumefaciens; Ar, A. rhizogenes; MB, microprojectile bombardment. Agrobacterium strain and tissue culture type: O, organogenesis; E, embryogenesis; and C, callus are indicated in parentheses.

transgenic resistance. Considerable progress has been made towards the successful management of important diseases of most legume crops through the search for host resistance. The durability of resistance against many of the diseases of legumes remains inadequately tested, in part because of the relatively recent development of resistant cultivars and perhaps partly because of the protective effects of the complex cropping systems in which most legumes are grown. There are a number of causes of site differential interactions where one of which is the 'breakdown' of race specific resistance (Allen, 1983). If race non- specific resistance tends to be qualitative trait,

then it may be highly significant as technologies are now available for genetic marking for characters under quantitative control (Edwards, 1992, Dudley, 1993). Recent advances in marker technology have paved the way to a new revolution in our ability to manipulate quantitative traits in crop improvement. Combined disease resistance is required in most legume production systems. This has proved relatively easy to attain in some cases like range of viruses in cowpea, and rosette of groundnut. A case of true multiple resistances conferred by the R3/I gene in common bean has been mentioned. The conserved sequences among genes for disease resistance cloned from widely different plant hosts (Kanazin *et al.,* 1996) seem likely to be useful in identifying evolutionarily related genes in legumes including soyabean. The importance of combined disease resistance in pigeon pea for resource poor farmers can't be overemphasized, recently, the line ICPL 87119, which is resistant to both wilt and sterility mosaic, has been released as 'Asha' (meaning 'hope') for general cultivation in India (Reddy *et al.,* 1990). Pea has been successfully transformed using immature cotyledons (Grant *et al.,* 1995) and a similar method is showing some success in chickpea. However, other legumes such as faba beans are proving to be very difficult to transform, particularly because of the lack of success in regenerating plants in tissue culture. Providing efficient transformation and regeneration systems are developed, the genetic engineering approach will be promising for developing cultivars with resistance to viruses using viral coat protein genes, like the work in progress on several viruses of groundnut (McDonald *et al.,* 1980*).*

Integrated Management

In any integrated program, host plant resistance should be the basic component since it influences other management practices. Management of legume diseases should address the cropping system as a whole, if full advantage is to be taken of available control measures. Other components include the adjustment of sowing date, use of cultivars of different duration, crop rotation, intercropping, cultivation and landform, plant population and spacing patterns. Interactions between different diseases, abiotic stresses such as drought and unfavorable temperatures also must be considered. The economic and socio – economic aspects of integrated management packages should be examined when these packages are being field-tested. Our ultimate aim must be the development of safe, economic and durable management strategies for a range of farm situations, this will probably be achieved only through a combination of measures into an integrated management system including cultural practices, crop and varietal mixtures and in some systems also chemicals, as well as host plant resistance. More recently, investigation of farmers management of common bean diseases in the Great Lakes region of Africa revealed that local strategies were based on microclimate regulation (through sowing density, time of sowing, choice of soil type, foliage reduction, weeding and staking), genetic diversity (use of species and varietal mixtures) and sanitation (seed selection and removal of debris) (Trutmann *et al.*, 1993). In these systems, it was concluded that enhanced disease management should be possible through improved resistance while maintaining variability, but emphasis should be given to technologies, which does not decrease the existing management flexibility. Genetic engineering for plant virus resistance is a rapidly growing approach to develop virus resistant genotypes. The use of virus genes has proven to be a versatile and broadly applicable strategy for achieving resistance and field experiments to date are very promising.

References

Akita, S., (1981b): Bulletin of the National Grassland Research Institute, 20, 93-102.

Alam, M., (1931): Administration report of the Botanical section 1930-31, Department of Agriculture, Bihar and Orissa, India, pp 42-65.

Allen, D.J., (1983): John Wiley & Sons, Chichester, UK,413 pp.

Anderson, C.W., (1955): Plant Disease Reporter, 39, 349-352.

Atkins CA, and Smith P.M.C., (1997): Springer-Verlag, Berlin, pp 283–304.

Babaoglu M, Davey MR, and Power, J.B., (2000): AgBiotechNet 2000, Vol 2, June ABN 050. http://www.agbiotechnet.com/reviews/june00/html/Babaoglu.htm.

Barhate, B.G., Bendre, N.J., Patil, J.V. and Kute, N.S., (2003): Legume Research, 26(2), 140-142.

Biswas, K.K., and Varma, A., (2000): Indian Phytopathology, 53, 134-141.

Bock, K.R. and Conti, M., (1974): CMI /AAB Descriptions of Plant Viruses No. 134, Common wealth Agricultural Bureaux, Farnham Royal, UK.

Bos, L. and Jaspars, E.M.J., (1971): CMI Descriptions of Plant Viruses No. 46, 4 pp.

Bos, L., Hampton, R.O. and Makkouk, K.M., (1988): Kluwer Academic Publishers, Dordrecht, The Netherlands, pp. 591-615.

Boulton, R.E., (1996): Plant Pathology, 45, 13-28.

Carr, A.J.H., (1984): British Crop Protection Council, Corydon, UK, pp. 72-80. Chakraborty, S., Singh, M., Kalloo, G., Banerjee, M.K. and Pandey, P.K., (1999): Annals of Applied Biology 134 Supplement, pp. 40-41.

Charity, J.A., Anderson, M.A., Bittisnich, D.J., Whitecross, M., and Higgins, T.J.V., (1999): Mol. Breed, 5, 357–365.

Chrispeels, M.J., M.F.G. Desa, and T.J.V. Higgins., (1998): Seed Sci. Res, 8, 257–263. Christou P (1997): Field Crop Res., 53, 83–97.

CIAT, (1990): Bean Program Centro Internacional de Agricultura Tropical, Cali, Colombia.

Cockbain, A.J., (1983): The Faba bean (Vicia faba L.) Butterworths, London, UK. Pp. 421-462.

Cockbain, A.J., Woods, R.D. and Calilung, V.C.J., (1983): Annals of Applied Biology, 102, 495-499.

Collmer, C.W., Marston, M. F., Taylor, J.C. and John, Molly, (2000): Molecular Plant- Microbe Interactions, 13(11); 1266-1270.

Davies DR, Hamilton J. and Mullineaux P.M., (1993): Plant Cell Rep., 12, 180–183.

De Kathen, A. and Jacobsen, H.J., (1990): Plant Cell Rep., 9, 276– 279.

Demler, S.A and de Zoeten, G.A., (1994): Webster, R.G. and Granoff, A. (eds) Encyclopedia of Virology Academic Press, San Diego, USA, pp. 1083-1089.

Demski, J.W., Alexander, A.T., Stefani, M.A. and Kuhn, C.W., (1983): Plant Disease, 67, 267- 269.

Dos Santos, A.A., Lin, M.T. and Kitajima, E.W., (1981): Phytopathology, 71, 890 (Abstr.).

Dudley, J.W., (1993): Crop Science, 33, 660-668.

Duffus, J.E., (1964): Phytopathology, 54, 736-738.

Edwards, M., (1992): Field Crops Research, 29, 241-260.

Edwardson, J.R. and Christie, R.G. (eds), (1986): Viruses infecting forage legumes. Vol. 1, Florida Agricultural Experiment Stations Monograph Series No. 14, 1-246.

El-Shemy HA, Khalafalla MM, Wakasa K. and Ishimoto M., (2002): Cell Mol. Biol. Lett., 7, 709-719.

Fagbola, O., Boulton, R.E., Jellis, G.J. and Dhillon, N.P.S., (1996): Plant varieties and seeds, 9, 43-51.

Fletcher, J.D., (1983): Journal of Agricultural Research, 28, 279- 282.

Fontana GS, Santini L, Caretto S, Frugis G. and Mariotti D., (1993): Plant Cell Rep., 12, 194–198.

Frame, J. and Harkness, R.D., (1987): Grass and Forage Science, 42, 213-223.

Galvez, G.E. and Castano M.J., (1976): Turrialba, 26, 205 – 207.

Galvez, G.E. and Morales, F.J., (1989b): Centro Internacional de Agricultura Tropical, Cali, Colombia, pp. 379-406.

Gibbs, A. J., Guissani- Belli, G. and Smith, H.G., (1968): Annals of Applied Biology, 61, 99-107.

Gibbs, A.J., Anupam Varma and Woods, R.D., (1966): Annals of Applied Biology, 58, 231-240.

Gilmour, J. and Pemberton, A. W., (1976): Disease Notice (Plant Pathology Laboratory, Harpenden, UK) No. 16, 1 P.

Gladstones, J.S., (1970): Field crop Abstracts, 23, 123-148.

Gondran, J., Bournoville, R. and Duthion, C., (1994): INRA, Versailles, 48 pp.

Goodman R.M. and Bird, J., (1978): CMI/AAB Descriptions of plant viruses No. 192, Common Wealth Agricultrural Bureaux, Farnham Royal, UK.

Grant, J., Pither, J.M., Fifield, W., Cooper, P. and Timmerman, V.G., (1998): European Association of Grain Legume Research, 372-373.

Grant, J.E. Cooper, P.A., McAra, A.E. and Frew, T.J., (1995): Plant Cell Reports, 15, 254-258.

Hampton, R., Beczner, L., Hagedorn, D., Bos, L., Inouye, T., Barnett, O., Musil, M. and Meiners, J., (1978): Phytopathology, 68, 989-997.

Hampton, R.O. and Baggett, J.R., (1970): Plant Disease reporter, 54, 355-358.

Hampton, R.O., (1969): Phytopathology, 59, 1029.

Hederow, Z.G., (1980): Tagungsberichte der Academics der Landwirtschaftswissenschaften der DDR, Berlin 184, 361-366.

Hinchee MAW, Connor-Ward DV, Newell CA, McDonnell RE, Sa-to SJ, Gasser CS, Fischhoff, DA, Re DB, Fraley, RT. And Horsch, R.B., (1988): Biotechnology, 6, 915–922.

Hogedron, D.J., (ed.) (1984): The American Phytopathological Society, St. Paul, Minnesota, USA, 57 pp.

Idris, A.M., Hiebert, E., Bird, J. and Brown, J.K., (2003): Phytopathology, 93(7); 774-783.

Jain, R.K., Bag, S., Umamaneswaran, K. and Mandal, B., (2007): J. Phytopathology, 155, 22-25.

Jain, R.K., McKern, N.M., Tolin, S.A., Hill, J.H., Barnett, O.W., Tosic, M., Ford, R.E., Beachy, R.N., Yu, M.H., Ward, C.W. and Shukla, D.D., (1992): Phytopathology, 82, 294-299.

Johnstone, G.R., (1978): Australian Journal of Agricultural Research, 29, 1003-1010. Johnstone, G.R. and McLean, G.D., (1987): Annals of Applied Biology, 110, 421-440. Jones, A., L., Johansen, I, E., Bean, S.J., Bach, 1. and Manle, A.J., (1998): J. Gen. Virol., 79, 3129-3137.

Jones, R.A.C. and McLean, G.D., (1989): Annals of Applied Biology, 114, 609-637. Kaiser, W.J., Klein, R.E., Larsen, R.C. and Wyatt, S.D., (1993): Plant Disease, 77, 922-926.

Kanazin, V. Marek, L.F. and Shoemaker, R.C., (1996): Proceedings of National Academy of Sciences, USA 93, 11746-11750.

Khan, J.A., Lohuis, D., Goldbach, R. and Dijkstra, J., (1993): Journal of General Virology, 74, 2243-2249.

Larsen, R.C., Kaiser, W.J. and Wyatt, S.D., (1996b): Plant Disease, 80, 709.

Larsen, R.C., Wyatt, S.D. and Kaiser, W.J., (1993): Phytopathology, 83, 1374 (Abstr.).

Madappattuparambil, Ravindran Beena, Tuli, Rakesh, Gupta, Aparna Dutta, Kirti, Pulugurtha Bharadwaja, (2008): Transgenic Plant Journal, Vol. 2, No. 2, pp.,157-164.

Makkouk, K.M., Bos, L., Horn, N.M. and Srinivasa Rao, B., (1993): John Wiley and Sons, Chichester, UK pp. 179- 192.

Maruthi, M.N., Manjunatha, B., Rekha, A.R., Govindappa, M.R., Colvin, J. and Muniyappa V., (2006): Annals of Applied Biology, 1-9.

Maruthi, M.N., Rekha, A.R., Govindappa, M.R., Colvin, J. and Muniyappa V., (2006): Plant Pathology, 55, 290.

Mc Donald, D. and Raheja, A.K., (1980): Advances in Legume Science, Royal Botanic Gardens, Kew, UK, pp. 501-514.

Mink, G.I., Vetten, H.J., Ward, C.W. Berger, P.H., Morals, F.J., Myers, J.M., Silbernagel, M.J. and Barnett, O.W., (1994): Archives of virology, 139, 231-235.

Morton, R.L., Schroeder, H.E., Bateman, K.S., Chrispeels, M.J. Armstrong, E. and Higgins, T.J.V., (2000): Proc. Acad. Sci., (USA) 97, 3820-3825.

Musil, M., (1966): Biologia (Bratislavia), 21, 133-138.

Musil, M., Micinski, B., Leskova, O. and Pruszynska, M., (1983): Zeszyty Probleowe Postepow Nauk Rolniczych, 291, 227-232.

Nagl, W. Ignacimuthu, S. and Becker, J., (1997): J Plant Physiol., 150, 625–644.

Narayan Rishi, (2004): Indian Phytopathology, 57(4), 396-407.

Osborn, H.T., (1935): Phytopathology, 25, 160-177.

Pacumbaba, R.P., (1995): Plant Disease, 79, 193-195.

Pant, V., Gupta, D., Roychoudhury, N., Malathi, V.G., Varma, A. and Mukherjee, S.K., (2001): Journal of General Virology, 82, 2559-2567.

Powell- Abel, P., Nelson, R.S., De, B., Hoffmann, N., Rogers, S.G., Fraley, R.T. and Beachy, R.N., (1986): Science, 232, 738-743.

Puonti-Kaerlas J, Eriksson T, and Engstr_M.P., (1990): Theor. Appl. Genet, 80, 246– 252.

Puonti-Kaerlas J, Eriksson T, and Engstr_M.P., (1992): Theor. Appl. Genet, 84, 443–450. Qusus, S., Cramer, C.L., Saghaimaroof, M.A. and Tolin, S.A., (1995): Phytopathology, 85, 632.

Rao, A.L.N. and Hiruki, C., (1985): Plant Disease, 69, 959-961.

Reddy, M. V., Sharma, S.B. and Nene, Y.L., (1990b): CAB International, Wallingford, UK, pp. 303-347.

Roy, Anirban and Malathi, V.G., (2004): J. Plant Biochemistry and Biotechnology, 13, 131-133.

Russo, M., Gallitelli, D. Vovlas, C. and Savino, V., (1984): Annals of Applied Biology, 105, 223-230.

Sanford, J.C. and Johnson, S.A., (1985): J. Theor. Biol., 11B, 395-405.

Schroeder HE, Schotz AH, Wardley-Richardson T, Spencer D, and Hig-gins T.J.V., (1993): Plant Physiol., 101, 751–757.

Shade R.E., Schroeder H.E., Pueyo J.J., Tabe L.M., Murdock L.L., Higgins T.J.V. and Chrispeels M.J., (1994): Biotechnology, 12, 793–796.

Singh, Sudheer, Kumar, Chakraborty, S.,Singh, Ashok Kumar, Pandey, P.K., (2005): Bioresource Technology, 1-8.

Steverson, W.R. and Hagedorn, D.J., (1969): Phytopathology, 59, 1051-1052 (Abstr.). Strausbaugh, C. A., Miklas, P.N., Singh, S.P., Myers, J.R., and Forster, R.L., (2003): Phytopathology, 93(6), 683-690.

Strausbaugh, C. A., Myers, J.R., Forster, R.L. and Mcclean, P.E., (2003): Phytopathology, 93(11), 1430-1436.

Tanimura, Y., Matskawa, I. and Banba, H., (1985): Bulletin of Hokkaido Prefectural Agricultural Experiment station, 5, 85-93.

Thurston, H.D., (1992): West View Press, Boulder, Colorado, USA.

Trick HN, Dinkins RD, Santarem ER, Di R, Samoylov V, Meurer C, Walker D, Parrott WA, Finer JJ, and Collins G.B., (1997): Plant issue Cult Biotechnol., 3, 9–26.

Trutmann, P., Voss, J. and Fairhead, J., (1993): International Journal of Pest management, 39, 334-342.

Usharani, K.S., Surendranath, B., Haq., Q.M.R. and Malathi, V.G., (2004): Current Sci., 80(6), 845-850.

Varma, A., (1993): Crop protection and sustainable Agriculture, Wiley, Chichester (Ciba Foundation Symposium 177) p. 140-157.

Veerisetty, V. and Brakke, M.K., (1977): Virology, 83, 226-231.

Veerisetty, V. and Brakke, M.K., (1978): Phytopathology, 68, 59-64.

Vishwa Dhar, and Gurha, S.N., (1998): Indian Jr. of Pulses Research, 11(1), 138-139.

Waterhouse, P.M., Gildow, F.E. and Johnstone, G.R., (1988): CMI/AAB Descriptions of plant viruses No. 339, Commonwealth Agricultural Bureaux, Farnham Royal, UK.

Whitehead, R. (eds), (1996): CAB International, Wallingford, UK.

Williams, R.J., (1976): Plant Disease Reporter, 60, 853-857.

Wilson, J. and Close, R.C., (1973): New Zealand Jr. of Agricultural Research, 16, 305-310.

Yamada T, Teraishi M, Hattori K and Ishimoto M., (2001): Plant Cell Tissue Organ Cult., 64, 47-54.

Yu Ming, H., Sanders, R., Ursin, V.M. and Gilbertson, R.L., (2000): Molecular Plant Microbe Interactions, 13, 297-308.

Zaumeyer, W.J., (1938): Journal of Agricultural Research, 56, 747-772.

Zubko EI, Kuchuk NV, Tumanova LG, Vikonskaya NA. And Gleba Y.Y., (1990): Biopolymers Cells, 3, 80–84.