Tropical Food Legumes: Virus Diseases of Economic Importance and Their Control

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Tropical Food Legumes: Virus Diseases of Economic Importance and Their Control

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Abstract

Diverse array of food legume crops (Fabaceae: Papilionoideae) have been adopted worldwide for their protein-rich seed. Choice of legumes and their importance vary in different parts of the world. The economically important legumes are severely affected by a range of virus diseases causing significant economic losses due to...
reduction in grain production, poor quality seed, and costs incurred in phytosanitation and disease control. The majority of the viruses infecting legumes are vectored by insects, and several of them are also seed transmitted, thus assuming importance in the quarantine and in the epidemiology. This review is focused on the economically important viruses of soybean, groundnut, common bean, cowpea, pigeonpea, mungbean, urdbean, chickpea, pea, faba bean, and lentil and begomovirus diseases of three minor tropical food legumes (hyacinth bean, horse gram, and lima bean). Aspects included are geographic distribution, impact on crop growth and yields, virus characteristics, diagnosis of causal viruses, disease epidemiology, and options for control. Effectiveness of selection and planting with virus-free seed, phytosanitation, manipulation of crop cultural and agronomic practices, control of virus vectors and host plant resistance, and potential of transgenic resistance for legume virus disease control are discussed.

1. INTRODUCTION

Legumes belong to the family Leguminasae (Fabaceae), consisting of four subfamilies, the Papilionoideae, Caesalpinoideae, Mimosoideae, and Swartzioideae (Lewis, Schrire, MacKinder, & Lock, 2005). The Papilionoideae includes the major food legumes, soybean (Glycine max), groundnut (peanut or monkeynut, Arachis hypogaea), common bean (bean, French bean, or kidney bean, Phaseolus vulgaris), cowpea (southern pea, Vigna unguiculata), pigeonpea (red gram, arhar, Cajanus cajan), chickpea (Garbanzo or bengal gram, Cicer arietinum), pea (field pea, Pisum sativum), mungbean (green gram, Vigna radiata), urdbean (black gram, Vigna mungo), faba bean (Vicia faba), and lentil (Lens culinaris). They are usually cultivated in the tropical and subtropical areas of the world. Soybean, groundnut, common bean, cowpea, pigeonpea, mungbean, and urdbean are usually cultivated during the hot season, while chickpea, pea, faba bean, and lentil are cultivated during the cool season. Legume seeds (also called pulses or grain legumes) are second only to cereals as a source of human diet and animal feed. Nutritionally, legume seeds are two to three times richer in protein than cereal grains. Groundnut and soybean seeds are also rich in lipids. Diversity and importance of various food legumes vary in different parts of the world (Fig. 9.1). Groundnut is by far the most widely cultivated legume. Soybean is cultivated in a much larger area, dominating the legume production in both area and production (Fig. 9.1). It is extensively used in food industry and also as biofuel. Common beans and other legumes, viz., soybean, groundnut, cowpea, and chickpea, are the major source of food in Latin America, while lentil, pigeonpea, chickpea, mungbean, and urdbean are important in South Asia.
In the Middle East and North Africa, faba bean, lentil, and chickpea are particularly important. Groundnut, cowpea, and common bean are the most important food legumes in Africa. As per the 2012 production statistics (FAOSTAT, 2012), soybean, bean, groundnut, chickpea, pigeonpea, cowpea, lentil, and pea are cultivated in an area of 198.7 million hectares around the world, soybean dominating with 72% of production area.

Virus diseases are the major biotic constraints to legumes production, especially in the tropics and subtropics (Loebenstein & Thottappilly, 2003; Rao, Kumar, & Holguin-Peña, 2008; Sastry & Zitter, 2014). Cultivated food legumes are susceptible to natural infection by at least 150 viruses, belonging to different genera (ICTV, 2012). The seed-transmitted viruses such as Bean common mosaic virus (BCMV), Soybean mosaic virus (SbMV), Cucumber mosaic virus (CMV), Alfalfa mosaic virus (AMV), Peanut mottle virus (PeMoV), Peanut stripe virus (PStV), Pea seed-borne mosaic virus (PSbMV), and Bean yellow mosaic virus (BYMV) are widely distributed and infect several legume crops. They have quarantine importance and also serve as primary source of inoculum in virus ecology and epidemiology (Albrechtsen, 2006; Sastry, 2013). Complex virus diseases like groundnut rosette are endemic only to Africa and chickpea stunt to Asia and Africa (Alegbejo & Abo, 2002; Kumar, Jones, & Waliyar, 2008). Peanut bud necrosis virus (PBNV), Pigeonpea sterility mosaic virus (PPSMV), Mungbean yellow mosaic virus (MYMV), and Mungbean yellow mosaic India virus (MYMIV) are confined to South East Asia (Kumar, Kumari, & Waliyar, 2008; Malathi & John, 2008; Mandal et al., 2012). Begomoviruses infecting common bean are prevalent in the Latin America (Navas-Castillo, Fiallo-Olive, & Sanchez-Campos, 2011). Faba bean necrotic yellows virus

![Figure 9.1 Percentage share of global production area of major food legumes. Total area = 198,764,140 ha. Source: FAO Production Statistics of 2012.](image-url)
(FBNYV) is predominant on cool season food legumes in West Asia and North Africa (Kumari & Makkouk, 2007; Makkouk & Kumari, 2009; Makkouk, Pappu, & Kumari, 2012). However, viruses such as BCMV, SbMV, PeMoV, AMV, and CMV are worldwide in distribution on legume crops (Loebenstein & Thottappilly, 2003). The virus that emerged as a great threat to groundnut during the last decade is Tobacco streak virus (TSV) (Kumar, Prasada Rao, et al., 2008). Many whitefly-transmitted legume begomoviruses have been characterized either as distinct species or as new pathotypes (Malathi & John, 2008; Qazi, Ilyas, Mansoor, & Briddon, 2007; Rey et al., 2012; Varma, Mandal, & Singh, 2011). The impact of virus diseases of food legumes on crop growth and yield is variable depending upon the crop cultivar, the virus strain or pathotype, and the time and duration of infection, season, location, and climate (Jones & Barbetti, 2012). The viruses infecting legumes have been reviewed (Bos, 2008; Hughes & Shoyinka, 2003; Malathi & John, 2008; Mishra, John, & Mishra, 2008).

In this chapter, the distribution, diversity of causal viruses, diagnosis, epidemiology, and control of virus diseases of economic significance of major annual food legumes are reviewed. In addition, begomoviruses infecting minor food legume crops are discussed.

2. VIRUS DISEASES OF MAJOR FOOD LEGUMES

The taxonomic position and modes of transmission of viruses causing or associated with diseases of major tropical food legumes are summarized in Table 9.1. For other biological and physicochemical characteristics of these viruses, refer to ICTvdB Management (2006) and ICTV (2012).

2.1. Soybean

Soybean is grown in tropical, subtropical, and temperate climates during warm, moist periods. The major soybean producing countries are the United States, Brazil, Argentina, China, India, Paraguay, Canada, Uruguay, and Ukraine (FAOSTAT, 2012). Nearly 70 viruses are known to naturally infect soybean worldwide (Hartman, Sinclair, & Rupe, 1999; Hill, 2003). Among them, diseases caused by SbMV, Tobacco ring spot virus (TRSV), PBNV, TSV, Soybean dwarf virus (SbDV), and begomoviruses are considered to be economically important (Wrather et al., 2010) (Table 9.1).
<table>
<thead>
<tr>
<th>Crop</th>
<th>Disease</th>
<th>Causal virus (acronym)</th>
<th>Virus genus</th>
<th>Modes of transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soybean</td>
<td>Mosaic</td>
<td><em>Soybean mosaic virus</em> (SbMV)</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td>Bud blight</td>
<td><em>Tobacco ring spot virus</em> (TRSV)</td>
<td>Nepovirus</td>
<td>Sap, nematode, seed</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Cowpea severe mosaic virus</em> (CPMSMV)</td>
<td>Comovirus</td>
<td>Sap, beetles (SP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Peanut bud necrosis virus</em> (PBNV)</td>
<td>Tospovirus</td>
<td>Sap, thrips (C)</td>
</tr>
<tr>
<td>Brazilian bud blight</td>
<td>Tobacco streak virus (TSV)</td>
<td></td>
<td>Ilarvirus</td>
<td>Sap, thrips-aided</td>
</tr>
<tr>
<td>Dwarf</td>
<td></td>
<td><em>Soybean dwarf virus</em> (SbDV)</td>
<td>Luteovirus</td>
<td>Aphids (C)</td>
</tr>
<tr>
<td>Yellow mosaic</td>
<td></td>
<td>Several begomoviruses</td>
<td>Begomovirus</td>
<td>Whiteflies (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(e.g., <em>Mungbean yellow mosaic virus</em> (MYMV), <em>Bean golden mosaic virus</em> (BGMV))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Groundnut</td>
<td>Rosette</td>
<td><em>Groundnut rosette assistor virus</em> (GRAV)</td>
<td>Luteovirus</td>
<td>Aphids (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Groundnut rosette virus</em> (GRV)</td>
<td>Umbravirus</td>
<td>Sap, aphids(C), GRAV-dependent</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Satellite RNA</td>
<td></td>
<td>Aphids (C), GRAV-, and GRV-dependent</td>
</tr>
<tr>
<td></td>
<td>Spotted wilt</td>
<td><em>Tomato spotted wilt virus</em> (TSWV)</td>
<td>Tospovirus</td>
<td>Sap, thrips (CP)</td>
</tr>
<tr>
<td></td>
<td>Bud necrosis</td>
<td><em>Peanut bud necrosis virus</em> (PBNV)</td>
<td>Ilarvirus</td>
<td>Sap, seed, thrips aided</td>
</tr>
<tr>
<td></td>
<td>Stem necrosis</td>
<td><em>TSV</em></td>
<td>Ilarvirus</td>
<td>Sap, seed, thrips aided</td>
</tr>
<tr>
<td></td>
<td>Clump</td>
<td><em>Peanut clump virus</em> (PCV)</td>
<td>Pecluvirus</td>
<td>Sap, seed, fungus</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Indian peanut clump virus</em> (IPCV)</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Mottle</td>
<td><em>Peanut mottle virus</em> (PeMoV)</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td>Stripe</td>
<td><em>Peanut stripe virus</em> (PStV)</td>
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<tr>
<td></td>
<td>Yellow mosaic</td>
<td><em>Cucumber mosaic virus</em> (CMV)</td>
<td>Cucumovirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
</tbody>
</table>

*Continued*
<table>
<thead>
<tr>
<th>Crop</th>
<th>Disease</th>
<th>Causal virus (acronym)</th>
<th>Virus genus</th>
<th>Modes of transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common Bean</td>
<td>Common mosaic and black root</td>
<td><em>Bean common mosaic virus</em> (BCMV) and <em>Bean common mosaic necrosis virus</em> (BCMNV)</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td>Golden mosaic</td>
<td>BGMV</td>
<td>Begomovirus</td>
<td>Whiteflies (C)</td>
</tr>
<tr>
<td></td>
<td>Golden yellow mosaic</td>
<td><em>Bean golden yellow mosaic virus</em> (BGYMV)</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Dwarf mosaic</td>
<td><em>Bean dwarf mosaic virus</em> (BDMV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mosaic due to CMV</td>
<td>CMV</td>
<td>Cucumovirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td>Cowpea</td>
<td>Mosaic due to potyviruses</td>
<td><em>Cowpea aphid-borne mosaic virus</em> (CABMV) and <em>Bean common mosaic virus—blackeye cowpea mosaic strain</em> (BCMV-BICM)</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td>Mosaic due to comoviruses</td>
<td><em>Cowpea mosaic virus</em> (CPMV) and CPSMV</td>
<td>Sobemovirus</td>
<td>Sap, seed, beetles (SP)</td>
</tr>
<tr>
<td></td>
<td>Mosaic due to sobemovirus</td>
<td><em>Southern bean mosaic virus</em> (SBMV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stunt</td>
<td>CMV</td>
<td>Cucumovirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BCMV-BICM</td>
<td>Potyvirus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Golden mosaic and yellow mosaic</td>
<td><em>Cowpea golden mosaic virus</em> (CGMV)</td>
<td>Begomovirus</td>
<td>Whiteflies (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Mungbean yellow mosaic India virus</em> (MYMIV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Dolichos yellow mosaic virus</em> (DoYMV)</td>
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</tr>
<tr>
<td></td>
<td>Mild mottle</td>
<td><em>Cowpea mild mottle virus</em> (CMMV)</td>
<td>Carlavirus</td>
<td>Sap, seed, whiteflies (NP)</td>
</tr>
<tr>
<td></td>
<td>Chlorotic mottle</td>
<td><em>Cowpea chlorotic mottle virus</em> (CCMV)</td>
<td>Bromovirus</td>
<td>Sap, beetles (SP)</td>
</tr>
<tr>
<td></td>
<td>Mottle</td>
<td><em>Cowpea mottle virus</em> (CPMoV)</td>
<td>Carmovirus</td>
<td>Sap, seed, beetles (SP)</td>
</tr>
</tbody>
</table>
### Pigeonpea Sterility mosaic

**Sterility mosaic**

Pigeonpea sterility mosaic virus (PPSMV)  
*Emaravirus*  
*Sap, mites (SP)*

**Yellow mosaic**

MYMV, *Rhycosia mosaic virus* (RhMV) and *Tomato leaf curl New Delhi virus* (ToLCNDV)  
*Begomovirus*  
*Whiteflies (C)*

### Mungbean and Urdbean Yellow mosaic

MYMV, *Mungbean yellow mosaic India virus* (MYMIV) and *Horsegram yellow mosaic virus* (HgYMV)  
*Begomovirus*  
*Whiteflies (C)*

<table>
<thead>
<tr>
<th>Leaf curl</th>
<th>PBNV</th>
<th>Vesnavirus</th>
<th>Sap, thrips (CP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leaf crinkle</td>
<td>Urdbean leaf crinkle virus disease (ULCVD)</td>
<td>uncharacterized</td>
<td>Sap, seed, beetles (SP)</td>
</tr>
</tbody>
</table>

### Chickpea Stunt

*Begomovirus*  
*Whiteflies (C)*

<table>
<thead>
<tr>
<th>Chlorotic stunt</th>
<th>Chickpea chlorotic stunt virus (CpCSV)</th>
<th>Polerovirus</th>
<th>Aphids (C)</th>
</tr>
</thead>
</table>
| Chlorotic dwarf | Chickpea chlorotic dwarf virus (CpCDV),  
*Chickpea redleaf virus* (CpRLV), *Chickpea yellow mosaic virus* (CpYV), *Chickpea chlorosis virus* (CpCV), *Chickpea chlorosis Australia virus* (CpAV)  
*Bean yellow dwarf virus* (BeYDV) | *Mastrevirus* | *Leaf hoppers (C)* |

*Continued*
<table>
<thead>
<tr>
<th>Crop</th>
<th>Disease</th>
<th>Causal virus (acronym)</th>
<th>Virus genus</th>
<th>Modes of transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pea</td>
<td>Mosaic due to potyviruses</td>
<td><em>Pea seed-borne mosaic virus</em> (PSbMV)</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Bean yellow mosaic virus</em> (BYMV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Enation mosaic</td>
<td><em>Pea enation mosaic virus-1</em> (PEMV-1)</td>
<td>Enamovirus</td>
<td>Sap, aphids (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Pea enation mosaic virus-2</em> (PEMV-2)</td>
<td>Umbravirus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Top yellows</td>
<td>BLRV</td>
<td>Luteovirus</td>
<td>Aphids (C)</td>
</tr>
<tr>
<td>Faba bean</td>
<td>Necrotic yellows</td>
<td><em>Faba bean necrotic yellows virus</em> (FBNYV)</td>
<td>Nanovirus</td>
<td>Aphids (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Faba bean necrotic stunt virus</em> (FBNSV)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leaf roll</td>
<td>BLRV</td>
<td>Luteovirus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mosaic and necrosis</td>
<td>BYMV</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td>Mottle</td>
<td><em>Broad bean mottle virus</em> (BBMV)</td>
<td>Bromovirus</td>
<td>Sap, seed, beetles (SP)</td>
</tr>
<tr>
<td>Lentil</td>
<td>Yellows and stunt</td>
<td>BLRV</td>
<td>Luteovirus</td>
<td>Aphids (C)</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>FBNYV</em></td>
<td>Nanovirus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mosaic and mottle</td>
<td>PSbMV</td>
<td>Potyvirus</td>
<td>Sap, seed, aphids (NP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BYMV</td>
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<td></td>
<td></td>
<td>CMV</td>
<td>Cucumovirus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Broad bean stain virus</em> (BBSV)</td>
<td>Comovirus</td>
<td>Sap, beetle (SP)</td>
</tr>
</tbody>
</table>

*NP, nonpersistent; SP, semipersistent; C, circulative; CP, circulative and propagative.
2.1.1 Mosaic

The yield losses due to mosaic disease caused by SbMV range from 8% to 50% under natural field conditions and can reach up to 100% in severe outbreaks (Arif, Stephen, & Hassan, 2002; Cui, Chen, & Wang, 2011; Hill, 2003) and when coinfected with Bean pod mottle virus (BPMV), Cowpea mosaic virus (CPMV), AMV, and TRSV (Cui et al., 2011; Hwang et al., 2011; Malapi-Nelson, Wen, Ownley, & Hajimorad, 2009). Symptoms induced by SbMV depend on the host genotype, the virus strain, the plant age, and the temperatures at which the plants are grown (Hill, 2003). Nine distinct strain groups, G1–G7, G7A, and C14 have been reported based on differential reactions on selected soybean cultivars (Cho & Goodman, 1979; Lim, 1985).

SbMV detection is based on ELISA, RT-PCR, real time-PCR, near infrared spectroscopy, and aquaphotomics (Cui et al., 2011; Jinendra et al., 2010). Plants grown from SbMV-infected seed often constitute the primary inoculum source with secondary spread, which occur rapidly, by aphids (Cui et al., 2011).

Control of SbMV is difficult because of its relatively broad host range, the number of aphid species that transmit the virus, and the frequency of seed transmission. Thus, utilization of virus-free seeds and control of aphid populations are effective management measures against SbMV. Rouging of early-infected plants in fields meant for seed production is recommended. Spraying with insecticides imidacloprid WP (wettable powder), benfuracarb EC (Commission Regulation), and acephate WP can reduce aphid population and SbMV incidence under field conditions (Kim, Roh, Kim, Im, & Hur, 2000). Soybean plants, cross-protected with an attenuated isolate of SbMV, showed negligible SbMV incidence (Kosaka & Fukunishi, 1994).

In order to achieve integrated control of SbMV in soybean, utilization of host–plant resistance was found to be the best option (Pedersen, Grau, Cullen, Koval, & Hill, 2007). A number of soybean accessions (germplasm) and cultivars carrying resistance to SbMV have been identified and used in the breeding programs (Cui et al., 2011). Since SbMV is genetically variable and continuously evolving via recombination and spontaneous mutations, strong directional breeding line selection can lead to the occurrence of resistance-breaking isolates (Gagarinova, Babu, Stromvik, & Wang, 2008).

Resistance against SbMV in soybean has been reported to be controlled by a single dominant gene (Wang, Gai, & Pu, 2003) or very closely linked genes against G1–G7 strain groups (Ma, Chen, Buss, & Tolin, 2004). Three independent dominant resistance loci $R_{sv1}$, $R_{sv3}$, and $R_{sv4}$ conferring partial or complete genetic resistance to all SbMV strains have been identified.
$Rs_v2$ was initially assigned to the resistance gene in the soybean cultivar OX670 and later dropped when it was known to actually possess two resistant genes $Rs_v1$ and $Rs_v3$ (Gunduz, Buss, Ma, Chen, & Tolin, 2001). $Rs_v1$ is a single locus, multiallelic gene (Zheng, Chen, & Gergerich, 2005). SbMV strain G7 overcomes the resistance conferred by $Rs_v1$ and results in systemic necrosis of virus-infected plants. The resistance conferring genes have been deployed in China, the United States, Canada, and other countries for developing SbMV-resistant soybean cultivars (Cui et al., 2011).

Incorporation of multiple resistance genes into soybean cultivars through gene pyramiding should become a high priority for soybean breeders to develop durable resistance to SbMV. Pyramiding viral resistance genes against SbMV is reported to be benefited by gene mapping and marker-assisted selection (MAS) with PCR-based markers for the $Rs_v3$ gene conferring resistance to three of the most virulent strains G5, G6, and G7 of SbMV (Jeong et al., 2002). Molecular markers of three resistance genes have been developed based on mapping with several molecular techniques such as restriction fragment length polymorphism, random amplified polymorphism DNA, amplified fragment length polymorphism (AFLP), simple sequence repeats (SSRs), and single nucleotide polymorphisms (SNPs) to assist in plant breeding programs (e.g., Hwang et al., 2006; Shi et al., 2009, 2011; Wang et al., 2011).

Arif and Hassan (2002) in Pakistan reported that PI 88788 germplasm line from China showed resistance to SbMV under field conditions, and the resistance in PI 88788 to SbMV-G1 was controlled by a single, partially dominant gene; however to SbMV-G7, the same gene was completely dominant (Gunduz, Buss, Chen, & Tolin, 2004). The inheritance and mapping of genes resistant to SbMV strain SC14 in soybean accessions in China have led to identification of markers for choosing resistance gene in soybean breeding programs and cloning of resistance genes (Li et al., 2006). $Rs_v1$-mediated resistance to SbMV-G7 strain in soybean genotypes, Williams 82, PI 96983, and L78–379 was shown to be due to SbMV strain–specific protein P3 (Hajimorad, Eggenberger, & Hill, 2006). The resistance to SbMV-G7, governed by $Rs_v3$ gene, was attributed to cytoplasmic inclusion cistron of the virus modulating virulence and symptom expression (Zhang et al., 2009). Cylindrical inclusion protein of SbMV strains, G7H and G5H, was shown to be the pathogenic determinant in the two resistant cultivars L29 and Jinpumkong-2 in Korea (Seo et al., 2009). Genes governing resistance in 12 soybean genotypes in China to SbMV strains, SC4 and SC8, was found to be present at different loci (Wang et al., 2012).
Development of transgenic disease resistance to soybean, despite progress in other important crop plants, has advanced slowly. Soybean cv. 9341 transformed with coat protein (CP) gene showed resistance to SbMV strains G2, G6, and G7 (Steinlage, Hill, & Nutter, 2002; Wang, Eggenberger, Nutter, & Hill, 2001). Investigations on virus-induced gene silencing utilizing SbMV-G2 strain and soybean cultivars with Rsv1-resistant gene are likely to provide new insight into the soybean signaling network required for incorporating stable resistance (Zhang, Grosic, Whitham, & Hill, 2012). The complete sequence of soybean genome will facilitate the molecular cloning and characterization of three resistance (R) genes and elucidating their resistance signaling pathways that are likely to provide a better understanding of the co-evolutionary events of the R genes and SbMV genome (Schmutz et al., 2010). Information from such studies will help to develop novel strategies against SbMV and other genetically related viruses. Importance of RNA silencing as a tool to uncover gene function and engineer novel traits in soybean was reported (Kasai & Kanazawa, 2012). Robust RNAi–based resistance to mixed infection of SbMV, AMV, and BPMV in soybean plants was developed by expressing separate short hairpins from a single transgene (Zhang et al., 2011). This approach has a potential to develop multiple virus resistance in soybean and other legumes. Three microRNAs (miRNAs) known to regulate gene functions, involved in soybean’s response to SbMV infection, were identified, and their interaction with genes conferring resistance is likely to elucidate mechanisms underlying pathogenesis by SbMV (Yin, Wang, Cheng, Wang, & Yu, 2013).

2.1.2 Dwarf

Soybean dwarf (SbD) disease, caused by SbDV, was reported from Japan, Indonesia, Africa, Australia, and New Zealand. SbDV host range includes pea, Trifolium species, bean, Lupinus species, Medicago species, and V. faba (Hartman et al., 1999). Several distinct strains of SbDV were reported based on symptomatology in soybeans, aphid vector relationships, physicochemical properties, and molecular characteristics. The complete nucleotide sequences of the genomic RNAs of four SbDV strains were determined (Terauchi et al., 2001). Specific monoclonal antibodies (MAbs) can discriminate different strains of SbDV in ELISA (Mikoshiba, Honda, Kanematsu, & Fujisawa, 1994). Dot-blot hybridization test was developed for the detection and discrimination of S, P, Y, and D strains of SbDV (Yamagishi, Terauchi, Kanematsu, & Hidaka, 2006).
As primary and secondary spread of SbDV is carried by aphid vectors, organophosphorus insecticides have been used in Japan to minimize virus spread. Soybean cultivars “Adams” and “Yuuzuru” have shown tolerance to SbDV. The highly resistant cultivar “Tsurukogane” was released in Hokkaido, Japan, in 1994 (Hartman et al., 1999). The virus can be introduced successfully into soybeans without the aid of aphid vectors by transferring full-length cDNA clone with the aid of a gene gun, thus facilitating screening of genotypes under laboratory conditions (Yamagishi, Terauchi, Honda, Kanematsu, & Hidaka, 2006). In an attempt to produce SbDV-resistant transgenic soybean plants, a vector construct containing inverted repeat-SbDV-CP genes spaced by β-glucuronidase sequences was delivered into soybean somatic embryos via microprojectile bombardment. The T₂ plants 2 months after inoculation with SbDV by aphids showed negligible SbDV-specific RNA and remained symptomless. Additionally, they contained SbDV-CP-specific small interfering RNAs (siRNAs) suggesting that the T₂ plants acquired resistance to SbDV through RNA silencing-mediated process (Tougou et al., 2006). The T₂ progenies derived from soybeans transformed with positive-sense SbDV-CP gene remained symptomless after inoculation with SbDV through aphids and additionally showed little SbDV-specific RNA (Tougou et al., 2007). These results show good prospects for generating genetically engineered SbDV-resistant soybean cultivars.

2.1.3 Bud blight
In soybean, bud blight symptoms (curving of the terminal bud followed by necrosis) are induced during disease development by several taxonomically distinct viruses such as SbMV (Hartman et al., 1999), TRSV (Hartman et al., 1999), TSV (Arun Kumar, Lakshmi Narasu, Usha, & Ravi, 2008; Hartman et al., 1999), Tomato spotted wilt virus (TSWV) (Nischwitz, Mullis, Gitaitis, & Csinos, 2006), PBNV (Bhat, Jain, Varma, & Lal, 2002), Cowpea severe mosaic virus (CPSMV) (Anjos & Lin, 1984), and Cowpea mild mottle virus (CPMMV) (Almeida, Piuga, et al., 2005). Among these, bud blight caused by TRSV and PBNV are discussed next.

Bud blight caused by TRSV was reported from the United States, China, and India. Yield reduction was attributed to reduced pod set and seed formation (Hill, 2003). TRSV can be seed-transmitted to the extent of up to 100% (Frison, Bos, Hamilton, Mathur, & Taylor, 1990) and can remain viable in seeds for at least 5 years. Detection of TRSV in soybean seed can be done by duplex real-time-PCR (Yi, Chen, & Yang, 2011). The virus is easily sap transmissible, and no efficient true arthropod vector of TRSV has been
identified. However, it is reported to be transmitted nonspecifically by few insects and mites (Aphis gossypii, Myzus persicae, Melanoplus differentialis, Epi-
trix hirtipennis, Thrips tabaci, and Tetranychus species). The dagger nematode (Xiphinema americanum) is an inefficient vector, and the infection generally remains confined to roots (Hill, 2003).

In the absence of seed-borne inoculum, the disease first appears on the plants located at the periphery in soybean fields and advances inward as the season progresses. The disease spread depends on the availability of inoculum, from crops and weeds, adjacent to the field. More infection occurs in fields next to legume pastures and relatively less on those next to maize (Zea mays) fields. The hosts among legumes, which may act as reservoirs include Crotalaria intermedia, Cyamopsis tetragonoloba, Lupinus spp., Melilotus spp., Phaseolus lunatus, bean, pea, Trifolium pretense, and cowpea (Hill, 2003).

Virus-free soybean seed should be used in commercial fields. It may be desirable to avoid fields with dagger nematodes. Since the disease spread depends on the TRSV-susceptible crops and weeds next to soybean fields and to the presence of insect vector populations (Hill, 2003), location of soybean fields next to maize fields is recommended. One genotype (PI 407287) of wild soybean (Glycine soja) was shown to be resistant to the virus (Hartman et al., 1999).

PBNV causes chlorosis and necrosis of leaves, stems and buds, and stunting of soybean (Bhat et al., 2002). The disease was shown to cause severe yield losses to soybean crops in Maharashtra, India (Arun Kumar, Lakshmi Narasu, Usha, & Ravi, 2006). Unlike TRSV, PBNV is not transmitted through soybean seed.

2.1.4 Brazilian bud blight
Brazilian bud blight, caused by TSV, is currently known to occur in several countries that include Brazil, Argentina, the United States, and India (Arun Kumar et al., 2008; Rebedeaux, Gaska, Kurtzweil, & Grau, 2004). The characteristic symptoms are chlorosis and necrosis of leaves, stems and buds, and stunting. Bud blight symptoms caused by TSV are similar to those caused by TRSV and PBNV. A TSV isolate causing soybean bud blight disease in Brazil (TSV-BR) was reported to be a distinct strain which shared 81.3% and 80.7% nucleotide sequence homology with the CP gene of TSV-WC and TSV-MV (mungbean isolate from India), respectively (Almeida, Sakai, et al., 2005). The TSV isolate that caused necrosis in Maharashtra, India, was characterized by analyzing CP gene sequences and designated as TSV-SB (Arun Kumar et al., 2008). TSV was reported to be transmitted through
soybean seed up to 90% (Frison et al., 1990). Thus, planting of virus-free seed is essential to minimize primary spread. Resistance of soybean to TSV has not been reported.

2.1.5 Yellow mosaic due to begomoviruses

Begomoviruses, viz., *Abutilon mosaic virus*, *Bean golden mosaic virus* (BGMV), *Euphorbia mosaic virus*, *Horsegram yellow mosaic virus* (HgYMV), MYMV, *Rhynchosia mosaic virus*, *Soybean crinkle leaf virus*, *Soybean golden mosaic virus*, *Tomato leaf curl Karnataka virus*, *Soybean mild mottle virus* (SbMMV), *Soybean chlorotic blotch virus* (SbCBV), *Soybean chlorotic spot virus* (SoCSV), and *Soybean yellow mosaic virus* have been reported to infect soybean under field conditions in different countries, and they are associated with yellow mosaic disease (YMD) (Alabi, Kumar, Mgbechi-Ezeri, & Naidu, 2010; Hartman et al., 1999; Malathi & John, 2008; Raj, Khan, Snehi, Srivastava, & Singh, 2006). Combined yield losses due to begomoviruses were estimated to exceed $300 million in black gram, mungbean, and soybean in India (Varma & Malathi, 2003). The identity of these begomoviruses is based on geminate virion morphology, whitefly transmission, and genome sequence analysis.

Sequence analysis of genomic components of the begomovirus isolates causing YMD in soybean from different locations of India revealed that they are the isolates of MYMV and MYMIV (Girish & Usha, 2005; Usharani, Surendranath, Haq, & Malathi, 2004, 2005). Recently, remote sensing technique was applied to determine the distribution of YMD in soybean (Gazala et al., 2013).

In Northwestern Argentina, sequence analysis of a begomovirus isolate from soybean indicated that it was closely related to *Sida mottle virus* (Rodriguez-Pardina, Zerbini, & Ducasse, 2006). In addition, BGMV, *Soybean blistering mosaic virus* (SbBMV), and *Tomato yellow spot virus* (ToYSV) were shown to infect soybean in Argentina (Alemandri et al., 2012). BGMV occurred at the highest incidence followed by SbBMV and ToYSV. Three distinct begomoviruses, i.e., BGMV, *Sida micrantha mosaic virus*, and *Okra mottle virus*, have been reported to naturally infect soybean sporadically in the Central Brazil based on phylogenetic analysis of DNA-A sequences (Fernandes, Cruz, Faria, Zerbini, & Aragao, 2009). Furthermore, the virus that induced chlorotic spots on soybean leaves in Brazil was identified as a novel begomovirus for which SoCSV name was proposed (Coco et al., 2013). In Nigeria, two distinct begomovirus isolates naturally infecting soybean, designated as SbMMV and SbCBV, were characterized (Alabi et al., 2010). The economic importance of these viruses is yet to be determined.
Since chemical control of whitefly vector that transmits YMD causal viruses is neither economical nor environment friendly, soybean germplasm was screened for YMD resistance in the Indian subcontinent under field conditions. Promising lines are yet to be utilized to develop YMD-resistant soybean cultivars (Akshay et al., 2013; Khan, Tyagi, & Dar, 2013; Kumar et al., 2014; Malek, Rahman, Raffi, & Salam, 2013). The cultivars Bossier and improved Pelican and the germplasm accessions TGm 119 and TGm 662 from the International Institute of Tropical Agriculture (IITA), Nigeria, were found to be resistant to *African soybean dwarf virus* (Hartman et al., 1999). Due to the wide genetic variation among the begomoviruses that infect soybeans, evaluation of resistance should be carried in multilocations to identify durable resistance. Screening of soybean germplasm against economically important begomoviruses by agroinoculation approach (as begomoviruses are not sap transmissible) is ideal to identify promising soybean cultivars under laboratory conditions.

### 2.2. Groundnut

The major groundnut-producing countries are China, India, Nigeria, the United States, Myanmar, Sudan, Argentina, Tanzania, and Indonesia (FAOSTAT, 2012). Thirty-two viruses have been reported to naturally infect this crop (Sreenivasulu, Subba Reddy, Ramesh, & Kumar, 2008). Of them, diseases caused by TSWV, PBNV, TSV, *Peanut clump virus* (PCV), *Indian peanut clump virus* (IPCV), PeMoV, PSTV, CMV, and groundnut rosette disease (GRD) caused by a complex of three viral agents, *Groundnut rosette assistor virus* (GRAV), *Groundnut rosette virus* (GRV), and a satellite RNA, are considered to be economically important (Table 9.1).

#### 2.2.1 Rosette

GRD is the most destructive disease in sub-Saharan Africa (SSA). It is known to occur in several African countries that include Malawi, Nigeria, Uganda, Senegal, Burkina Faso, Coté d’Ivoire, South Africa, Niger, and Kenya (Wangai, Pappu, Pappu, Deom, & Naidu, 2001). Numerous epidemics of rosette have been reported in Africa resulting in substantial crop losses (Alegbejo & Abo, 2002; Naidu et al., 1999). Infection before flowering results in over 90% of crop loss. Based on symptoms, rosette is categorized into the chlorotic rosette, which is ubiquitous in SSA, while the green rosette occurs in West Africa, Uganda, and Angola; the third less frequently occurring type, the mosaic rosette, is recorded only in East and Central Africa. Among the three causal agents of GRD, GRAV is the helper.
virus involved in transmission of GRV and satellite RNA; GRV is the helper virus for satellite RNA replication, and disease symptoms are caused by satellite RNA. It also helps encapsidation of GRV RNA into GRAV particles through an unknown mechanism (Naidu et al., 1999). Thus, three components are intricately dependent on each other and play a crucial role in the biology and perpetuation of the disease (Alegbejo & Abo, 2002; Naidu et al., 1999; Taliansky, Robinson, & Murant, 2000). Sequence diversity of rosette disease causal agents from different geographic regions was studied (Deom, Naidu, Chiyembekeza, Ntare, & Subrahmanyam, 2000; Wangai et al., 2001). The causal viruses are readily transmitted by Aphis craccivora in a persistent manner.

The GRD can be tentatively diagnosed in the farmer’s fields based on the characteristic symptoms. In the laboratory, GRD diagnosis is based on sap inoculation onto test plants such as Chenopodium amaranticolor, triple antibody sandwich-ELISA, dot-blot hybridization, gel electrophoresis for satellite RNA, and RT-PCR analysis (Blok et al., 1995; Breyel et al., 1988; Naidu, Robinson, & Kimmins, 1998; Rajeswari, Murant, & Massalski, 1987).

The GRD epidemiology is complex as it involves interactions between two distinct viruses and a satellite RNA, an aphid vector and the host plant in several areas of SSA. None of the three agents of rosette complex is seedborne, and therefore, primary infection is introduced into the crop by viruliferous aphids likely to be derived from off season groundnut volunteers and self-sown plants. Secondary spread occurs from sources within the crop. The spread of GRD is complicated because a single aphid may not always transmit the three viral agents (Naidu, Miller, Mayo, Wesley, & Reddy, 2000). Plants that show symptoms but lack GRAV play no role in the spread of the disease because the CP of GRAV is required for encapsidation and transmission of GRV and satellite RNA. Therefore, the number of plants that possess all the three agents play a crucial role in the secondary spread of the disease in a given field, while the number of plants that show typical GRD symptoms influence yield.

Several approaches to manage GRD include application of pesticides to reduce vector populations, crop cultural practices to delay onset and spread of both the vector and the disease (reviewed by Alegbejo & Abo, 2002; Naidu et al., 1999; Ntare, Olorunju, & Waliyar, 2002; Olorunju & Ntare, 2003; Thresh, 2003). The control of aphid vectors by spraying the groundnut crop with insecticides could effectively control the disease (Davies, 1975). Many resource-poor farmers cannot afford pesticides and
do not control GRD in the SSA. Early sowing may allow the crop to get established before aphid populations reach their peak and thus reduce the incidence of the disease. Dense plant stands discourage aphid infestation since aphids prefer light airy conditions. Nonetheless, only limited success has been achieved when these approaches were not combined.

Sources of resistance to GRD were first identified in groundnut landraces of late maturing Virginia type in West Africa. Resistance to this disease was also identified in the early maturing Spanish types. Resistant Virginia types were used in breeding program throughout SSA resulting in the development of several resistant cultivars (e.g., RMP 12, RMP 91, KH 241-D, and RG 1). Resistance among these cultivars was found to be effective against both chlorotic and green rosette and was governed by two independent recessive genes (Nigam & Bock, 1990). However, these cultivars being long duration were not widely grown. Early maturing chlorotic rosette-resistant Spanish types (90–110 days) suitable for diverse ecosystems of SSA were subsequently released (Bock, Murant, & Rajeswari, 1990; Naidu et al., 1999; Subrahmanyan, Hildebrand, Naidu, Reddy, & Singh, 1998). The majority of early maturing groundnut lines, evaluated in Nigeria in 2001 and 2002, showed resistance to GRD, early leaf spot, and late leaf spot (Iwo & Olorunju, 2009). Of the nine groundnut breeding lines possessing high yield and resistance to all the three diseases, ICGV-IS-96805 performed well at four locations and can be grown widely in SSA. These genotypes showed resistance to GRV but not to GRAV (Olorunju, Kuhn, Demski, Misari, & Ansa, 1991, 1992; Subrahmanyan et al., 1998). The resistance to GRV was shown to breakdown under high inoculum pressure and/or adverse environmental conditions. Most of the earlier studies on inheritance of disease resistance were based on visual symptoms and are applicable only to GRV and its satellite RNA, but not to GRAV. Immunity to all the three causative components of GRD was identified in wild *Arachis* species (Murant, Kumar, & Robinson, 1991; Subrahmanayam, Naidu, Reddy, Kumar, & Ferguson, 2001).

Dwivedi, Gurtu, Chandra, Upadhyaya, and Nigam (2003) determined AFLP diversity among selected rosette-resistant groundnut germplasm (ICGs 3436, 6323, 6466, 9558, 9723, 10347, 11044, 11968, and 12876) and one susceptible (ICG 7827) groundnut accession to identify DNA markers linked with resistance to GRD. Resistance to the aphid vector has been identified in groundnut genotype EC 36892 and in the breeding line ICG 12991 that was shown to be controlled by a single recessive gene. Herselman et al. (2004) first reported the identification of molecular markers
closely linked to aphid resistance and GRD and constructed the first partial genetic map for cultivated groundnut using bulked segregant analysis and AFLP analysis.

Attempts have been made to exploit pathogen-derived resistance (PDR) (GRAV replicase and CP genes, and/or satellite RNA-derived sequences) to develop durable resistance to GRD (Taliansky, Ryabov, & Robinson, 1998). At present, it is not known that any transgenic groundnut lines, that possess GRD resistance, are in the pipeline for future deregulation (Reddy, Sudarshana, Fuchs, Rao, & Thottappilly, 2009).

2.2.2 Spotted wilt

Spotted wilt disease of groundnut caused by TSWV was reported from North and South Americas (Argentina, Brazil, and the United States), several African countries (South Africa, Nigeria, Kenya, Malawi, and Uganda), and Australia (Culbreath, Todd, & Brown, 2003). Losses up to 100% have been reported due to this virus (Culbreath & Srinivasan, 2011). In Asia, a similar disease was shown to be caused by a distinct tospovirus, later named as PBNV (Reddy, 1998). In addition to groundnut, TSWV was reported to naturally infect other legumes, viz., soybean, pea, Tephrosia purpurea, urdbean, mungbean, cowpea, Crotalaria juncea, Canavalia gladiata, faba bean, chickpea, and lentil (EPPO, 1997). TSWV produces a variety of symptoms, viz., concentric ring spots on leaflets, terminal bud necrosis, and severe plant stunting and mottled seed.

ELISA and Western blot analysis were used to differentiate various strains of tospoviruses into serogroups (Adam, Yeh, Reddy, & Green, 1993; Sreenivasulu, Demski, Reddy, Naidu, & Ratna, 1991). TSWV and PBNV are distinct virus species and TSWV belongs to the serogroup I and PBNV to the serogroup IV in the genus Tospovirus (Satyanarayana et al., 1998). The relationships between the isolates of different serogroups were studied by molecular hybridization and nucleotide sequence comparison (Tsompana & Moyer, 2008). Field diagnosis of TSWV infections based on symptoms may mislead as the virus induces a variety of symptoms, often influenced by environment. ELISA, dot-blot hybridization, RT-PCR, immunocapture-RT-PCR (IC-RT-PCR), and real-time fluorescent RT-PCR were applied for the detection of TSWV and other tospoviruses (Bandla et al., 1994; Boonham et al., 2002; Huguenot et al., 1990; Jain, Pappu, Pappu, Culbreath, & Todd, 1998; Resende, de Avila, Goldbach, & Peters, 1991; Rice, German, Mau, & Fujimoto, 1990; Ronco et al., 1989; Weekes, Mumford, Barker, & Wood, 1996).
Global status of tospovirus epidemics in diverse cropping systems and control measures has been reviewed by Pappu, Jones, and Jain (2009). Transmission by thrips appears to be the only means of virus spread (Riley, Joseph, Srinivasan, & Diffie, 2011). The principal vectors of TSWV, *Frankliniella fusca*, and *F. accidentalis* occur on groundnut throughout the Southeastern United States. Since *F. fusca* is the predominant species that reproduces on groundnut, it is considered to be the most important vector.

Adjustment to planting dates and planting at high density are valuable practices to reduce TSWV incidence (Culbreath, Branch, Holbrook, & Tilman, 2009, Culbreath, Tillman, Gorbet, Holbrook, & Nischwitz, 2008). The use of conservation tillage (no-tillage, minimum tillage, or strip tillage) in groundnut results in a lower incidence of spotted wilt and reduced severity of foliar fungal diseases compared to conventional tillage (Cantonwine et al., 2006; Monfort, Culbreath, Stevenson, Brenneman, & Perry, 2007). In general, chemical control of thrips does not significantly reduce the incidence of spotted wilt of groundnut. In some cases, the application of insecticides increased the incidence. Seed treatment or in-furrow application of the neonicotinoid insecticide imidacloprid increased incidence of the disease compared to the nontreated control susceptible cultivars (Todd & Culbreath, 1995).

Field resistance to TSWV was observed in the cultivars Southern Runner, Georgia Browne, Georgia Green, UF MDR 98, Tamrun 96, C-99R, and ViruGard (Culbreath et al., 2003). Intensive screening of breeding lines in multiple breeding programs has resulted in the identification of several sources with moderate to high levels of field resistance to TSWV than that in Georgia Green. They include breeding lines F NC 94002 and F NC 94022, which are used in generating TSWV-resistant groundnut cultivars (Culbreath & Srinivasan, 2011). Newly identified field-resistant groundnut genotypes have recently been evaluated under laboratory conditions against TSWV or thrips to understand the mechanism of resistance (Sundaraj, Srinivasan, Culbreath, Riley, & Pappu, 2014). Thrips feeding and survival were suppressed on some resistant genotypes compared with susceptible genotypes. In Brazil, three peanut breeding lines (IC-1, IC-34, and ICGV 86388) showed resistance to TSWV under glass house and field tests (Nascimento et al., 2006).

An integrated genetic linkage map of cultivated groundnut, constructed from the populations of two recombinant inbred lines (RILs), was employed to map TSWV resistance trait (Qin et al., 2012). Two major quantitative trait loci (QTLs) for TSWV resistance were identified for each RIL.
Wang et al. (2013) studied the genetic mapping and QTL analysis for TSWV and leaf spot disease resistance using F₂ and F₅ generations based on genetic maps derived from Tifrunner × GT-C20 cross.

Genetic engineering methods have been attempted to incorporate resistance to TSWV in groundnut (Li, Jarret, & Demski, 1997). They used nucleocapsid gene of TSWV Hawaiian L isolate to transform the groundnut cultivar New MEXICO Valencia A. The engineered plants showed resistance to TSWV. Groundnut cv. MARC I transformed with CP gene of TSWV showed lower spotted wilt incidence than the field-resistant cv. Georgia Green. Cultivar AT 120 transformed with the antisense nucleocapsid gene of TSWV also showed lower incidence of spotted wilt than comparable controls (Culbreath et al., 2003).

Integration of multiple disease suppressive factors is necessary for controlling TSWV in groundnut. The adaption of genetic, chemical, and cultural practices for disease management was enhanced greatly by the development and use of spotted wilt risk index. Virus index has become an important tool by which growers can assess the relative risk of spotted wilt incidence in a particular field and for the identification of suitable disease suppressive factors that best apply to their situation. Application of the various options such as planting at high density with field-resistant cultivars such as Georgia Green and C-11-2-39, application of phorate and conserved tillage had contributed to substantial reduction of TSWV incidence in Georgia (Culbreath & Srinivasan, 2011).

### 2.2.3 Bud necrosis

Bud necrosis disease (BND) of groundnut was first reported from India. It is known to occur in Nepal, Sri Lanka, Myanmar, Thailand, and parts of China (Reddy, 1998). BND was shown to be caused by a distinct tospovirus, PBNV (synonym *Groundnut bud necrosis virus*, GBNV). The virus also infects mungbean, urdbean, cowpea, pea, soybean, and lablab bean under field conditions (Mandal et al., 2012). The symptoms of BND caused by PBNV and spotted wilt caused by TSWV are similar on groundnut.

PBNV can be identified by characteristic chlorotic and/or necrotic ring spot symptoms on cowpea, cv. C-152. Various formats of ELISA, RT-PCR, and IC-RT-PCR have been used for detection of PBNV in plant samples (Hobbs, Reddy, Rajeshwari, & Reddy, 1987; Thein, Bhat, & Jain, 2003). Disease diagnosis becomes more difficult when there is coinfection of PBNV and TSV because both the viruses produce terminal bud necrosis. The incidence and progress of BND is dependent on several
environmental factors and cropping practices which influence multiplication and spread of the vector, *Thrips palmi*. Primary as well as the secondary spread occurs through inoculum derived from alternate hosts that include mungbean, pepper (*Capsicum annuum*), potato (*Solanum tuberosum*), and the weed *Ageratum conyzoides* (Reddy, Amin, McDonald, & GhaneKar, 1983). Management of BND depends upon the control of *T. palmi*. Even though several weeds have been identified as sources of virus and vector thrips (Reddy et al., 1983), their eradication in the tropics is not practical. Rouging of early-infected plants in the fields can create gaps, which can lead to increased incidence. Insecticidal control of thrips was not effective in reducing virus incidence (Wightman & Amin, 1988). Botanical pesticides, neem leaf and seed extracts, castor cake, and monochrotophos, were found to lower BND incidence (Gopal, Muniyappa, & Jagadeeshwar, 2011).

Depending on the arrival of *T. palmi*, the sowing dates need to be adjusted to avoid them. A good crop canopy results in lower disease incidence (Reddy, 1998). For example, in Southern India, groundnut crops sown early with the onset of rains (mid to late June) escaped PBNV infection as thrips vector infestation usually occurred in July and August (Reddy, Buiel, et al., 1995). In contrast, Thira, Cheema, and Kang (2004) observed maximum PBNV infection in groundnut crops sown during May in Northern India. Further, maintenance of optimum plant density and intercropping with cereal crops such as pearl millet (*Pennisetum glaucum*), sorghum (*Sorghum bicolor*), and maize have contributed to lower percentage of PBNV incidence (Reddy, Buiel, et al., 1995a).

Tolerance or resistance to PBNV and/or thrips vector has been identified in germplasm and breeding lines. Robut 33–1, a cultivar commonly grown by marginal farmers in Asia and Africa, showed field resistance (Reddy, 1998). Several wild *Arachis* germplasm lines showed resistance to PBNV (Reddy et al., 2000). Genotypes ICGV 86388, IC 34, and IC 10 were found to be resistant to PBNV in Thailand (Pensuk, Daengpluang, Wongkaew, Jogloy, & Patanothai, 2002). It was subsequently shown that multiple genes governed resistance to PBNV (Pensuk, Jogloy, Wongkaew, & Patanothai, 2004). Groundnut breeding lines ICGV 90009, ICGV 86999, ICGV 86329, ICGV 91177, ICGV 91234, ICGV 94252, and TG 26 were found to be resistant to PBNV in India (Gopal et al., 2010).

The popular Spanish groundnut cultivar, JL 24, was engineered with *N* gene and the plants from T1 and T2 generations showed partial resistance to PBNV (Chander Rao, Bhatnagar-Mathur, Kumar, Reddy, & Sharma, 2013). The promising transgenic lines are yet to be deregulated.
2.2.4 Stem necrosis

Stem necrosis disease (SND) caused by TSV in India was first reported by Reddy et al. (2001). The disease may have been existing for a long time and attributed to PBNV because of striking similarity in symptoms that include terminal bud necrosis (Prasada Rao et al., 2003). The disease occurred on nearly 225,000 ha in Andhra Pradesh, India, in the year 2000 causing crop losses that exceeded $65 million. TSV is known to occur on groundnut in India, Pakistan, South Africa, and Brazil. The virus has a wide natural host range and infects many crops that include sunflower (Helianthus annuus), soybean, cowpea, mungbean, sunnhemp (Crotalaria juncea), green gram, and black gram (Jain, Vemana, & Sudeep, 2008; Kumar, Prasada Rao, et al., 2008), and it is widely expanding its host range. Characteristic symptoms are necrosis on stems and petioles and necrotic spots on pod shells. Genomes of various isolates of TSV have been partially sequenced (Jain et al., 2008). PBNV and TSV can be distinguished by assays on selected hosts that include cowpea, and P. vulgaris cv. Topcrop. TSV can be detected by ELISA, nucleic acid hybridization, and RT-PCR (Prasada Rao et al., 2003).

Three thrips species Megalurothrips usitatus, F. schultzei, and Scirtothrips dorsalis assist the virus to transmit through pollen. Sunflower and marigold (Tagetes patula) could act as sources of inoculum. Primary source of inoculum is likely to be provided by numerous weeds. The disease is often found near fields surrounded by the parthenium (Parthenium hysterophorus) weed, suggesting its role in providing the primary source of inoculum.

Measures suggested for SND control include the destruction of virus sources, installation of barrier crops, and maintenance of optimum plant population and controlling of thrips through seed treatment. Removal of parthenium from the vicinity of the groundnut fields is expected to reduce the disease incidence. Border and intercropping with maize, pearl millet, or sorghum around the groundnut fields may decrease the disease incidence by obstructing thrips movement. Rouging of early-infected plants may not limit or restrict further spread of the disease. Cultivation of groundnut near sunflower and marigold should be discouraged because they act as a source of virus inoculum and/or thrips. Maintenance of sufficient plant density is important to discourage landing of thrips. Seed treatment with systemic insecticides (imidacloprid) may prevent vector infestation at early stages of crop growth. Limited germplasm screening revealed low disease incidence in ICGV 92267, 99029, 01276, ICG 94379, Kadiri 7, and Kadiri 9 groundnut genotypes (Kumar, Prasada Rao, et al., 2008; Vemana & Jain, 2013).
Efforts to produce engineered groundnut plants with viral genes yielded encouraging results. Groundnut cultivars Kadiri 6 and Kadiri 134 were genetically engineered with TSV-CP gene, and integration of the gene was confirmed in T1, T2, and T3 generations. Engineered plants did not produce symptoms when sap inoculated with TSV (Mehta et al., 2013), but they should be evaluated under field conditions.

2.2.5 Clump

Peanut clump disease (PCD) is known to occur in India, Pakistan, and West Africa (Bragard, Doucet, Dieryck, & Delfosse, 2008; Reddy, Bragard, Sreenivasulu, & Delfosee, 2008). The causal virus of PCD that occurs in the Indian subcontinent is referred to as IPCV, whereas the virus that occurs in Africa is referred to as PCV. IPCV and PCV are causing indistinguishable symptoms. Both PCV and IPCV are shown to infect other economically important poaceous crops and pigeonpea. The annual loss due to PCD on global scale has been estimated to exceed US $38 million (Reddy & Thirumala-Devi, 2003). Furthermore, the disease also has quarantine importance, because the casual viruses are seed transmissible in groundnut, cereals, and millets (Delfosse et al., 1999; Reddy et al., 2008). Several strains of IPCV and PCV have been identified based on serological and genomic diversity. IPCV isolates have been grouped into three distinct serotypes, viz., IPCV-H (Hyderabad), IPCV-D (Durgapura), and IPCV-L (Ludhiana) (Nolt, Rajeshwari, Reddy, Barathan, & Manohar, 1988), whereas the PCV isolates are placed into five distinct groups by using MAbs (Huguenot, Givord, Sommermeyer, & Van Regenmortel, 1989; Manohar, Dollet, Dubern, & Gargani, 1995).

PCD can be readily identified in the farmers' fields by the characteristic symptoms and patchy distribution of infected plants. *C. amaranticolor* is a good diagnostic host for PCV and IPCV. ELISA and nucleic acid hybridization-based tests as well as RT-PCR have been used for the detection of these viruses (Dieryck, Delfosse, Reddy, & Bragard, 2010; Huguenot et al., 1989; Manohar et al., 1995; Reddy et al., 2008). Both PCV and IPCV are soil-borne and transmitted by the plasmodiophorid obligate biotrophic parasite, *Polymyxa graminis* (Reddy et al., 2008).

For the control of PCD, the following cultural practices are suggested:
(a) early sowing of groundnut crop before the onset of monsoon rains;
(b) use of pearl millet as a bait crop to reduce the inoculum load in the soil.
To achieve this, bait crop should be planted soon after the onset of monsoon preferably under irrigation and uprooted in 3 weeks after germination;
(c) avoid rotation with highly susceptible cereal crops such as maize and wheat (*Triticum aestivum*); (d) sowing groundnut crops during postrainy season; (e) rotation with dicot hosts to reduce the inoculum in the soil; and (f) soil solarization during hot summer months. This is achieved by covering well-irrigated soils with a transparent polythene sheet (preferably biodegradable) for at least 3 weeks (Reddy et al., 2008).

No resistance to IPCV was found in nearly 9000 *Arachis* germplasm lines. Resistance was identified in wild *Arachis* species, and it is yet to be incorporated into cultivated groundnut (Reddy & Thirumala-Devi, 2003). Four genetically engineered groundnut lines with IPCV-H-CP gene and two with IPCV-H Rep gene have been developed (Sharma et al., 2006). These events were reported to show resistance to PCD based on contained field trials conducted between 2002 and 2005 on the experimental farm of International Crops Research Institute for Semi-arid Tropics (ICRISAT), Patancheru, India. However, these genotypes have never been evaluated for biosafety, and information on durability of resistance is not available.

### 2.2.6 Mottle and Stripe

PeMoV and PStV are among the several potyviruses reported to infect groundnut naturally. They cause mottle and stripe symptoms, and it is difficult to make a distinction between the infections of these two viruses under field conditions. Symptoms induced by necrotic strains of these two viruses mimic the infections of TSWV and PBNV in groundnut (Sreenivasulu et al., 1988). They are seed-transmitted and relatively widely distributed. These two viruses are also reported to infect other legumes, viz., common bean, soybean, cowpea, peas, and white lupin. In Georgia, losses due to mottle were estimated up to 70%, and in India, it may reach 40% in susceptible groundnut cultivars (Reddy & Thirumala-Devi, 2003). Yield reductions by PStV in groundnut in Georgia were about 7% in experimental plots (Lynch, Demski, Branch, Holbrook, & Morgan, 1988) but can reach up to 70% in early-infected plants. In Northern China, annual yield losses due to PStV are estimated at over 200,000 tons of pods (McDonald, Reddy, Sharma, Mehan, & Subrahmanayam, 1998).

ELISA, dot-blot hybridization, RT-PCR, and IC-RT-PCR-based tests were employed for detection of both PeMoV and PStV in leaf and seed samples (Bijaisoradat & Kuhn, 1988; Dietzgen et al., 2001; Gillaspie, Pittman, Pinnnow, & Cassidy, 2000; Gillaspie, Wang, Pinnnow, & Pittman, 2007; Hobbs et al., 1987).
The transmission of PeMoV through the seed in groundnut (0–8.5%) and other grain legumes (cowpea, mungbean, and common bean) is contributing to the primary spread of PeMoV. In nature, PeMoV and PStV are transmitted by aphids in a nonpersistent manner. Alternate crops (cowpea, soybean, clover, pea, navy bean, French bean, and white lupin) and weeds (Centrosena pubescence, Catsetum macrocarpum, Calopogonium caeruleum, Crotalaria straita, Desmodium siliquiosum, Pueraria phaseoloides, and beggarweed, Desmodium spp.) are facilitating the survival and spread of PeMoV by aphids (Demski & Reddy, 1997).

Since the primary source of PeMoV or PStV inoculum is groundnut seed, planting should be done with seed lots obtained from disease-free areas. Genotypes in which PeMoV is not transmitted through the seed, such as ICG 2716 (EC 76446–292), ICG 7013 (NCAC 17133), and ICG 1697 (NCAC 17090), are useful in containing the spread of PeMoV. These lines were used in conventional breeding program to transmit the nonseed transmissible trait to high yielding groundnut cultivars. The seed of advanced breeding lines from these crosses has been tested for frequency of the virus transmission. Two nonseed transmitting high yielding groundnut genotypes (ICGS 65 and ICGS 76) were identified (Reddy & Thirumala-Devi, 2003). High yielding groundnut genotype ICG 89336 was found to be tolerant to PeMoV. Arachis chacoense and Atriplex pusilla as well as wild Arachis species have been reported to be resistant to PeMoV (Demski & Sowell, 1981). However, these resistant sources are yet to be utilized in breeding programs.

Enforcing of strict quarantine regulations in countries where PStV is known to be restricted to certain locations is important to avoid introduction of the virus into the virus-free locations. Only certified groundnut seed is to be moved between the locations and to countries with no record of PStV occurrence. Attempts made to control PStV by using 10% milk suspension, metasystox or milk alternated with metasystox or pyrimidine carbamate (systemic aphicide) were unsuccessful. Application of plastic film mulch in groundnut field in China was found to reduce PStV incidence (Demski et al., 1993). Attempts to identify genotypes which do not transmit PStV through seed were unsuccessful. Resistance to PStV could not be found in cultivated groundnut but was identified in some wild Arachis species (Culver, Sherwood, & Melouk, 1987). However, this resistance is yet to be transferred to cultivated high yielding groundnut cultivars.

Groundnut plants transformed with PStV-CP gene exhibited high levels of resistance to the virus (Higgins, Hall, Mitter, Cruickshank, & Dietzgen, 2004). Groundnut plants transformed with full-length untranslatable form of
CP gene and 3’ untranslated region (UTR) of an Indonesian blotch strain of PSTV showed resistance to PSTV infection, and the resistance was stably inherited over at least five generations (Dietzgen et al., 2004). However, these genetically engineered plants are yet to be commercialized.

2.2.7 Yellow mosaic
Yellow mosaic on groundnut, caused by CMV, was first reported from Northern parts of China (Xu & Barnett, 1984) and later from Argentina (De Breuil, Giolitti, & Lenardon, 2005). Crop losses up to 40% were reported from China, and the symptoms caused by CMV are described by Xu and Barnett (1984). Of the two strains of CMV reported to naturally infect groundnut in China, CMV-CA was the predominant strain and CMV-CS was of minor importance. Bioassays on cowpea, cucurbits, tobacco, Datura stramonium, and Chenopodium species were useful for the initial diagnosis of CMV isolates (Palukaitis & García-Arenal, 2003). ELISA, dot-blot hybridization, and RT-PCR were optimized for routine detection of the virus (Dietzgen et al., 2001; Palukaitis & García-Arenal, 2003). The CMV-CA isolate was seed transmissible (2–4%) in groundnut. Therefore, the primary spread is presumably initiated through the seed-borne inoculum, whereas the aphids may contribute to its secondary spread. Planting with CMV-free groundnut seed reduced disease incidence. Cultural practices like mulching with transparent plastic sheets and rouging of diseased seedlings at early stages of crop growth reduced disease incidence in China (Reddy & Thirumala-Devi, 2003). No resistance to CMV in the cultivated groundnut was reported.

2.3. Common bean
Common bean is an important grain legume crop cultivated in Myanmar, India, Brazil, China, the United States, Mexico, Tanzania, Kenya, and Rwanda (FAOSTAT, 2012). Among the large number of viruses infecting common bean, diseases caused by certain species of Potyvirus, Begomovirus, and Cucumovirus genera were considered to be economically important (Table 9.1). The symptoms caused by these viruses on common bean were described by Morales (2003) and Schwartz, Steadman, Hall, and Foster (2005).

2.3.1 Common mosaic and black root
BCMV and Bean common mosaic necrosis virus (BCMNV) cause common mosaic and black root diseases, respectively. BCMV predominates in the Western hemisphere, whereas BCMNV occurs in Eastern Africa, Dominican
Republic, and Haiti. They cause significant yield losses that can reach up to 80% (Morales, 2003). Substantial yield losses were reported in mixed infections with BCMV and Bean rugose mosaic virus (BRMV) (Castillo-Urquiza, Maia, Carvalho, Pinto, & Zerbini, 2006).

Several BCMV isolates have been distinguished on the basis of symptoms produced on P. vulgaris cultivars. BCMV and BCMNV strains cause two main types of symptoms on beans: “common mosaic” characterized by green vein banding and leaf malformation and “black root” characterized by systemic necrosis and plant death (Morales, 2003; Schwartz et al., 2005). BCMV and BCMNV strains, occurring on lima beans from Peru, were characterized using differential bean varieties and RT-PCR (Melgarejo, Lehtonen, Fribourg, Rannali, & Valkonen, 2007).

BCMV is seed-transmitted up to 83% in P. vulgaris and 7–22% in Tapari bean plants. The necrosis-inducing strains of BCMV and BCMNV are seed and pollen transmitted in common bean genotypes that lack the dominant I gene. Seed-borne inoculum contributes to primary spread, and secondary spread occurs through aphid vectors which transmit both viruses in a non-persistent manner. The planting of dominant I gene and recessive I+ common bean cultivars side by side had resulted in major epidemics of black root because of the availability of seed-transmitted inoculum and the existence of several aphid species that can transmit these viruses (Morales, 2003).

Losses due to BCMV and BCMNV may be in principle curtailed by planting with certified seeds, control of aphid vectors by oil application, timely sowing of crops, use of optimum plant densities, and intercropping with maize. However, growing resistant cultivars was considered to be the best option for reducing crop losses by these viruses (Morales, 2003). Symptom severity could be reduced in French bean through seed treatment with Sanosil (a commercial formulation containing hydrogen peroxide and silver which can mask the expression of BCMV symptoms) and Pseudomonos fluorescens (plant growth-promoting bacterium) (Bhuvanendra Kumar, Uday Shankar, Prakash, & Shekar Shetty, 2005). Treatment with 15 mA electricity for 15 min resulted in substantial reduction of transmission of BCMV through bean seed (Hormozi-Nejad, Mozafari, & Rakhshandehroo, 2010).

The occurrence of numerous BCMV strains has important implications for the development of resistant cultivars. In the New World and Europe, where mosaic-inducing strains of BCMV are mostly prevalent, cultivars with the “I” gene provided effective protection against the virus for more than 50 years (Morales, 2003). Since this gene also prevents seed transmission, it has provided valuable means of eliminating quarantine risk. Gene
combinations of \(bc-u\) (strain-nonspecific epistatic gene) plus any of the \(bc-1\), \(bc-I^2\), \(bc-2\), or \(bc-2^2\) genes confer recessive resistance. The combination of genes \(bc-u\), \(bc-2^2\), \(bc-3\), and \(I^\prime\) gives durable resistance to all the known strains of BCMV. Nonetheless, it has been shown to be a considerable breeding task to incorporate these genes into beans (Morales, 2003; Schwartz et al., 2005). A number of BCMV/BCMNV resistance genes have been tagged including the dominant \(I^\prime\) gene and the recessive \(bc-3\), \(bc-2\), and \(bc-1^2\) genes. These genes can be distinguished by inoculation with different virus isolates and by a range of molecular marker tags that are available for each gene (reviewed by Blair, Beaver, Nin, Prophete, & Singh, 2006; Kelly, Gepts, Miklas, & Coyne, 2003; Miklas, Kelly, Beebe, & Blair, 2006). The genetic and molecular characterization of the \(I^\prime\) locus of the \(P. vulgaris\) was studied (Eduardo Vallejos et al., 2006). In India, the presence of \(I^\prime\) gene was confirmed in some of the bean accessions of diverse origin when they were evaluated for resistance against BCMV strains and for inheritance patterns in selected cultivars against the strain NL-1 (Sharma et al., 2008). In Spain, the introgression and pyramiding of genes conferring genetic resistance to BCMV and anthracnose local races into breeding lines A25 and A3308 was reported (Ferreira, Campa, Perez-Vega, Rodriguez-Suarez, & Giraldez, 2012). The prospects of MAS for common bean diseases including BCMV and BCMNV were reviewed (Tryphone et al., 2013). Resistance to fungal angular leaf spot and BCMNV diseases was incorporated into adapted common bean genotype in Tanzania using molecular markers (Chilangane et al., 2013).

2.3.2 Golden mosaic, golden yellow mosaic, and dwarf mosaic

Several begomoviruses, viz., BGMV, Bean golden yellow mosaic virus (BGYMV), Bean dwarf mosaic virus (BDMV), Bean summer death virus, Bean yellow dwarf virus, and Bean calico mosaic virus (BCaMV) have been reported to naturally infect common bean in the tropical countries (Morales, 2006; Navas-Castillo et al., 2011; Schwartz et al., 2005). Later, natural occurrence of several other begomoviruses on common bean has been reported, and their economic significance is yet to be determined (e.g., Fiallo-Olive et al., 2013; Jyothi, Nagaraju, Padmaja, & Rangaswamy, 2013; Kamaal, Akram, Pratap, & Yadav, 2013; Shahid, Ikegami, & Natsuaki, 2012; Venkataravanappa et al., 2012). These examples indicate the emergence of new begomoviruses capable of infecting common bean.

Symptoms of golden yellow mosaic resemble BGMV infection and hence were considered to be caused by the same virus. However, advances
in molecular techniques have revealed that the golden mosaic and golden YMDs are caused by related but distinct begomovirus species, BGMV and BGYMV, respectively (Morales, 2003).

Studies in Brazil on the begomoviruses infecting legume crops and weeds revealed that the recombination of viral genomes in weeds such as *Macroptilium lathyroides* (Rodriguez-Pardina et al., 2006; Silva et al., 2011) has contributed to their rapid evolution. DNA hybridization and PCR-based methods were applied for specific detection of BGMV, BGYMV, BCaMV, and BDMV in field-collected bean samples (Karkashian, Ramos-Reynoso, Maxwell, & Ramirez, 2011; Potter, Nakhla, Mejia, & Maxwell, 2003). Field spread of these viruses is by *Bemisia tabaci* in a persistent manner. Female whiteflies were found to be more efficient vectors of BGMV than males. The epidemics of these viruses depended on the presence of suitable whitely reproductive hosts such as tomato (*Solanum lycopersicum*), eggplant (*Solanum melongina*), soybean, and tobacco (*Nicotiana tabacum*). Additionally, the development of pesticide-resistant whitefly populations reported in different countries has contributed to the spread of the viruses they transmit (Morales, 2003, 2006).

The incidence of BGMV, BGYMV, and BDMV decreases with increasing distance from preferred hosts of the vector. There are also opportunities for reducing severity of diseases by manipulating sowing time (during the rainy season) so as to escape peak infestation by *B. tabaci*. In the Dominican Republic, planting in November significantly reduced the disease. Also, seed treatment and spraying with systemic insecticides like carbofuran or aldicarb in combination with mineral oil were found to reduce begomovirus incidence (Morales, 2003).

Resistance to BGMV was reported in black-seeded common bean cultivars in South America. *P. vulgaris* accessions possessing partial resistance or tolerance to BGMV include Porrillo Sintetico, Porrillo 70, Turrialba 1, and ICA-Pijao. They have been used successfully in breeding black-seeded cultivars such as ICTA Quetzal and Negro Huastoco in Guatemala and Mexico, respectively. Additional sources of resistance were identified in 188 accessions selected from a set of 1660 accessions of bean germplasm. Sources of resistance to BGYMV have been identified in black-seeded Mesoamerican genotypes, which are at best tolerant and have the ability to escape infection under field conditions. Other BGYMV resistance genes discovered are *bgm* or *bgm-1* in bean genotypes of Mexican “Durango” race or the *bgm-2* of Andean origin (Morales, 2003). These genes were successfully used in the development of BGYMV-resistant cultivars. However, the
presence of these genes resulted in deformed pods that could be avoided through incorporation of \textit{Bgp-1} gene that contributed to normal pod development (Roman, Castaneda, Sanchez, Munoz, & Beaver, 2004). Blair, Rodriguez, Pedraza, Morales, and Beebe (2007) mapped \textit{bgm-1} conferring resistance to BGYMV and linkage with \textit{bc-1} conferring strain-specific resistance to BCMV. Resistant cultivars to BDMV infection include Porillo Sintetico, DOR 41, Red Mexican UI 35, and pinto UI 114. Furthermore, the sources of resistance to BGMV were also effective against BDMV. A single dominant gene, \textit{Bdm}, conferred BDMV resistance in crosses between Othello and Topcrop bean cultivars (Seo, Gepts, & Gilbertson, 2004).

Scientists at the Brazilian Agricultural Research organization (EMBRAPA) produced genetically engineered bean cv. Olathe with a hairpin (hp) construct containing the Rep gene from BGMV. Transformed plants showed resistance to BGMV even under mixed infection with BCMNV and BRMV (Bonfim, Faria, Nogueira, Mendes, & Aragao, 2007). Two lines that showed high degree of resistance to BGMV in field trials under high vector pressure were identified. Further evaluations under field conditions for agronomic traits have been carried out in three regions of Brazil. The results confirmed superior performance of engineered plants in multilocation tests (Aragao & Faria, 2009). This engineered bean has received approval from Brazilian National Technical Commission on Biosafety, thus became the first deregulated native crop cultivar in Latin America (Tollefson, 2011). It is expected to receive wide acceptance from farmers in Brazil. Specific PCR-based test was developed for the detection of engineered BGMV-resistant common bean Embrapa 5.1 in Brazil (Dinon et al., 2012). Such tests could be useful to assess the performance of this crop.

### 2.3.3 Mosaic due to CMV

Several strains of CMV occurring worldwide can induce different symptoms in common bean ranging from mild mosaic to severe plant malformation and yield losses varying from 5% to 75% depending on the cultivar, age of infection, virus strain, and environmental conditions (Morales, 2003; Schwartz et al., 2005). ELISAs, dot-immunobinding assays (DIBAs), rapid immunofilter assay, and IC-RT-PCR were used for distinguishing CMV isolates (Palukaitis & Garcia-Arenal, 2003; Zein, Nakazawa, Ueda, & Miyatake, 2007). At least six bean infecting strains of CMV are seed borne (up to 10%) in beans and thus can be disseminated to long distances in seed shipments. Both seed and aphid transmission of CMV may be erratic and influenced by several factors (Schwartz et al., 2005).
In Chile, Turkey, and Iran, legume crop losses are caused by CMV warrant application of specific management measures. Use of virus-free bean seeds is probably the least costly management measure. It may also be useful to destroy reservoir hosts or to isolate the crop from such hosts. Management of aphid vector populations in crops and inoculum reservoir hosts may reduce the natural spread of CMV. This can be achieved by planting barrier crops that are immune to CMV infection, applying sticky traps, and mulching with aluminum foil. Developing new bean crop varieties resistant to CMV either by conventional breeding methods or by genetic engineering is gaining momentum (Makkouk et al., 2012). Several species of the genus Phaseolus such as P. acutifolius, P. adenanthus, P. leptostachyus, P. palyanthus, P. trilobus, and some accessions of P. coccineus are resistant to CMV. However, the resistance genes are yet to be transferred to P. vulgaris (Schwartz et al., 2005). The different strategies of PDR in developing transgenic plants resistant to CMV infection are reviewed by Morroni, Thompson, and Tepfer (2008) and need to be utilized to develop CMV-resistant common bean. Genes involved in resistance response in common bean cv. Othello were identified by inoculating the geminivirus (BDMV) reporter in transgenic Nicotiana benthamiana. The identified RT4-4 gene did not confer resistance to the reported geminivirus, but it activated a resistance related response (systemic necrosis) to seven strains of CMV from tomato and pepper but not to a strain from common bean (Seo et al., 2006).

2.4. Cowpea

Cowpea is the most widely cultivated indigenous legume in SSA, especially by smallholder farmers, because of its tolerance to drought and ability to thrive in poor soils. Over 80% of cowpea production is confined to West Africa with Niger, Nigeria and Burkina Faso alone contributing 83% of the production area and 77% of the total production (FAOSTAT, 2012). It is the second most important food legume after groundnut (Boukar, Bhattacharjee, Farokun, Kumar, & Gueye, 2013). Cowpea is susceptible to over 140 viruses and about 20 of these viruses are known to have widespread distribution (reviewed in Hampton & Thottappilly, 2003). They include BCMV–blackeye cowpea mosaic strain (BCMV–BlCM), Coupea aphid-borne mosaic virus (CABMV), Coupea chlorotic mottle virus (CCMV), CMV, Coupea golden mosaic virus (CGMV), Coupea mottle virus (CPMoV), CPMV, CPSMV, and Southern bean mosaic virus (SBMV; Table 9.1). BCMV–BlCM, CABMV, and CMV were detected in the majority of cowpea producing countries.
The viruses infecting cowpea are vectored by aphids (BCMV-BlCM, CABMV, and CMV), by beetles (CCMV, CPMV, CPSMV, CPMoV, and SBMV), or by whiteflies (CPMMV and CGMV). With the exception of CGMV and CCMV, all the other viruses are known to be seed-transmitted at a variable rate between none to 55% depending on virus strain, cowpea genotype, and time of infection (Bashir & Hampton, 1996; Salem, Ehlers, Roberts, & Ng, 2010).

Establishment of cowpea infection is dependent on the volunteer plants including cultivated and noncultivated species, insect vector density, and cultivar susceptibility. Therefore, measures targeting vector control, cultural management to eliminate volunteer sources including use of virus-free seed stocks (Biemond et al., 2013; Sharma & Varma, 1984) and resistant cultivars (reviewed in Hampton & Thottappilly, 2003) can significantly reduce the incidence, spread, and damage to cowpea crops. However, insect control measures by using pesticides and cultural measures other than growing virus-free seed is seldom feasible for smallholder farmers in SSA.

Protocols for screening germplasm for resistance by mechanical inoculation and/or natural infection facilitated by vectors under field conditions have been reported (Gillaspie, 2007; Goenaga, Quiles, & Gillaspie, 2008). Additionally, serological and nucleic acid-based diagnostic tools (Amayo et al., 2012; El-Kewey, Sidaros, Abdelkader, Emeran, & EL-Sharkawy, 2007; Naimuddin, 2010; Ojuederie, Odu, & Ilori, 2009; Roy & Malathi, 2004; Salem et al., 2010) have contributed to the sensitive detection and rapid evaluation of cowpea germplasm, leading to identification of acceptable levels of resistance by a number of research groups around the world (reviewed in Hampton & Thottappilly, 2003). However, these intensive efforts are limited to a few viruses, viz., BCMV-BlCM, CABMV, CMV, and CGMV. In the majority of cases, resistance identified is not immunity but tolerance. Cowpea genetics and list of resistant genes utilized for developing cultivars with resistance to one or more cowpea viruses are reviewed (Hampton & Thottappilly, 2003). At IITA, research work is being continued to identify durable resistant cowpea varieties and also to determine the genetic determinants of virus resistance in cowpea germplasm. Resistance to two potyviruses was found in germplasm accessions, TVu401, TVu1453, and TVu1948, and in breeding lines, IT82D-885, IT28D-889, and IT82E–60 (Gumedzoe, Rossel, Thottappilly, Asselin, & Huguenot, 1998). However, resistance to multiple virus infection is scarce in cowpea, and recent studies are putting greater emphasis on multiple virus resistance. These efforts have already resulted in identification of multiple virus resistance to three virus species
(BCMVC-BlCM, CMV, and SBMV) in breeding lines, IT98K-1092-1 and IT97K-1042-3 (Ogunsola, Fatokun, Boukar, Ilori, & Kumar, 2010). RILs are being established for mapping studies to identify DNA markers linked to multiple virus resistance. For instance, resistance to CGMV in crosses between IT97K-499-25 × Canapu T16 was shown to be controlled by a single dominant gene (Rodrigues, Santos, & Santana, 2012). CABMV resistance was conditioned by a single recessive gene or more than one recessive gene (Orawu, Melis, Laing, & Derera, 2013). Resistance in cowpea to BCMV-BlCM was attributed to a single recessive gene pair (Arshad, Bashir, Sharif, & Malik, 1998). Very limited efforts have focused on breeding for vector resistance. Recently at IITA, 92 wild cowpea accessions were evaluated in a greenhouse for resistance to cowpea aphid, A. craccivora, which is a prominent pest and a vector for at least 11 viruses in SSA (Souleymane, Aken’Ova, Fatokun, & Alabi, 2013). This resulted in the identification of only TVNu1158 as the best aphid-resistant accession for incorporation into cultivated cowpea, while other accessions were found to be susceptible to aphid infestation and severe damage (Souleymane et al., 2013).

Progress in the development of varieties with superior performance depends on the availability of germplasm with desired traits. IITA conserves over 14,000 accessions of cultivated cowpea in its gene bank, and the USDA has duplicates of most of the IITA cowpea lines for safe keeping (Boukar et al., 2013; Dumet et al., 2012). Besides cultivated cowpea lines, some accessions of wild cowpea relatives are also conserved in the gene bank at IITA. However, there are not many reports in literature on the use of wild cowpea relatives for the genetic improvement of cultivated varieties for pest and disease resistance (reviewed in Boukar et al., 2013).

Application of DNA marker technologies for cowpea improvement has been very slow when compared to many other crops. However, recent advances in molecular biological techniques, genomics, and bioinformatics have been opening new vistas for molecular breeding in cowpea (Boukar et al., 2013; Diouf, 2011; Ehlers et al., 2012). In addition, SNP genotyping platforms, high-density consensus genetic map with more than 1000 markers, and QTL(s) linked to important biotic and abiotic resistance traits including resistance to foliar thrips, Fusarium wilt (FW), root-knot nematode, bacterial blight, ashy stem blight, and Striga have been established (Ehlers et al., 2012). Similar genomic resources are required to fast track development of virus-resistant cowpea.

High levels of resistance to several cowpea viruses, especially multiple virus infections are limited in cowpea germplasm. Efficient and stable
transformation of cowpea and stable transmission of transgenes to progeny have been a major bottleneck for the development of cowpea transgenics. Good protocols for Agrobacterium-mediated transformation (Chaudhury et al., 2007; Popelka, Gollasch, Moore, Molvig, & Higgins, 2006) and also biolistic transformation (Ivo, Nascimento, Vieira, Campos, & Aragao, 2008) of cowpea have been developed and showed stable inheritance of the transgenes. Cowpea transformed with Bt gene (Popelka et al., 2006) is under field tests in Nigeria, Burkina, and Ghana. Similar transformation techniques combined with transgenically induced PTGS (posttranscriptional gene silencing) or RNAi can be exploited for the control of both DNA and RNA viruses infecting cowpea. The concept of using RNAi construct to silence the CPSMV proteinase cofactor gene and CABMV-CP gene is explored in transgenic cowpea. In the symptomless resistant lines, the resistance was homozygosis dependent. Only homozygous plants remained uninfected while heterozygous plants presented relatively mild symptoms (Cruz & Aragao, 2014). In Arlington line, cowpea extreme resistance to CPMV is controlled by a dominant locus designated Cpa. Using the transgenic approach and mutational analysis of the 24 k Pro gene, Fan, Niroula, Fildstein, and Bruening (2011) demonstrated the participation of protease in eliciting extreme resistance. These recent developments in cowpea transformation form a basis for strong cowpea genetic improvement program to enhance cowpea productivity.

2.5. Pigeonpea

Pigeonpea is an important grain legume crop predominately grown in the Indian subcontinent, covering an area of ~3.53 million hectares in India alone. It is also grown in southern and eastern Africa, the Caribbean and China. Fifteen viruses are known to naturally infect pigeonpea (Kumar, Kumari, et al., 2008). Of these, diseases caused by the PPSMV and very recently whitefly-transmitted bipartitate begomoviruses have been shown to be economically important (Table 9.1) (Jones et al., 2004; Kumar, Jones, & Reddy, 2003; Reddy, Raju, & Lenne, 1998).

2.5.1 Sterility mosaic

Sterility mosaic disease (SMD) is a serious constraint for pigeonpea cultivation in India, Bangladesh, Nepal, Thailand, Myanmar, and Sri Lanka with an estimated annual loss of over US$ 300 million in India alone (Kumar et al., 2007). SMD is characterized by partial or complete cessation of flower production (sterility), excessive vegetative growth, stunting, chlorotic ring spots...
or mosaics on the leaves, and reduction in the size of leaves. The yield losses due to SMD depend on at what stage the crop is infected; early infection (<45 days) can lead to a yield loss from 95% to 100%, whereas late infections can lead to 26–97% yield losses. In addition, infection from SMD predisposes the plants to subsequent infection by fungal diseases and spider mites attack (Kumar et al., 2003).

SMD was shown to be caused by PPSMV, with a negative-strand segmented RNA genome, classified under the newly created genus, *Emaravirus* (Kumar et al., 2003; Mielke-Ehret & Mühlbach, 2012). PPSMV is transmitted by the eriophyid mite *Aceria cajani* in a semipersistent manner (Kulkarni et al., 2003). Recently, an isolate of PPSMV from ICRISAT–Patancheru (India) was fully sequenced and was shown to contain five segments of RNA (Elbeaino, Digiaro, Uppala, & Sudini, 2014). The PPSMV isolates collected from SMD-affected plants in Coimbatore (C), Bangalore (B), Dharwad (D), Gulbarga (G), Varanasi (V) from India, and Nepalgunj (N) from Nepal have shown differences in virulence and biochemical properties (Kumar, Jones, & Waliyar, 2004, Kumar, Kumari, et al., 2008; Kumar, Latha, Kulkarni, Raghavendra, & Saxena, 2005). DAS–ELISA, DIBA, and PCR have been developed to detect PPSMV (Kumar et al., 2003, 2007). Very recently, degenerate PCR primers for amplification of partial RdRp sequences of emaraviruses have been shown to detect PPSMV isolates (Elbeaino, Whitefield, Sharma, & Digiaro, 2013).

Natural occurrence of PPSMV and its vector has been recorded only on cultivated and wild pigeonpea. PPSMV is not known to be seed-transmitted. Seed treatment or soil and foliar application of a number of organophosphorous–based insecticides or acaricides (carbofuran and aldicarb for soil application, and oxythioquinox, kethane, dinocap, monocrotophos, tedion, and metasystox as foliar sprays) recommended for the management of the vector mites is seldom practiced by farmers due to high cost and environmental issues (Ghanekar, Sheila, Beniwal, Reddy, & Nene, 1992; Rathi, 1997). Destruction or rouging of SMD–infected stubbles, ratoon, weeds, or wild species of pigeonpea that support PPSMV and *A. cajani* multiplication are effective in the eradication of SMD inoculum, but rarely practiced under small holder subsistence farming systems (Reddy, Sharma, & Nene, 1990). Thus, major emphasis has been placed on the development of SMD–resistant cultivars and hybrids as the most effective and realistic approach to reduce the crop losses.

Sources of SMD resistance have been identified in the pigeonpea germplasm collection at ICRISAT (Ghanekar et al., 1992; Nene, 1995).
However, the occurrence of distinct strains/isolates of PPSMV in different locations makes it difficult to incorporate broad-spectrum resistance. Resistance to diverse isolates of PPSMV has been reported in very few cultivars such as ICP7035, and it has been released for cultivation (Rangaswamy et al., 2005). Wild Cajanus species were shown to have resistance to multiple isolates of PPSMV.

Screening for resistance to three PPSMV isolates from South India was conducted for 115 wild Cajanus accessions belonging to six species, C. albicans, C. platycarpus, C. cajanifolius, C. lineatus, C. scarabaeoides, and C. sericeus. Accessions, ICP 15614, 15615, 15626, 15684, 15688, 15700, 15701, 15725, 15734, 15736, 15737, 15740, 15924, 15925, and 15926 showed resistance to all the three isolates (Waliyar, 2005). These accessions are cross-compatible with cultivated pigeonpea. As a result, it should be possible to incorporate this resistance through conventional breeding. Most of the wild species did not support multiplication of mites, and the majority of the accessions inoculated with viruliferous mites were resistant to PPSMV, but were susceptible by graft inoculation, suggesting that the observed resistance was vector mediated. Some of the wild species of pigeonpea resistant to infestation by mites have a thicker leaf cuticle and epidermal cell wall, which hinders mite’s stylet penetration into the leaf epidermal cells (Reddy, Sheila, Murthy, & Padma, 1995). The wild species, C. scarabaeoides (ICPW 94), which is resistant to all the isolates of PPSMV, was used in the crossing program, and the progeny was tested for SMD resistance, resulting in both resistant and moderately resistant plants. Hybrid lines derived from interspecific crosses involving C. acutifolius and C. platycarpus have shown resistance to Patancheru isolate of PPSMV under field conditions (Mallikarjuna et al., 2011). Recently, new sources of resistance to FW and SMD were identified in a minicore collection of pigeonpea germplasm at ICRISAT (Sharma, Rameshwar, & Pande, 2013; Sharma et al., 2012). High level of resistance to SMD was found in 24 accessions, which originated from India, Italy, Kenya, Nepal, Nigeria, the Philippines, and the United Kingdom. Combined resistance to FW and SMD was found in five accessions of pigeonpea (ICP 6739, 8860, 11015, 13304, and 14819), and these diverse accessions should be utilized in the breeding programs.

Studies on the inheritance of SMD resistance trait in various cultivars of pigeonpea against several isolates of PPSMV have led to different interpretations on the genetics of inheritance of SMD. Studies by Srinivas, Reddy, Jain, and Reddy (1997) showed that the resistance to SMD was dominant in the two crosses (ICP 7035 × ICP 8863 and ICP 7349 × ICP 8863), and
recessive in another cross (ICP 8850 × ICP 8863). The disease reaction for the Patancheru isolate of PPSMV appeared to be governed by a single gene with three alleles, with one resistance allele exhibiting dominance, and the other being recessive over the allele for susceptibility. However, monogenic inheritance of SMD resistance was noticed in the cross ICP 8850 × ICP 8863. The nature of inheritance of SMD resistance in two crosses involving resistant and susceptible pigeonpea cultivars revealed that the resistance is controlled by recessive gene and appeared to be monogenetic in one cross (TTB 7 × BRG 3) and governed by two independent nonallelic genes exhibiting complimentary epistasis in another cross (ICP 8863 × ICP 7035) (Ganapathy et al., 2012).

Ganapathy et al. (2009) generated two AFLP primer pairs, comprising four markers, polymorphic for SMD-resistant and -susceptible bulks of the F2 population derived from TTB7 (susceptible) and BRG3 (resistant) parents. After screening over 3000 SSR markers on parental genotypes of each mapping population, intraspecific genetic maps comprising of 11 linkage groups and 120 and 78 SSR loci were developed for ICP 8863 × ICPL 20097 and TTB 7 × ICP 7035 populations, respectively (Gnanesh, Bohra, et al., 2011). The genotypes with high polymorphism as revealed by SSR markers were identified, and they were recommended for developing mapping populations (Naik et al., 2012). Composite interval mapping-based QTL analysis by using genetic mapping and phenotyping data provided four QTLs for Patancheru PPSMV isolate and two QTLs for Bangalore PPSMV isolate (Gnanesh, Ganapathy, Ajay, & Byre, 2011). Identification of different QTLs for resistance to Patancheru and Bangalore SMD isolates is an indication of involvement of different genes conferring the resistance to these two isolates (Gnanesh, Ganapathy, et al., 2011).

Complete sequence of pigeonpea genotype ICPL 87119 genome, an inbred line tolerant to SMD, popularly known as Asha was published (Varshney et al., 2012). Hence, it should be possible in the near future to identify the R genes, NBS-LRR, which contains a nucleotide-binding site and a leucine-rich repeat, involved in conferring resistance to PPSMV, using modern genomics and bioinformatics-based approaches such as next-generation sequencing and microarrays.

2.5.2 Yellow mosaic
YMD of pigeonpea, caused by whitefly-transmitted begomoviruses, occurs in Sri Lanka, India, Jamaica, Nepal, and Puerto Rico (Reddy et al., 2012). Although the incidence of YMD in pigeonpea is low, the late sown
pigeonpea can show higher incidence resulting in a yield loss up to 40% (Beniwal, Deena, & Nene, 1983). Various begomovirus species that include MYMV, Rhynchosia mosaic virus, and Tomato leaf curl New Delhi virus have shown to be associated with YMD (Biswas & Varma, 2000).

2.6. Mungbean and urdbean

Mungbean is a nutritious grain legume crop containing 23.6% of easily digestible protein and 51% carbohydrates. It is cultivated in China, Thailand, the Philippines, Vietnam, Indonesia, Myanmar, Bangladesh, India, and in the hot and dry regions of Southern Europe and Southern United States (Nair et al., 2013). Urdbean is widely cultivated in India, Myanmar, Thailand, the Philippines, and Pakistan. Among several viruses reported to naturally infect these two pulse crops (Biswas, Tarafdar, & Biswas, 2012; Makkouk, Kumari, Huges, Muniyappa, & Kulkarni, 2003), yellow mosaic caused by begomoviruses, leaf curl caused by PBNV, and leaf crinkle caused by Urdbean leaf crinkle virus (ULCV) are considered to be economically important (Table 9.1) (Biswas et al., 2012; Malathi & John, 2008; Mandal et al., 2012; Qazi et al., 2007). Mixed infections by PBNV, MYMIV, and ULCV are common in both the crops in India and are often synergistic resulting in crop losses exceeding 90% (Biswas et al., 2012).

2.6.1 Yellow mosaic

Three begomoviruses, MYMV, MYMIV, and HgYMV, were shown to cause mungbean yellow mosaic disease (MYMD) symptoms (Islam, Sony, & Borna, 2012; Malathi & John, 2008). MYMIV occurs in Northern India, Pakistan, Nepal, Bangladesh, and Indonesia, while MYMV is confined to Thailand, Vietnam, and Peninsular region of India (Ilyas, Qazi, Mansoor, & Briddon, 2010; Islam et al., 2012; Shahid et al., 2012; Tsai et al., 2013). HgYMV occurs only in South India (Borah & Dasgupta, 2012; Varma & Malathi, 2003). Enzyme immunoassays, ISEM, nucleic acid hybridization, and PCR-based tests were employed for the detection of begomoviruses associated with YMD of mungbean and urdbean (Malathi & John, 2008). Hyperspectral remote sensing of yellow mosaic severity and associated chlorophyll losses in MYMD affected urdbean was analyzed and developed logistic regression models with spectral ratios for disease assessment (Prabhakar et al., 2013).

Since MYMV is not seed-transmitted, primary source of inoculum is contributed by numerous alternate hosts of the virus and the whitefly vector. Management approaches for MYMD have been reviewed (Malathi & John,
2008). Usually farmers do not use pesticides to control the vector. However, seed treatment and spraying with imidacloprid contributed to relatively low disease incidence (Malathi & John, 2008; Sethuraman, Manivannan, & Natarajan, 2001). Marigold as a trap crop along with yellow sticky traps (8/ha) recorded reduced disease incidence (Salam, Patil, & Byadgi, 2009). Integrated management of major mungbean diseases (wet root rot, leaf spots, and yellow mosaic due to MYMV) by using different combinations of an insecticide, fungicide, and bioformulation as seed treatment, with or without foliar sprays was recently reported (Dubey & Singh, 2013). Such studies should always include their cost-effectiveness.

Of over 10,000 mungbean germplasm lines screened for resistance to MYMV from 1977 to 2003, in Punjab, India, 31 germplasm lines were found to be resistant (Singh, Sharma, Shanmugasundaram, Shih, & Green, 2003). Subsequently, several reports have been published on the identification of germplasm lines/cultivars exhibiting varied levels of resistance to local isolates of causal viruses (e.g., Ahmad et al., 2013; Akthar et al., 2011; Habib, Shad, Javaid, & Iqbal, 2007; Paul, Biswas, Mandal, & Pal, 2013; Shad, Mughal, Farooq, & Bashir, 2006). Several mungbean and urdbean MYMV-resistant germplasm lines were identified in multilocation tests (Biswas et al., 2012; Malathi & John, 2008; Mondol, Rahman, Rashid, Hossain, & Islam, 2013). The inheritance of MYMV resistance in mungbean has been reported to be conferred by a single recessive gene (Reddy, 2009), a dominant gene (Sandhu, Brar, Sandhu, & Verma, 1985), two recessive genes (Ammavasai, Phogat, & Solanki, 2004), and complimentary recessive genes (Shukla & Pandya, 1985). Use of different source of MYMV resistance for genetic studies and infection by different strains of the same virus or distinct begomovirus species may have led to these conflicting results. A report on the genetic analysis of resistance to MYMV suggested that the resistance is governed by two recessive genes (Dhole & Reddy, 2012). Due to the divergence in begomoviruses, these studies need to be supported with precise virus identification.

Genetic markers that will aid in selecting MYMD-resistant breeding lines have been identified. A SCAR marker linked to MYMV resistance gene was identified with a distance of 6.8 cm (Souframanien & Gopalakrishna, 2006). Two marker loci, YR4 and CYR1 (CYR1 was completely linked with MYMIV resistance), were employed in multiplex PCR reaction to screen quickly and reliably urdbean germplasm and breeding lines for resistance to MYMV (Maiti, Basak, Kundagrami, Kundu, & Pal, 2011). A single dominant gene was shown to govern resistance in a cross that
involved mungbean genotypes DPU 88-31 (resistant) × AKU 9904 (susceptible). Resistance genes were mapped using SSR markers. Out of 361 markers, 31 were found to be polymorphic between the parents (Gupta, Gupta, Anjum, Pratap, & Kumar, 2013). Markers CEDG 180, mapped at a distance of 12.9 cm, were found to be linked with resistance gene in analyses on bulked segregants. Mapping of QTL for MYMIV resistance in mungbean using an F₈ RIL generated in Thailand from a cross between NM10-12-1 (MYMIV resistance) and KPS2 (MYMIV susceptible) was done and field evaluated in India and Pakistan (Kitsanachandee et al., 2013).

Agroinoculation, using dimeric constructs of DNA-A and DNA-B, of sprouted seeds and seedling was shown to be a useful tool for screening crop germplasm for virus resistance. This technique was applied for screening of mungbean progenies (F₂) derived from a cross between Vamban (Gg) 2 (susceptible) × KMG 189 (MYMV resistant) (Karthikeyan et al., 2011). Later, a similar approach was used for screening of 78 mungbean germplasm lines for resistance to MYMV (Sudha et al., 2013).

In order to develop genetically engineered MYMIV-resistant mungbean genotypes, MYMV-Vig CP, replication-associated protein (Rep-sense, Rep-antisense, truncated Rep, nuclear shuttle protein, and movement protein) genes were agroinoculated with partial dimers of MYMV-Vig and analyzed for viral DNA accumulation. Both mungbean and tobacco model systems have shown that engineered plants containing replicase gene and AC4 hp RNA gene showed resistance to MYMV (Haq, Ali, & Malathi, 2010; Shivaprasad, Thillaichidambaram, Balaji, & Veluthambi, 2006; Sunitha, Shanmugapriya, Balamani, & Veluthambi, 2013). None of the laboratory-generated resistant plants has been tested under contained field trials, and hence, the deregulation of genetically engineered MYMIV-resistant mungbean and urdbean is less likely to happen in the near future.

2.6.2 Leaf curl

Leaf curl caused by PBNV is widespread on mungbean and urdbean in the Indian subcontinent. Symptoms of the disease include necrosis of terminal bud, leaves, petioles, stems, and pods. Early infection results in crop losses of up to 90% (Biswas et al., 2012). Based on N gene sequence analysis, PBNV strains were classified into eight different evolutionary clusters irrespective of their geographical origin or host (Mandal et al., 2012). Sowing of mungbean during second half of May to the first half of June in summer, late sowing in spring and intercropping with pearl millet (at 2:1 ratio) resulted in relatively low disease incidence. Among the various insecticides (imidacloprid,
thiamethoxam, acetamiprid, fipronil, dimethoate, fenvalerate, and azadirachtin), imidachloprid gave the most satisfactory control of *T. palmi* and low PBNV incidence in mungbean (Sreekanth, Sriramulu, Rao, Babu, & Babu, 2004a, 2004b). Of 39 mungbean genotypes screened for resistance to PBNV under field conditions, accessions LGG 460, 480, 491, and 582 consistently showed lower disease incidence than the susceptible genotypes (Sreekanth, Sriramulu, Rao, Babu, & Babu, 2002).

### 2.6.3 Leaf crinkle

Leaf crinkle disease on urdbean is widely distributed in India and Pakistan and reported to cause crop losses up to 100% (Biswas et al., 2012; Reddy, Tonapi, Navi, & Jayaram, 2005). Based on the symptoms and transmission, the causal agent of this disease is considered to be a virus; however, there are no reports of its isolation or characterization.

### 2.7. Chickpea

Chickpea is the third most important pulse crop. It is extensively grown in India, Australia, Turkey, Myanmar, Ethiopia, Iran, Canada, the United States, Pakistan, and Tanzania (FAOSTAT, 2012). There are two distinct types of cultivated chickpea: *desi* and *kabuli*. Several viruses have been reported to naturally infect chickpea in different parts of the world (Abraham, Menzel, Lesemann, Varrelmann, & Vetten, 2006, Abraham, Menzel, Varrelmann, & Vetten, 2009; Kanakala, Sakhare, Verma, & Malathi, 2012, Kanakala, Verma, Vijay, Saxena, & Malathi, 2013; Kumar, Jones, et al., 2008; Mumtaz, Kumari, Mansoor, Martin, & Briddon, 2011; Nahid et al., 2008; Schwinghamer, Knights, Breeder, & Moore, 2009). Among them, viruses causing or associated with stunt and chlorotic dwarf diseases are considered to be economically important (Table 9.1).

#### 2.7.1 Stunt

Chickpea stunt disease was first reported from Iran, and it occurs in North Africa, the Middle East, South Africa, Australia, Indian subcontinent, Spain, Turkey, and the United States (Kumar, Jones, et al., 2008). The causal agent of stunt was originally attributed to *Pea leaf roll virus* (syn. *Bean leaf roll virus*, BLRV) (Horn & Reddy, 1996; Nene & Reddy, 1987; Reddy & Kumar, 2004). Subsequently, BLRV and *Chickpea stunt luteovirus* (CpLV) were regarded as causal agents. They indicated that CpLV was probably a strain of BLRV but no comparative studies were reported. Later, studies showed that a leafhopper-transmitted geminivirus, *Chickpea chlorotic dwarf virus*...
CpCDV, was also capable of producing symptoms similar to those referred to as chickpea stunt in India and Pakistan (Horn, Reddy, Roberts, & Reddy, 1993). Surveys of chickpea with stunt symptoms in both India and Pakistan and follow-up serological and electron microscopic studies showed that the etiology of stunt disease was more complex than that was previously thought. The relative prevalence of the luteoviruses appeared to vary in the different chickpea growing areas of the Indian subcontinent. CpCDV- and CpLV-like isolates were widely distributed in India and Pakistan, whereas BLRV-like and Beet western yellows virus-like isolates were of minor importance (Horn, Reddy, van den Heuvel, & Reddy, 1996). The stunt disease is more common in the kabuli genotypes in Pakistan than in the desi types. The yield loss was nearly total if infection occurred in the early stage of growth; if infection occurred at the flowering stage, the yield losses could be as high as 75% (Horn, Reddy, & Reddy, 1995).

A new member of the genus Polerovirus named Chickpea chlorotic stunt virus has been reported to naturally infect chickpea (Abraham et al., 2006). It is persistently transmitted by A. craccivora and Acyrthosiphon pisum.

### 2.7.2 Chlorotic dwarf

CpCDV produces symptoms very similar to those of stunt that include leaf rolling, yellowing, necrosis, and stunting and was shown to be caused by a mastrevirus. It was recorded in India (Horn et al., 1993), Pakistan (Horn et al., 1995; Nahid et al., 2008), Iran and Sudan (Makkouk et al., 2003), Egypt, Iraq, Syria, and Yemen (Kumari, Makkouk, & Attar, 2006, Kumari et al., 2008). When the infection occurs before flowering, the yield loss was reported to be 100% in chickpea (Horn et al., 1993). Molecular characterization of the mastrevirus isolates associated with stunt disease in several countries was reported (Kanakala et al., 2012; Mumtaz et al., 2011; Nahid et al., 2008). The mastrevirus associated with severe stunting, reduction in leaf size, drying and eventual death of chickpea cultivars around Delhi, India, was characterized, and the differences between mastreviruses originating from Africa, the Middle East, Asia, and Australia were compared (Kanakala et al., 2012). Recently, five other mastrevirus species, Chickpea red leaf virus, Chickpea yellows virus, Chickpea chlorosis virus, Chickpea chlorosis Australia virus, and Tobacco yellow dwarf virus were found in Australia (Hadfield et al., 2012; Thomas, Parry, Schwinghamer, & Dann, 2010). All the mastrevirus isolates infecting chickpea in Africa, Australia, and Asia were reclassified on the basis of 78% nucleotide identity in the genomic DNA and were grouped into one species, CpCDV (Muhire et al., 2013). CpCDV
was also found to infect faba bean, lentil, French bean, pigeonpea, and lablab bean (Makkouk et al., 2003). Tissue-blot immunoassay (TBLA) was applied for the detection of CpCDV in chickpea and faba bean (Kumari, Najar, Attar, Loh, & Vetten, 2010).

The management of stunt disease relies upon identification of resistant sources and introgression of resistance genes into desired chickpea genotypes. Over 10,000 germplasm lines have been screened for resistance to stunt at Hisar, India, which is a hot spot for CpCDV, and the lines GG 669 and ICCC 10 were found to be field-resistant. Resistance was expressed as slower symptom development, compared to the susceptible line WR 315. Chickpea lines identified as resistant at Hisar showed 40–70% infection when screened at Junagadh, India (Horn et al., 1996). Nine entries were found to be resistant, and 33 entries were moderately resistant in field screening tests conducted at Junagadh during the years 2008 and 2009 (Chickpea research highlights, IIPR, Kanpur, India, 2009). Various strategies to control virus infections on chickpea were evaluated in Australia (Schwinghamer et al., 2009). Effective field screening for resistance to chickpea stunt viruses should include serological assaying of both susceptible and resistant genotypes and evaluation under greenhouse conditions against virus types and strains.

At present, evaluation of CpCDV resistance is conducted on the basis of natural infection in the field, which is bound to be erroneous due to vagaries in vector population and similar symptoms by unrelated viruses. Kanakala et al. (2013) devised an agroinoculation technique that involves the delivery of CpCDV genomic DNA through Agrobacterium tumefaciens, thus facilitating precise screening for CpCDV resistance. They screened 70 chickpea genotypes both under field conditions and by agroinoculation. The genotype SCGP-WR-29 showed resistance under field conditions but exhibited 80% incidence under agroinoculation, indicating that the resistance was presumably due to nonpreference by the vector. The true virus resistance was identified in the genotypes L-550, GNG-1499 (Gauri), and IPC 09-07, which did not express any symptoms and did not show the presence of virus DNA in PCR tests. This type of resistance needs to be exploited for generating CpCDV-resistant chickpea cultivars.

The experiments conducted by International Center for Agricultural Research in the Dry Areas (ICARDA, Syria) in collaboration with Agriculture Research organization in Sudan showed that delayed planting of chickpea cultivars Shendi and ICCV-2 (late October, early or late November) and irrigation at short intervals resulted in reduced CpCDV incidence in Northern Sudan.
2.8. Pea

Pea is widely grown in the temperate regions. Its cultivation in tropics is restricted to cool season. Diseases caused by PSbMV, BYMV, *Pea enation mosaic virus* (PEMV)-1, PEMV-2, and BLRV are economically important (Table 9.1) (Kraft & Pfleger, 2001; Makkouk et al., 2003, 2012).

2.8.1 Mosaic caused by *PSbMV* and *BYMV*

PSbMV is widely distributed largely due to its high levels of seed transmission, thus facilitating entry through international exchange of germplasm. PSbMV is transmitted in a nonpersistent manner by the aphid species *A. pisum*, *M. persicae*, and *A. gossypii* (Kraft & Pfleger, 2001). ELISA, dot-immunobinding assay, and PCR-based tests were reported for virus detection and characterization (Makkouk et al., 2012). Managing the aphid vectors could offer some protection by reducing the aphid-mediated secondary spread from the initial disease foci that resulted from planting of PSbMV infected seed. Genetic resistance for seed transmission of PSbMV provides the best option for reducing the virus spread and its subsequent establishment especially in new areas where the virus was not previously reported (Kraft & Pfleger, 2001). Resistance, characterized as immunity, is controlled by a single recessive gene *sbm*. The *sbm-1* gene confers resistance to P-1 and P-2 isolates, and *sbm-2* and *sbm-3* to P-2 isolates, while only *sbm-4* to P-4 isolates of PSbMV (Johansen, Keller, Dougherty, & Hampton, 1996; Provvidenti & Alconero, 1988). The *sbm-1* gene is linked to *wlo* on chromosome 6 of the *Pisum* genome while *sbm-2* is linked to *mo* on chromosome 2. Recessive genes *sbm-3* and *sbm-4* are also found on chromosome 6, but linkages have not been established. Two homologous genes, translation initiation factor *eIF4E* and *eIF(iso)4E*, governed resistance to PSbMV and BYMV, respectively, at the *sbm-1* and *sbm-2* locus (Bruun-Rasmussen et al., 2007; Gao et al., 2004). In Czech Republic, *eIF4E*-specific molecular markers for PSbMV resistance were developed (Smýkal, Safarova, Navratil, & Dostalova, 2010). The genome of pea was fully sequenced thus permitting marker-assisted breeding (Smýkal et al., 2012).

BYMV was reported on pea in Syria, Egypt, Italy, and Libya and is seed-transmitted in peas, faba beans, lentils, lupins, and a number of forage legumes. ELISA and RT-PCR were used for the detection of BYMV (Makkouk et al., 2012). Spatial isolation of pea fields from virus reservoir hosts limited the spread of the virus by aphids. Several BYMV-resistant pea cultivars and breeding lines were developed using a single recessive gene, *mo* (Kraft & Pfleger, 2001).
2.8.2 Enation mosaic
Pea enation mosaic disease is caused by PEMV-1 and PEMV-2 (Makkouk et al., 2012). PEMV-1 is transmitted efficiently in a persistent manner by at least eight aphid species. *A. pisum* is considered to be the most efficient vector. ELISA- and RT-PCR-based tests for detection of PEMV were reported (Chomic et al., 2010; D’Arcy, Torrance, & Martin, 1989). Resistance to PEMV infection in peas and lentil was reported (Aydin, Muehlbauer, & Kaiser, 1987). PDR- and RNAi-mediated strategies have potential for introducing resistance to PEMV.

2.8.3 Top yellows
Top yellows disease is caused by BLRV. Yield is affected due to misshapen and/or poorly filled pods (Kraft & Pfleger, 2001; Makkouk et al., 2003, 2012). *Medicago*, *Trifolium*, and *Vicia* species act as sources of virus inoculum. Extensive secondary spread was reported if infestation by aphids was not controlled. Therefore, judicious application of insecticides coinciding with aphid monitoring can reduce incidence and spread of BLRV. Spatial isolation of pea fields from virus sources may not help as the virus is persistently transmitted by aphids. Rouging of initially infected pea plants reduced the secondary spread. Resistance to BLRV is inherited as a single recessive gene, designated *lr*. Another recessive gene, *lrv* confers tolerance to BLRV in pea. Several resistant/tolerant pea cultivars available from seed companies are listed (Kraft & Pfleger, 2001).

2.9. Faba bean
Faba bean is extensively cultivated in West Asia and North African (WANA) countries. Among several viruses reported to naturally infect this crop, FBNYV, *Faba bean necrotic stunt virus* (FBNSV), BLRV, BYMV, and *Broad bean mottle virus* (BBMV) are economically important (Table 9.1) (Jellis, Bond, & Boulton, 1998; Makkouk et al., 2012). Faba bean breeding for resistance to different types of diseases including viruses was reviewed (Sillero et al., 2010).

2.9.1 Necrotic yellows and necrotic stunt
Necrotic yellows and necrotic stunt diseases were reported to be caused by two distinct species of the genus *Nanovirus*, FBNYV and FBNSV, respectively. Of these two, FBNYV was widely distributed (Algeria, Egypt, Lebanon, Libya, Spain, Tunisia, Ethiopia, Jordan, Morocco, Syria, and Turkey). In addition to faba bean, it naturally infects other food legume crops pea,
chickpea, cowpea, common bean, and lentil. ELISA, TBIA, dot-blot hybridization, and PCR-based tests were employed to differentiate FBNYV from other viruses infecting faba bean (Makkouk & Kumari, 2009). FBNYV is transmitted efficiently by aphids *A. pisum*, *A. craccivora*, and relatively less efficiently by *Aphis fabae*, in a circulative persistent manner. FBNYV is not known to be transmitted by seeds or mechanically (Makkouk & Kumari, 2009). The recommended integrated virus management practices consisted of (a) seed treatment with imidacloprid before planting, (b) judicious application of aphicides, (c) planting at an appropriate time to avoid peak number of viruliferous aphids, (d) planting to provide high-density crop stand, and (e) planting with resistant genotypes (Makkouk & Kumari, 2009).

### 2.9.2 Leaf roll

Leaf roll disease in faba bean is caused by BLRV. The occurrence of this disease has been reported in the Mediterranean countries (Egypt, Morocco, Tunisia, Lebanon, Syria, and Spain) and Australia. BLRV can be diagnosed by ELISA, TBIA, and RT-PCR (El-Beshehy & Farag, 2013; Freeman et al., 2013; Makkouk et al., 2012; Ortiz, Castro, & Romero, 2005). BLRV is transmitted by aphids (*A. pisum*, *A. craccivora*, *A. fabae*, and *M. persicae*) in a circulative, nonpropagative manner. Seed treatment with imidacloprid before planting and adjustment to sowing dates can reduce the disease incidence. Application of aphicides (organophosphorus, carbamate, and pyrethroid) was shown to reduce disease incidence (Jellis et al., 1998). Genotypes resistant to BLRV have been reported in faba bean (Makkouk, Kumari, & van Leur, 2002) and lentil (Makkouk, Kumari, Sarker, & Erskine, 2001). At ICARDA, faba bean accessions BPL 756, BPL 757, BPL 758, BPL 769, BPL 5278, and BPL 5279 were found to be resistant to BLRV (Kumari & Makkouk, 2003; Makkouk et al., 2002).

### 2.9.3 Mosaic and necrosis

Mosaic and necrosis in faba bean are caused by BYMV. It was reported from Israel, Italy, Lebanon, Libya, Morocco, Syria, Tunisia, Greece, and Turkey. A high incidence, up to 100%, has been reported in some regions of Egypt, Iraq, and Sudan with relatively warm winters (Makkouk et al., 2012). BYMV is seed-transmitted in faba bean up to 2.4% (Kaiser, 1973). BYMV was detected in several commercial faba bean seed samples up to 9.2% (Sayasa, Iwasaki, & Yamamoto, 1993). Primary source of inoculum was shown to come from seed (Jellis et al., 1998). Therefore, use of virus-free seed was recommended for planting to minimize disease incidence.
Adjustment to sowing dates, spraying with mineral oils, soil mulching with reflective polythene sheets, and ensuring that faba bean crops were not grown in the vicinity of known over wintering virus sources were recommended for reducing BYMV incidence (Mahdy, Fawzy, Hafez, Mohamed, & Shahwan, 2007). Faba bean accessions, BPL 1351, BPL 1363, BPL 1366, and BPL 1371, were found to be resistant to BYMV (Kumari & Makkouk, 2003; Makkouk & Kumari, 2009; Makkouk et al., 2012). In Egypt, resistance in faba bean to BYMV infection was analyzed through diallel mating scheme including reciprocals of six faba bean genotypes with varied resistance and susceptibility to BYMV infection. Resistance was inherited polygenically (El-Bramawy & El-Beshehy, 2012).

2.9.4 Mottle
Mottle disease in faba bean is caused by BBMV. Depending on time of infection, grain yield losses of up to 55% have been reported (Makkouk, Bos, Azzam, Kumari, & Rizkallah, 1988, Makkouk, Bos, Rizkallah, Azzam, & Katul, 1988). High incidence of BBMV was recorded in Morocco, Sudan, Tunisia, Syria, Egypt and Algeria (Makkouk et al., 2012). BBMV was detected in seed and plant tissues by employing ELISA or TBIA (Makkouk, Bos, Azzam, et al., 1988). BBMV was transmitted by mechanical inoculation and by beetle vectors (Acalymma trivittata, Diabrotica undecimpunctata, and Spodoptera exigua). Seed transmission up to 1.4% was reported, especially when BBMV occurred in mixed infection with BYMV. Planting with virus-free seed was recommended (Makkouk, Bos, Rizkallah, et al., 1988). Faba bean genotypes resistant to BBMV are currently not available.

2.10. Lentil
Lentil, one of the world’s oldest cultivated plants, originated in the Middle East and spread through Western Asia to the Indian subcontinent (Erskine, Muehlbauer, Sarker, & Sharma, 2009). Of the several viruses reported to naturally infect lentil (Makkouk et al., 2003), diseases caused by BLRV, FBNYV, PSbMV, CMV, BBSV, and BYMV are of economic significance (Table 9.1).

2.10.1 Yellows and stunt
Yellowing and stunting diseases in lentil, caused by BLRV and FBNYV, were already discussed under faba bean. BLRV is an important virus reported on lentil from Bangladesh, Ethiopia, Iran, Iraq, Syria, Tunisia, and the United States. When plants were infected at the preflowering stage, yield reductions up to 91% were reported. FBNYV was reported on lentil
from Ethiopia, Iran, Iraq, Pakistan, Syria, and Turkey (Kumari, Attar, Mustafayev, & Akparov, 2009). No sources of resistance have been recorded for these two viruses.

### 2.10.2 Mosaic and mottle

Mosaic and mottling diseases in lentil are caused by PSbMV, BYMV, BBSV, and CMV. The natural occurrence of PSbMV was reported from Algeria, Egypt, Ethiopia, Iran, Iraq, Jordan, Morocco, New Zealand, Pakistan, Syria, Tunisia, and Turkey (Kumari et al., 2009). Crop losses of up to 61% were reported from Pakistan (Kumari & Makkouk, 1995; Kumari, Makkouk, & Ismail, 1996). Seed transmission rates of PSbMV in lentil varied widely (0–44%) depending on the cultivar and virus isolate (Kumari et al., 2009). Under field conditions, the virus can over-winter in hairy vetch (Vicia villosa) and volunteer peas. From these sources, the virus is transmitted by aphids to nearby lentil crops.

Even though CMV occurs worldwide, its natural occurrence on lentil was reported only from Australia, Ethiopia, India, Iran, Nepal, New Zealand, Pakistan, and Syria. It is easily sap transmissible and nonpersistently vectored by more than 60 different aphid species. Its transmission through lentil seed varied from 0.05% to 37% (Kumari et al., 2009).

BBSV was reported on lentils from Ethiopia, Iran, Jordan, Syria, and Turkey. Grain yield losses varied from 14% to 61%, and the seed transmission rates were found to range from 0.2% to 32.4% when 19 lentil genotypes were inoculated at flowering stage. Infection of lentil plants at preflowering, flowering, and pod stages resulted in a seed-transmission rates of 20.6%, 19.1%, and 1.5%, respectively. BBSV is sap transmissible and by beetles *Apion aestival*, *Apion arrogans*, *Sitona crinite*, *Sitona limosa*, and *Sitona lineatus* (Kumari et al., 2009). ELISA, TBIA, and PCR were employed for the detection of lentil viruses (Kumari et al., 2009; Kumari & Makkouk, 2007).

Five viruses affecting lentil were seed-borne. For such viruses, planting with virus-free seed was recommended, especially when the virus was also vectored by insects. Seed transmission of BBSV was reduced to zero when seeds were exposed to 70 °C for 28 days; but this treatment caused an unacceptable reduction (57%) in seed germination (Kumari & Makkouk, 1996). Thermotherapy may be useful to eliminate seed-borne viruses from germplasm accessions, meant for conservation. The use of lentil cultivars resistant to the virus or to seed transmission is an effective control option (Kumari et al., 2009).
Over-wintering or over-summering crops, which could act as sources of infection for such nonpersistent viruses as BYMV, PSbMV, and CMV, should be avoided through spatial isolation that will adequately reduce virus spread. In contrast, persistently transmitted viruses such as BLRV and FBNYV can be carried from lucerne fields over long distances making it more difficult to avoid virus spread from these sources (Kumari et al., 2009).

Field experiments at ICARDA showed that seed treatment with the systemic insecticide imidacloprid significantly improved yields of moderately resistant and susceptible lentil genotypes, but had no effect on the yield of resistant genotypes. Seed treatment was also effective in increasing yields from BLRV and FBNYV-inoculated plots, but had no effect in SbDV-inoculated plots (Makkouk & Kumari, 2001).

Four lentil accessions (PI 212610, PI 251786, PI 297745, and PI 368648) were found to be resistant to PSbMV (Makkouk & Kumari, 2009). The genotype ILL 7163 was shown to be highly resistant to BYMV. Additionally, ILL 75 showed resistance to BLRV, FBNYV, and SbDV and ILL 74, ILL 85, ILL 213, ILL 214, and ILL 6816 genotypes were resistant to FBNYV and BLRV. Two cultivars from the United States, “Redchief” and “Palouse,” were tolerant to PSbMV infection, expressed as very low grain yield loss in addition to low seed-transmission rate (Kumari & Makkouk, 1995). “Redchief” was also reported to be tolerant to BBSV with low seed-transmission rates (Makkouk & Kumari, 1990).

3. VIRUS DISEASES OF MINOR FOOD LEGUMES

Minor food legume crops that have regional or local importance are hyacinth bean (Lablab purpureus, Syn. field bean, lablab bean, pole bean, dolichos bean, or Indian bean), horse gram (Macrotyloma uniflorum) and lima bean (P. lunatus). Viruses infecting minor food legume crops have been summarized (Makkouk et al., 2003; Odedara, Hughes, Odebode, & Odu, 2008). Only the diseases caused by begomoviruses on these crops have been shown to have the potential to cause significant crop losses in tropical environments and are briefly discussed.

3.1. Hyacinth bean

Hyacinth bean is thought to have originated in India, and spread to South and Eastern Asia, Africa, and the Americas (Murphy & Colucci, 1999; Shivashankar, Kulkarni, Shashidhar, & Mahishi, 1993). For over six decades, dolichos yellow mosaic disease attributed to Dolichos yellow mosaic virus
(DoYMV), was considered to be the major constraint of hyacinth bean production (Maruthi et al., 2006). DoYMV was identified as a geminivirus based on electron microscopy (Raj, Aslam, Srivastava, & Singh, 1988), serological and nucleic acid hybridization tests (Swanson, Varma, Muniyappa, & Harrison, 1992). DoYMV was poorly transmitted (to a maximum of 18.3%) by B. tabaci and had a narrow host range restricted to L. purpureus and L. purpureus var. typicum. MYMIV was also reported to be the causal agent of YMD of L. purpureus based on genome sequence analysis (Singh, Chakroborthy, Singh, & Pandey, 2006). YMD of pole bean in South India could be effectively managed by adopting integrated management practices that include border cropping with African tall maize, seed treatment with imidacloprid 70% WSW@ 5.0 kg, use of reflective mulches, spraying with triazophos 30 days after sowing (DAS) and with thiomethoxam 45 DAS. These measures contributed to yield of 32.2 tons/ha with a cost:benefit ratio of 1:3.17 (Jyothi et al., 2013).

Of the 300 L. purpureus genotypes screened under laboratory and field conditions, genotypes VRSEM 894, VRSEM 860, and VRSEM 887 showed no overt symptoms and did not show the virus in PCR tests (Singh, Kumar, Rai, & Singh, 2012). These genotypes have the potential for generating DoYMV-resistant L. purpureus cultivars.

3.2. Horse gram

Horse gram is mainly cultivated in the Indian subcontinent and Africa (Jayan & Maya, 2001). YMD was characterized by yellow mosaic on leaves, coupled with reduction of leaf size and plant height in severely infected plants. This virus was initially identified as HgYMV (Muniyappa, Rajeshwari, Bharathan, Reddy, & Nolt, 1987). In addition to horse gram, HgYMV infects French bean, groundnut, lima bean, mungbean, pigeonpea, soybean and bambara groundnut in India. HgYMV was identified as a distinct species of Old World bipartite begomoviruses (Barnabas, Radhakrishnan, & Ramakrishnan, 2010). Indigofera hirsuta, a legume weed, was shown to serve as a natural reservoir of HgYMV. Horse gram genotypes tolerant/resistant to HgYMV were identified (Muniyappa, Reddy, & Mustak Ali, 1978), and they are yet to be exploited in breeding programs. A wild relative of horse gram, Macrotyloma axillare, was found to be immune to virus infection.

3.3. Lima bean

Lima bean is native to Central America. Among several viruses reported to infect this crop, begomoviruses, viz., BCaMV (North America and
Mexico), BGMV (Latin America and the Caribbean) and Lima bean golden mosaic virus (Nigeria) are considered to be important (Makkouk et al., 2003). Lima bean crop is widely grown in Nepal. The frequently observed yellow mosaic symptoms are shown to be caused by MYMIV based on genome sequence analysis (Shahid et al., 2012). In SSA, lima bean is naturally infected by Lima bean golden mosaic begomovirus (Hughes, Naidu, & Shoyinka, 2001). The symptoms are golden mosaic and yellowing. The virus isolates associated with this disease in Nigeria were identified as strains of SbCBV based on sequence analysis of DNA-A component (Alabi et al., 2010).

4. CONCLUSIONS AND FUTURE PROSPECTS

Virus diseases have been shown to have significant impact on the production of major tropical and subtropical food legume crops soybean, groundnut, common bean, cowpea, pigeonpea, mungbean, urdbean, chickpea, pea, faba bean, and lentil (Table 9.1). Among the causal viruses, SbMV, TSWV, PBNV, PCV, IPCV, GRV, CMV, PShBVMV, BCMV, BCMNV, BLRV, BGMV, BGYMV, BYMV, PeMoV, PSTV, FBNYV, CpCDV, TSV, MYMIV, and MYIMIV are economically important. They infect more than one legume crops. Viruses transmitted through the seed of food legumes have quarantine importance and also serve as primary virus source in virus ecology and disease epidemiology (Sastry, 2013). Intensive cropping and changes to cropping systems as a result of increased access to irrigation facilities and abuse of pesticides are some of the factors aiding vector multiplication and spread. Occurrence of tospoviruses and TSV on numerous hosts, other than their natural hosts, is of major concern (e.g., Jones, 2009; Rojas & Gilbertson, 2008; Varma et al., 2011).

For the management of legume viruses, several control options available are selection and planting of virus-free seed, adjustment of crop cultural (agronomic) practices, chemical, physical, and biological control of virus vectors, and planting of virus-resistant crops developed through conventional and/or nonconventional breeding methods. These measures were practiced well to minimize the occurrence of important legume virus diseases (e.g., Hema, Gogoi, Dasgupta, & Sreenivasulu, 2014; Hooks & Fereres, 2006; Kumar, Jones, et al., 2008, Kumar, Kumari, et al., 2008; Malathi & John, 2008; Reddy et al., 2009; Sreenivasulu et al., 2008). With the exception of seed treatment to minimize spread of seed-transmitted viruses, pesticide use should be avoided as far as possible for controlling vectors of especially nonpersistently and semipersistently transmitted viruses. Large-scale screening of germplasm should be ideally done under field
conditions, preferably at hotspot locations. Laboratory screening for nonsap transmissible viruses, such as some of the begomoviruses, agroinoculation methods are available (Kanakala et al., 2013). Bacterial artificial chromosome (BAC) libraries of pulse crops have the potential to accelerate gene discovery and enhance molecular breeding in these crops (Yu, 2012). Advances in the development of transgenic pulse crops have been reviewed (Eapen, 2008). Groundnut (e.g., Chander Rao et al., 2013), soybean (e.g., Grossi-de-Sa, Pelegrini, & Fragoso, 2011), common bean (Tollefson, 2011), and mungbean (Haq et al., 2010; Sunitha et al., 2013; Yadav, Shukla, & Chattopadhyay, 2009) have been genetically engineered with virus genome-derived genes/sequences and resistance against targeted viruses has been evaluated. Of these, only genetically engineered common bean resistant to BGMV has been commercialized in Brazil (Tollefson, 2011). RNAi-based approaches are being exploited to develop virus resistance in chickpeas (Nahid, Amin, Briddon, & Mansoor, 2011). Despite the progress in genomics of legumes (Sharma, Upadhyaya, Varshney, & Gowda, 2013; Varshney, Mohan et al., 2013; Varshney, Song, et al., 2013) and availability of tools for transforming the plants, incorporation of resistance to economically important legume viruses by genetic engineering is yet to be accomplished on a commercial scale.

Sensible integration of the various options available for control remains the best choice for virus disease control. It should, however, be emphasized that selecting the best measures for each virus–crop combination and production system requires knowledge of the epidemiology of the causal virus in a given agroecosystem and the mode of action and effectiveness of each individual control measure (Jones, 2009; Jones & Barbetti, 2012). Each strategy must be affordable to the farmer and fulfill the requirements of being environmentally friendly and socially acceptable.

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