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Detection, Understanding and Control of Soybean Mosaic Virus

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1. Introduction

Among 67 or so viruses that are able to infect soybean, 27 are considered a threat to the soybean industry (Tolin and Lacy, 2004; Saghai Maroof et al., 2008). *Soybean mosaic virus* (SMV) is the most prevalent virus and is recognized as the most serious, long-standing problem in many soybean producing areas in the world (Wang, 2009). SMV is a member of the genus *Potyvirus* in the family *Potyviridae*. The disease caused by SMV was first documented in the USA in 1915 by Clinton (1916) and SMV was named by Gardner and Kendrick (1921). Since then, the virus has been found in China, Japan, South Korea, Canada, Bazil, Australia and many other countries wherever soybean is grown. Infection by SMV usually results in severe yield losses and seed quality reduction. It has been reported that yield losses usually range from 8 to 50% under natural field conditions (Hill, 1999; Arif and Hassan, 2002) and reach up to 100% in severe outbreaks (Liao et al., 2002). Since SMV is a seed-borne viral pathogen and aphids can efficiently spread it from plant to plant while they feed, it is difficult to control the virus and produce SMV-free seeds. Furthermore, SMV often infects soybeans with other viruses such as *Bean pod mottle virus* (BPMV), *Alfalfa mosaic virus* (AMV) and *Tobacco ringspot virus* (TRSV) (Wang, 2009). Such synergistic infections with two or more viruses cause much more severe damages than infection by each virus alone (Hill et al., 2007; Wang, 2009). Utilization of soybean cultivars resistant to SMV is considered the most effective way of controlling the diseases. Extensive screening for soybean gemplasm resistant to SMV has resulted in the identification of three independent resistant genes, i.e., *Rsv1*, *Rsv3*, and *Rsv4* (Hayes et al., 2000; Gunduz et al., 2002; Liao et al., 2002; Zheng et al., 2005; Li et al., 2010a). Interestingly, several naturally occurring resistance-breaking SMV isolates have also been reported that can break all three or two soybean resistance loci (Choi et al., 2005; Gagarinova et al., 2008a). The development of durable genetic resistance to SMV becomes a research priority for soybean breeders and soybean pathologists. This may depend on advances in the understanding of the SMV life cycle and molecular SMV-soybean interactions.

2. General biology of SMV

2.1 Physical and biological properties

As a potyvirus, SMV virions consist of a capsid that is filamentous, flexuous rod-shaped with 650-760 nm in length and 15-18 nm in width (ICTVdB Management, 2006).

Encapsulated in the viral particle is the viral genome which is a linear, positive-sense, single-stranded RNA molecule. The thermal inactivation point (10 min) of particles is 55 to 60°C. (Bos, 1972). The decimal exponent (DEX) of the dilution end point (DEP) is 10^{-3}～10^{-5}, and the longevity *in vitro* (LIV) is 2～5 days at 25°C (Hill, 1999). SMV is relatively stable at pH 6.0 and loses infectivity at pH values higher than 9 or lower than 4 (Galvez, 1963). The cylindrical, pinwheel-shaped inclusions formed by the viral CI protein, a characteristic cellular phenotype for potyvirus infection, are often found in the cytoplasm of infected cells (A. Wag, unpublished).

2.2 Host range and symptoms

In comparison with other potyviruses, SMV has a relatively narrow host range. It infects six plant families, i.e., *Fabaceae* (also *Leguminosa*), *Amaranthaceae*, *Chenopodiaceae, Passifloraceae, Schropulariaceae and Solanaceae*, but mostly the *Leguminosae* including soybean and its wild relatives (Galvez, 1963; Hill, 1999). The symptoms induced by SMV depend on host genotype, virus strain, plant age at infection, and environment. In SMV-infected soybeans, symptoms commonly observed include rugosity, dark green vein banding and light green interveinal areas, stunting, leaf curling and seed coat mottling, male sterility, flower deformation, less pubescent, necrosis, sometimes necrotic local lesions, systemic necrosis and bud blight (ICTVdB Management, 2006). Some of these SMV symptoms may be masked at temperatures above 30°C (Hill, 1999).

2.3 Transmission

Up to 30% or more of the seeds from SMV-infected soybean plants carry SMV depending on cultivar and time of infection before flowering (Bos, 1992). SMV-infected seeds are the primary inoculum source, though weeds and other plants may also serve as a reservoir of SMV. Further spread within and among soybean fields is through the activity of more than 32 different aphid species of 15 different genera in a non-persistent manner (Cho and Goodman, 1982; Arif and Hassan, 2002; Steinlage et al., 2002). Some aphid species are *Acyrthosiphom pisum*, *Aphis craccivora*, *A. fabae*, *A. glycine*, *A gossypii*, *Myzus persicae*, *Rhopalosiphum maidis* and *R. padi*. In addition, SMV can be efficiently sap-transmitted with or without the use of abrasives.

2.4 Strains

Numerous SMV isolates have been reported worldwide. In the United States, at least 98 isolates of SMV have been documented (Cho and Goodman, 1979, 1982, 1983). Based on their differential reactions in two susceptible, i.e., Clark and Rampage, and six resistant soybean cultivars including Buffalo, Davis, Kwangyo, Marshall, Ogden and York, SMV isolates were classified into seven distinct strain groups, G1 through G7 (Cho and Goodman, 1979). Later, two more groups, G7A and C14 were added (Buzzell and Tu, 1984; Lim, 1985). Similarly, five strains (A to E) have been identified in Japan (Takahashi et al., 1963; Takahashi et al., 1980). In Canada, a necrotic strain, SMV-N, and a number of G2 isolates were identified (Tu and Buzzell, 1987; Gagarinova et al., 2008a; Viel et al., 2009). But SMV-N shares high sequence similarity with the G2 group and is thus considered a G2 isolate (Gagarinova et al., 2008b).

In South Korea, SMV was also monitored by pathotype. All the SMV G strains, including G1 through G7, SMV-N, G5H, G7a and G7H, have been found (Seo et al., 2009), but dominant

strains varied in different times. For instance, G5 caused about 80% of the SMV damages in the early 1980s, whereas in the late 1980s, G5H was a dominant strain, responsible for over 65% of the SMV-caused losses (Cho et al., 1983; Kim, 2003). More recently, G7H became the most prevalent strain and accounted for approximately 50% of SMV incidence in soybean fields (Kim et al., 2003; Seo et al., 2009). Due to the genetic variability of SMV and strong selection pressure, resistance breaking isolates may occur. Indeed, several resistance-breaking isolates including CN18 were identified in soybean fields in South Korea (Choi et al., 2005).

In China, SMV isolates were grouped into strains based on geographical distributions and responses in soybean resistant cultivars (Wang et al., 2003; Guo et al., 2005; Wang et al., 2005; Zhan et al., 2006). For instance, SMV isolates found in Northeast China were grouped into three strains, No. 1, 2, 3 using three soybean cultivars (Lv et al.,1985), whereas SMV isolates identified in Jiangsu Province were classified into strains Sa, Sb, Sc, Se, Sg and Sh based on pathogenicity in 10 other cultivars (Pu et al., 1982; Chen et al., 1986). Recently, a more comprehensive study using 10 soybean cultivars has classified 606 SMV isolates identified in South China into 21 strains, SC1 through SC21 (Li et al., 2010b). Since an SMV isolate may be grouped into different groups when different sets of soybean cultivars are used, obviously this creates difficulty for SMV identification, exchange of resistance germplasm, and comparison of resistance assay results. It is necessary to establish a standardized system for strain classification in China as well as other countries in the world.

It is worth to mention that SMV grouping based on their sequence similarity may be a more reliable approach. As more and more SMV isolates are sequenced, the complete genome sequences and partial gene sequences can be used to study the phylogenetic relationship and molecular variability (Frenkel et al., 1989; Saruta et al., 2005; Schirmer et al., 2005; Wetzel et al., 2006; Gagarinova et al., 2008b; Seo et al., 2009). The outcomes of these studies will certainly provide new insights into the evolutionary process of SMV in relation to its natural hosts.

2.5 Diagnosis

Because the initial infection source of SMV is mainly the seeds from SMV-infected soybean plants, the accurate diagnosis of SMV is very important for its effective management. A conventional method for SMV detection is to inoculate diagnostic hosts (indicator plants) to observe differential visual symptoms and determine the host range. The diagnostic host species and symptoms for SMV are: *Chenopodium album* or *C. quinoa*, chlorotic local lesions; *Lablab purpureus*, necrotic local lesion; *Macroptilium lathyroides*, systemic mosaic; *Phaseolus vulgaris*, systemic mosaic with some strains in some cultivars, but often latent or no infection; *P. vulgaris cv.* Top Crop, necrotic local lesions at 30°C in detached leaves (ICTVdB Management, 2006). The advantage of this method is relatively simple and does not require expensive instrument and complex techniques. This method, however, is not sensitive and requires considerate time and greenhouse space.

To overcome these shortcomings, serological assays have been used for SMV detection. These include the direct double sandwich – enzyme-linked immunosorbent assay (DAS-ELISA), indirect-ELISA, tissue-print immunoassay (TPIA), dot immunobinding assay (DIBA), immunosorbent electron microscopy (ISEM), immunofluorescence and western blotting. Some of these methods, particularly ELISA-related technology, can be used for the detection and quantitative estimation of a large number of soybean samples and thus are commonly used for SMV detection and screening. In addition to protein-based technology, highly sensitive polymerase chain reaction-based technology such as reverse transcription polymerase chain reaction (RT-PCR) and real-time PCR have also been widely used for SMV

detection. The SMV viral genome-derived PCR products may be sequenced, allowing comparison analysis with the published SMV sequence and providing the most accurate information for identification of virus strains.

3. Molecular biology of SMV

3.1 Sequence and genomic organization

The genome of SMV is a positive single-stranded [(+)ss] monopartite RNA molecule. Complete sequencing of 45 SMV isolates including all G1 to G7 strains suggests the SMV genome is approximately 9600 nucleotides (nt) in length (Jayaram et al., 1992; Gagarinova et al., 2008a). There is a virus genome-linked protein (VPg) at the 5' end and a poly(A) tail at the 3' end. The genomic RNA contains one long open reading frame (ORF) and a smaller ORF resulting from a frame-shift, both together encoding 11 mature viral proteins (Berger et al., 2005; Chung et al., 2008). From the N to C terminus of the two polyproteins, the 11 mature proteins are: P1 (the first protein), HC-Pro (the helper component/protease), P3 (the third protein), P3N-PIPO (resulting from the frame-shift in the P3 cistron), 6K1 (the first 6 kDa peptide), CI (the cylindrical inclusion protein), 6K2 (the second 6 kDa peptide), NIa-VPg (nuclear inclusion "a"–viral genome-linked protein; also VPg), NIa-Pro (nuclear inclusion "a" protein–the protease), NIb (the nuclear inclusion "b" protein), CP (coat protein) (Jayaram et al., 1992). Most of these viral proteins are multi-functional.

3.2 SMV-host interactions

Due to the availability of a large number of SMV isolates as well as numerous soybean cultivars, the SMV-soybean interaction may be the most complex of virus-plant interactions. As briefly discussed above, SMV is classified into different strains based on their different responses on several susceptible and resistant cultivars. These responses include susceptible (mosaic or crinkling), necrotic, or resistance (symptomless). In soybeans, three independent, dominant resistance loci, *Rsv1*, *Rsv3* and *Rsv4* conferring genetic resistance to partial or all SMV strains have been identified. *Rsv2* was initially assigned to the resistance gene in cultivar OX670 and later dropped when it was revealed to actually possess two resistance genes, *Rsv1* and *Rsv3* (Gunduz et al., 2001).

Rsv1 is a single-locus, multi-allelic gene. It was initially named *Rsv* and found in PI96983 soybean (Kiihl and Hartwig, 1979) and later renamed *Rsv1* (Chen et al., 1991). Subsequent studies on soybean cultivars resistant to SMV revealed additional eight different *Rsv1* alleles, *Rsv1-t*, *Rsv1-y*, *Rsv1-m*, *Rsv1-k*, *Rsv1-r*, *Rsv1-h*, *Rsv1-s* and *Rsv1-n* from cultivars Ogden, York, Marshall, Kwanggyo, 'Raiden', 'Suweon 97', LR1 and PI507389, respectively (Kiihl and Hartwig, 1979; Buss et al., 1997; Buzzell and Tu, 1989; Chen et al., 1991, 1993, 1994, 2001, 2002; Ma et al., 1995; Zheng et al., 2005). The responses of soybeans carrying these *Rsv1* alleles to different SMV strains are diverse, ranging from extreme resistance and necrosis to mosaic symptoms. The nine *Rsv1* alleles confer resistance to lower numbered SMV strain groups (G1 to G3), but allow mosaic or necrotic reactions to higher numbered strain groups (G5 to G7). *Rsv1* was mapped to the molecular linkage group F (soybean chromosome 13) in a cluster of resistance genes (Gore et al., 2002; Hwang et al., 2006). *Rsv3*, another single dominant gene, was found in cultivar Columbia, conditioning systemic necrosis against SMV strains G1 and G4 (Buzzell and Tu, 1989). Resistance cultivar L29, a Williams isoline contains an *Rsv3* allele derived from cultivar Hardee (Gunduz et al., 2002). Soybean plants carrying *Rsv3* alleles are resistant to higher

numbered strains G5 through G7, but susceptible to lower numbered strains G1 to G4 (Gunduz et al., 2002). *Rsv3* was mapped to the molecular linkage group B2 (soybean chromosome 14), also containing a cluster of disease resistance genes (Jeong et al., 2002). *Rsv4* is the third resistance gene of soybean, independent of *Rsv1* and *Rsv3*. It was reported in PI486355 and isolated in V94-5152, derived form hybrid PI486355 x Essex (Buss et al., 1997). Columbia was also found to carry *Rsv4* (in addition to *Rsv3*) (Ma et al., 2002). *Rsv4* is dominant, non-necrotic and non-strain specific, conferring resistance to all strains of SMV (Ma et al., 1995). It was mapped to the molecular linkage group D1b (soybean chromosome 2) where no other resistance genes have been found (Hayes et al., 2000; Saghai Maroof et al., 2010).

To study *Rsv1*-SMV interaction, Hajmorad et al. (2005) used the *Rsv1* gene in soybean PI96983 conferring extreme resistance to several G strains but susceptible to two G7 isolates, G7 and G7d. The former induced a leathal systemic hypersensitive response (LSHR) in PI96983, whereas the later, an experimentally evolved variant of G7, caused systemic mosaic symptoms. Through SMV chimeric infectious clones resulting from swapping different genomic regions of G7 and G7d and further SMV mutants through point mutagenesis, P3 was narrowed down to be the elicitor of *Rsv1*-mediated LSHR (Hajimorad et al., 2005). However, further studies using G7 and other SMV strains suggest the absence of P3 elicitor function alone is not sufficient to gain virulence and HC-Pro complementation of P3 is required for G7 to gain virulence (Hajimorad et al., 2006, 2008). To elucidate SMV-*Rsv3* interaction, Zhang et al. (2009) used a G2 isolate (SMV-N) that systemically infects *Rsv3* soybean and a G7 isolate which is restricted by *Rsv3* (Zhang et al., 2009). Infection test using recombinant SMVs from exchanging fragments between the avirulent G7 and the virulent SMV-N concluded that both the N- and C-terminal regions of the CI protein are required for *Rsv3*-mediated resistance (Zhang et al., 2009). For G2-*Rsv4* interaction, Chowda-Reddy et al. (2010) constructed two infectious clones corresponding to a naturally occurring resistance-breaking isolate and its closely related non-resistance-breaking avirulent isolate (Gagarinova et al., 2008a). Using the similar strategy described above, they determined that P3 in the G2 strain is an avirulent elicitor for *Rsv4* (Chowda-Reddy et al., 2010). Despite these studies, how these resistance genes control resistance to SMV and how the elicitors trigger resistance response to SMV remain unknown.

In susceptible cultivars, SMV-soybean interaction is a compatible reaction. After entry into the cell, SMV proceeds viral genome translation and replication, vial particle assembling, and cell-to-cell and long-distance movement. Such compatible virus infection often induces and suppresses host gene expression at the global level (Whitham et al., 2006). Babu et al. (2008) assessed transcriptional changes in susceptible soybean cultivar Williams 82 infected by SMV using microarray. A number of transcripts encoding proteins for hormone metabolism, cell-wall biogenesis, chloroplast functions and photosynthesis were shown to be repressed at 14 days post infection (Babu et al., 2008). Theses changes were associated with the highest levels of SMV genomic RNA in the host cells and the progression of mosaic and vein clearing symptoms (Babu et al., 2008). The expression levels of a number of transcripts corresponding to genes involved in defense were either downregulated or not affected at the early stages of infection, but upregulated at the late stages (Babu et al., 2008). These data suggest that in susceptible cultivars, the plant immune response is not activated until the late time point of infection and such a delayed defense response may be critical for SMV to establish its systemic infection.

3.3 SMV replication

SMV enters soybean cells either through a mechanical abrasion or by an aphid vector. Subsequently, uncoating of the viral RNAs in the cytoplasm, translation of the polyprotein and replication of the viral genome occur. Accumulated evidence suggests the replication of eukaryotic positive-strand RNA viruses including plant potyviruses is associated with intracellular membranous structures (Wei and Wang, 2008). These membranous vesicles have been proposed to provide a scaffold for anchoring the virus replication complex (VRC), confine the process of RNA replication to a specific safeguarded cytoplasmic location to prevent the activation of host defense responses, and to recruit the components required for replication and maintain the proper concentrations of these components (Wileman, 2006). The potyvirus VRC-containing vesicles seem to originate at endoplasmic reticulum exit sites (ERES), traffic along the microfilaments and target chloroplasts for replication (Wei et al., 2010). It has been shown that in potyvirus-infected plant cells, these vesicular compartments contain a number of host proteins, i.e., heat shock cognate 70-3 (Hsc70-3), poly(A)-binding protein (PABP), eEF1A and eIF(iso)4E, and several non-structural viral proteins, such as the 6K2, NIb (the viral RNA dependent RNA polymerase, RdRp), NIa (including NIa-VPg or NIa-Pro or as a precursor protein) and CI (Dufresne et al., 2008; Thivierge et al., 2008; Cotton et al., 2009; Wei et al., 2010a).

The 6K2 (also called 6K) protein is an integral membrane protein that induces the formation of 6K-containing membranous vesicles at ERES (Wei and Wang, 2008). These ER-derived vesicles further target chloroplasts where they amalgamate and induce chloroplast membrane invaginations (Wei and Wang, 2008; Wei et al., 2010a). Thus, 6K plays a pivotal role in the formation and targeting of VRC-associated vesicles. Since NIb has the RNA polymerase activity (Hong and Hunt, 1996), NIa-VPg (also called VPg) is the genome-linked protein (Murphy et al., 1996; Schaad et al., 1996) and CI has the helicase activity (Laín et al., 1990), it is not surprising that these viral proteins are present in VRC-associated vesicles and involved in the replication of potyviruses (Revers et al., 1999). It has been suggested that NIa is brought into VRCs by the domain for the 6K protein present on the intermediate precursor protein 6K-NIa (Restrepo-Hartwig and Carrington, 1992; Thivierge et al., 2008). The NIb protein (RdRP) has been reported to be present in the vesicles induced by 6K-NIa due to its physical interaction with NIa or as an intermediate precursor (6K-NIa-NIb) (Daròs et al., 1999; Dufresne et al., 2008; Thivierge et al., 2008). The viral RNA is probably enclosed to VRCs by the RNA-binding domain of NIb (Thivierge et al., 2008; Dufresne et al., 2008).

The host proteins, heat shock cognate 70-3 (Hsc70-3), poly(A)-binding protein(PABP) and eEF1A are recruited by VRC likely through interactions with NIb, and eIF(iso)4E is present in the replication vesicles as an interactor with VPg (Dufresne et al., 2008; Thivierge et al., 2008). Within the induced vesicles, the RdRp binds to the 3' termini of the viral (+)RNA to initiate transcription of negative strand replicative form RNA. This (-)RNA intermediate is used as a template to produce progeny (+)RNAs that are delivered to the cytoplasm for translation or encapsidation. The CP binds positive-sense progeny (+)RNAs to form progeny virions with VPg attaching on the end. The potyvirus replication possibly begins with uridylylation of VPg which acts as a primer for progeny RNA synthesis, a process shared by the *Picornaviridae* family (Puustinen and Mäkinen, 2004).

3.4 SMV cell-to-cell and long-distance movement

After replication and assembling, nascent virus particles will spread to neighboring cells and further to other parts of the plant. The first process is called cell-to-cell movement and

the latter long-distance movement. Cell-to-cell movement of viruses occurs through plasmodesmata (PD), a specialized intercellular organelle, unique to the plant kingdom. This is an active process mediated by virus-encoded protein(s) termed movement protein (MP). Potyviruses do not encode a dedicated MP, and movement functions have been allocated to several proteins. Of the 11 potyvrial proteins, CP, VPg, HC-Pro, CI and P3N-PIPO have been suggested to have functions in intercellular transport (Dolja et al., 1994, 1995; Nicolas et al., 1997; Rojas et al., 1997; Carrington et al., 1998; Wei et al., 2010b; Wen et al., 2010). Accumulating evidence indicates that HC-Pro and VPg are essential in other aspects of the infection process such as viral genome replication or suppression on host defense (RNA silencing) (Kasschau and Carrington, 1998; Puustinen and Mäkinen, 2004), whereas CP, CI and P3N-PIPO are likely to be MPs of potyviruses. The potyviral CP is a three-domain protein with variable N- and C-terminal regions exposed on the particle surface and a conserved core domain that interacts with viral RNA (Allison et al. 1985; Shukla and Ward 1988). The C terminal part of SMV CP contains two small regions (amino acids 190-212 and 245-249) required for CP-CP interaction and virus assembly (Kang et al., 2006). Mutations in the CP-core domain result in defective cell-to-cell movement and virion assembly (Dolja et al., 1994, 1995; Rojas et al., 1997; Jagadish et al. 1993), suggesting potyviruses likely move as virions. The potyvirus CI has been suggested to play a role in cell to cell movement (Laín et al. 1990; Eagles et al. 1994; Klein et al. 1994). High-resolution ultrastructural analyses indicate that CI forms the cone-shaped structures at the cell periphery adjacent to PD (Rodríguez-Cerezo et al. 1997; Roberts et al. 1998, 2003). In the case of the newly found P3N-PIPO protein, it has been shown that mutation of the putative SMV PIPO domain impeded cell-to-cell movement (Wen et al.,2010) and that P3N-PIPO mediated the formation of CI conical structures at PD (Wei et al., 2010b). Based on the model suggested by Wei et al. (2010b), cell-to-cell movement may be initiated when the recruitment of nascent virus particles by CI or self-interacting CI structures at membrane-bound sites of replication adjacent to chloroplasts. Then CI-virion complexes may associate with either pre-targeted P3N-PIPO followed by trafficking to PD, or with PD-associated P3N-PIPO. CI structures accumulate from P3N-PIPO-anchored sites at PD, forming thread-like structures that might recruit additional virus particles for transport. Virus particles fed through the CI structures and PD to enter the adjacent cell may be facilitated by PD-traversing CI complexes.

Long-distance movement is the process by which the virus moves from the mesophyll via bundle sheath cells, phloem parenchyma, and companion cells into phloem sieve elements (SE) where they are translocated, then unloaded at a remote site from which further infection will occur (Carrington et al. 1996). Due to the complexity of the various cellular structures involved, the molecular mechanism of potyviral long-distance movement is poorly understood. Four potyviral proteins, i.e., CP, HC-Pro, VPg and 6K2, have been suggested to be implicated in long distance movement. HC-Pro and CP are both multifunctional proteins (Shukla et al., 1994; Mahajan et al., 1996; Maia et al., 1996). The central region of HC-Pro and both termini of CP were shown to be essential for virus long-distance movement (Dolja et al., 1994, 1995; Cronin et al., 1995; López-Moya and Pirone, 1998). VPg affecting long-distance movement may function through its direct or indirect interaction with host components (Schaad et al., 1997). The 6K2 protein of *Potato virus A* is a host-specific determinant for long-distance movement in *N. tabacum* and *N. benthamiana* (Spetz and Valkonen, 2004). Potyvirus long-distance movement also requires a set of host factors. Strain and virus specific restriction of movement in plants has been described in several virus-host systems. Genetic characterization from natural ecotype variations and

chemically induced mutants has revealed that at least three dominant genes named *RTM1*, *RTM2* and *RTM3* are involved in the restriction of long-distance movement of potyviruses in the *Arabidopsis* accession Columbia (Col-0) (Mahajan et al., 1998; Whitham et al., 1999). These RTM factors affect the potyvirus long-distance transport through direct or indirect interaction with CP (Decroocq et al., 2009).

4. Control of SMV

The best strategy against plant viruses is either through the physical separation of the pathogen and host to avoid, or through the deployment of genetic resistance to prevent or limit the extent of the infection (Maule et al., 2007). In practice, the SMV-infected seeds and aphid transmission are the most prevalent factors causing SMV in fields. Thus, utilization of virus-free seeds and avoiding aphid transmission are an effective management measure against SMV in farming practices. Although considerable time and cost may be required for developing varieties, breeding for genetic stable varieties with the appropriate range of resistances is still the preferred and reliable approach to control the disease.

4.1 Breeding using natural resistance genes

All the three genetically identified resistance genes (*Rsv1*, *Rsv3* and *Rsv4*) have been deployed in China, the United States, Canada and other courtiers for controlling SMV. A number of soybean accessions (germplasm) and cultivars carrying resistance to SMV have been identified and used in the breeding program. V94-5152 confers an early resistance at the *Rsv4* locus to SMV G1 to G7. The OX670, Tousan 140 and Hourei soybean were shown to possess two genes, *Rsv1* and *Rsv3* (Gunduz et al. 2001, 2002), conferring resistance to G1 to G7 too. In China, Zao18 and J05 also carry *Rsv1* and *Rsv3* (Liao et al. 2002; Zheng et al., 2006). Zao18 was reported to be resistant to all strains found in Northeast China and majority strains in Southern China, while J05 is resistant to the most virulent strains of SMV in Northeastern China (Zheng et al., 2000). In addition, Columbia soybean was shown to carry two genes, *Rsv3* and *Rsv4*, for resistance to SMV G1 to G3 and G5 to G7 (Ma et al. 2002, 2004). All these soybean genotypes resistant to SMV are the valuable resource for breeding programs. Since the seed-borne, aphid-transmitted SMV is genetically variable and continually evolving via RNA recombination and spontaneous mutations by its own error-prone RdRp, strong directional selection would lead to the occurrence of resistance-breaking isolates (Choi et al., 2005; Saruta et al., 2005; Gagarinova et al., 2008a). Incorporation of multiple resistance genes into a soybean genotype (cultivar or variety) through gene pyramiding becomes a priority for soybean breeders to develop durable resistance to SMV. To assist in breeding programs, molecular markers of all the three resistance genes have been developed based on fine mapping with several molecular techniques such as restriction fragment length polymorphism (RFLP), random amplified polymorphism DNA (RAPD), amplified fragment length polymorphism (AFLP), simple sequence repeats (SSR) and single nucleotide polymorphisms (SNP) (Yu et al., 1994; Hayes et al., 2000; Gore et al., 2002; Jeong et al., 2002; Jeong and Saghai Maroof, 2004; Hwang et al., 2006; Shi et al., 2008).

4.2 Development of genetic resistance through biotechnology

Pathogen-derived resistance (PDR) is an established, effective approach to engineer resistance to plant viruses in plants. It has been used to develop genetic resistance against a

wide range of plant viruses including potyviruses (Powell-Abel et al., 1986; Lius et al., 1995; Di et al., 1996; Wang et al., 2009). This approach requires generation of transgenic plants with partial viral genomes. The resulting resistance is often mediated by RNA silencing or posttranscriptional gene silencing (PTGS) which induces sequence-specific degradation of viral RNA (Waterhouse et al., 2001). But the RNA silencing-mediated resistance may be overcome in two scenarios (Huang et al., 2010), i.e., in mixed infections by a strong silencing suppressor from unrelated viruses (Mitter et al., 2001) or through mutations during virus replication by the viral RdRP that lacks proofreading activities (Kang et al., 2005b).

To develop PDR against SMV, the CP and 3'-UTR region of the SMV N isolate (strain G2) was engineered into soybean (Wang et al., 2001). The expression of the single copy of the partial viral genome segment was controlled by the cauliflower mosaic virus *35S* promoter. Two transgenic lines showed high resistance to all SMV strains or isolates tested including G2, G6, G7 and an isolate named A15 obtained from South Carolina (Wang et al., 2001). None of the transgenic lines showed immunity to SMV infection (Steinlage et al., 2002). Since the transgenes that produce mRNA containing an intron-spanned hairpin structure usually induce high level of PTGS (Smith et al., 2000; Waterhouse et al., 2001), it is expected that soybean transformed with such constructs may obtain stronger resistance or immunity to SMV.

4.3 Novel strategies to control SMV

During the past several years, several new strategies have been developed against plant viruses. At the protein level, one approach is to engineer transgenic plants producing desirable proteins that can inhibit activities of essential viral proteins (Sanfaçon, 2009). For instance, transgenic plants expressing single-chain antibodies specific for the viral RdRp were shown to be resistant to a tombusvirus and several related viruses (Boonrod et al., 2004). Broad application of this approach may be hindered by the adverse pleiotropic effects of the antibodies and low levels of protein accumulation due to unwanted PTGS (Sanfaçon, 2010). At the RNA level, one of the reported approaches is to utilize artificial miRNAs which are small RNA molecules of 21-25 nucleotides long and negatively regulate the expression of their target genes in plants. miRNA precursors can be modified to produce artificial miRNA specifically targeting virus of interest and to induce resistance to the virus (Niu et al., 2006). Artificial miRNAs may be designed to target conserved regions of a virus family or related viruses to gain broad resistance. Since the miRNAs approach eliminates the potentially undesired recombination events due to their short length and does not involve translation because of their untranslatable nature, it becomes a very promising technology for controlling plant viruses. An alternative approach to the utilization of transgene-derived miRNAs is the direct application of PTGS inducer, long double-strand RNA (dsRNA) (Tenllando et al., 2003). Spray onto the surface of plant leaves with bacteria-produced double-strand RNA was shown to be efficient against different viruses (Tenllando et al., 2003). Limitations of this approach may rely on the maintenance of effectiveness of dsRNA, costs of dsRNA production and safety of large-scale application of dsRNA in the field.

Advances in the understanding of molecular virus-plant interactions as well as the virus life cycle will certainly assist the development of novel antiviral strategies. One emerging technology is to induce recessive resistance. The rational of this approach is that plant viruses have a relatively small genome that encodes a limited number of proteins and thus must depend on host gene products to fulfill their life cycle. Silencing or mutation of host

factors required for virus infection will generate genetic resistance to the virus. Over the last decade, a number of host factors required for potyvirus infection have been identified and examples include eIF4E, eIF(iso)4E, eIF4G, PABP, AtRH8 and PpDDX (Lellis et al., 2002; Ruffel et al., 2002, 2005; Nicaise et al., 2003, 2007; Gao et al., 2004; Kang et al., 2005; Decroocq et al., 2006; Bruun-Rasmussen et al., 2007; Dufresne et al., 2008; Huang et al., 2010). Recently Piron et al. (2010) have reported a successful story about how to deploy host factors for the development of resistance to potyviruses in tomato. They exploited a chemical-induced tomato mutant population, screened for mutants of eIF4E and eIF4G using a reverse genetic tool, TILLING (Targeting Induced Local Lesions IN Genomes), and identified a splicing mutant of eIF4E. The mutant was shown to be immune to two plant potyviruses (Piron et al., 2010). This example is particularly encouraging as the mutant did not involve in genetic transformation. The technology may be adapted for the generation of resistance to viral diseases in virtually any crops including soybean.

5. Conclusion remarks

Soybean is one of a few most important crops in the world and serves as a principal dietary food and oil source. Soybean oil is also considered a promising alternative to fossil oil. SMV is one of the major biotic factors that adversely affect soybean production. In this chapter, we have briefly discussed SMV as a pathogen, SMV-soybean interactions and its current and future control strategies. The current measures to control damages caused by SMV are (1) the development and use of soybean cultivars carrying at least one resistance gene, (2) the use of SMV-free seeds, (3) the selection of proper planting time, and (4) the control of aphid with pesticides. Along with the climate change (such as global warming), the emergence of new severe isolates (including resistance-breaking isolates) and new vectors such as *Aphis glycines*, and an increasing rate of synergic infections of SMV and other soybean viruses, the current measures are becoming less and less effective. After extensive screening over a considerable time, isolation of new natural SMV resistance genes from existing germplasm is not optimistic. New technologies must be developed to deal with SMV. One of future research directions might be on identification of soybean genes that are required for SMV infection. These genes will be new targets for manipulation against SMV. SMV starts from infected seeds but spreads from plant to plant by aphids. Unfortunately, very little study has been done on SMV-aphid interactions. This may be a topic of collaborative research between plant virologists and entomologists. Recently, the complete draft sequence of soybean has been released (Schmutz et al., 2010). The availability of the complete soybean genome sequence will certainly facilitate the molecular cloning and characterization of the three *R* genes and elucidating their resistance signaling pathways, and provide a better understanding of the co-evolutionary events of the *R* genes and the SMV genome. Information from these studies will help develop novel strategies against SMV and other related viruses.

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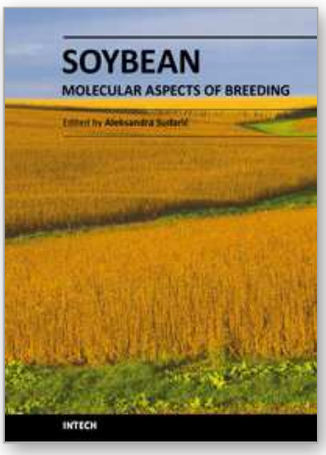
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