Australian Journal of Crop Science AJCS
AJCS 8(5):806-813 (2014) ISSN:1835-2707

Inheritance and mapping for resistant genes of soybean adult-plant and seed coat mottling to soybean mosaic virus №3 strain

Yanzhi Mao[1, 5†], Zhe Yang[1, 3†], Kang Wang[1, 2†], Dawei Xin[1, 2†], Hongwei Jiang[2, 4†], Chunyan Liu[4†], Xue Han[4†], Guohua Hu[4†*], Qingshan Chen[1, 2†*]

[1]Key Laboratory of Soybean Biology in Chinese Ministry of Education (key Laboratory of Soybean Biology and Breeding/Genetics of Chinese Agriculture Ministry), Northeast Agricultural University, Harbin 150030, China
[2]Agronomy College, Northeast Agriculture University, Harbin 150030, China
[3]Soybean research institute, Heilongjiang Academy of Agricultural Sciences, Harbin 150086, China
[4]The Crop Research and Breeding Center of Land-Reclamation, Harbin 150090, China
[5]KeShan Branch Academy of Heilongjiang Academy of Agricultural Sciences, KeShan 161606, China

[*]Corresponding authors: qshchen@126.com, hugh757@vip.163.com
†These authors contributed equally to this work

Abstract

SMV3 is one of the SMV strains of northeast China, it has the strongest pathogenicity and can cause serious consequences when it occurs around the large planting area. The objective of this study was to evaluate the genetic regularity of resistance to SMV3 and to map the resistant genes associated with SSR markers for soybean adult-plant and seed coat mottling. Two F_2 populations, the parents, and the relative $F_{2:3}$ populations were used to evaluate the genetic regularity underlying SMV3 resistance. One F_2 population (288 individual plants) was constructed by susceptible cultivar 3C624 crossed with resistant cultivar DongNong 8143, another F_2 population (300 individual plants) was constructed by resistant cultivar DongNong 8143 crossed with susceptible cultivar 3C624. 1000 pairs of SSR molecular markers and genetic analytic software were used to map the resistant genes, 11 SSR markers associated with the resistant genes were mapped successfully. The results indicated that adult-plant resistance to SMV3 of DongNong 8143 was controlled by one single dominant nuclear gene; seed coat mottling resistance to SMV3 were controlled by two genes which were inherited dependent to each other; the resistance of adult-plant and seed coat mottling to SMV3 also inherited jointly, the linkage distance was 22.07cM and 15.79cM, respectively. Two resistant genes of seed coat mottling $R_{SMV3\text{-}S1}$, $R_{SMV3\text{-}S2}$ were named by using Gene Interaction V1.0, they were mapped on the linkage group F and associated with SSR markers. The order and linkage distance between SSR markers and two resistant genes were Satt030-10.1cM-$R_{SMV\text{-}S1}$-11.5cM-Sat_240, Satt114-6.5cM- $R_{SMV\text{-}S2}$-7.5cM-Satt335. This research provides the useful information for breeders to select the two types of SMV3 resistance simultaneously in soybean breeding through molecular marker assisted selection (MAS).

Keywords: Soybean; SMV3; Resistant genes; SSR; MAS.
Abbreviations: SMV_soybean mosaic virus; SMV3_soybean mosaic virus №3 strain; SSR_simple sequence repeat; LG_linkage group; MAS_marker assisted selection.

Introduction

Soybean mosaic virus (SMV) is a common disease of soybean world wide and occurs wherever soybean is grown and causes yield loss and seed quality deterioration in many soybean production areas worldwide (Hunst and Tolin, 1982; Ross, 1983; Hartman et al., 1999). It is a seed-borne disease and propagates by aphids (Hill et al., 1980; Guo and Zhang, 1987). It can also result in seed-coat mottling and seed quality deterioration (Ross, 1983; Hill et al., 1980). There are fewer resistant cultivars for breeder to cultivate new resistant lines or materials, and SMV always occurs in the planting area commonly in China. SMV disease can reduce the yield of soybean (Ross, 1983; Ren et al., 1997), it could decrease the yield of soybean by 10% to 30% in general and 50% to 100% in serious outbreaks (Ross, 1983; Buss et al., 1985). However, the breeders could cultivate the resistant materials to control SMV, and the application of resistant cultivars is the most effective, economical and environment friendly approach in SMV resistant breeding (Fu et al., 2006; Liao et al., 2011).

Soybean mosaic disease was caused by more than one SMV strain (Conover, 1948). In America, 98 isolates of SMV were collected and classified into seven strains, which were designated as G1-G7 on a set of differential cultivars (Cho and Goodman, 1979, 1982). In Japan, there are five SMV strains (A to E) identified (Takahashi et al., 1980). In Korea, a study reported the emergence of SMV isolates capable of overcoming all of the known resistance genes (Choi et al., 2005). In China, SMV has been re-classified into 21 strains based on SMV isolate reactions to a set of soybean differentials (Wang et al., 2003a; Guo et al., 2005; Li et al., 2010). SMV3 is the most virulent strain widespread in Northeastern China, it has the same pathogenicity as other five virulent strains (SC-4, SC-7, SC-8, SC-13 and SC-17), which reported in other studies (Guo et al., 2005; Li et al., 2010). To date, the resistant inheritance of SMV has been studied extensively. Most studies have focused on the resistance of adult-plants to SMV, but in fact there are two SMV resistant traits: adult-plant resistance to SMV, and

resistance to seed coat mottling. Concerning the inheritance of adult-plant resistance, in most studies, the resistance to SMV was identified to be controlled by a single dominant gene (Kiihl and Hartwig, 1979; Buzzell and Tu, 1989; Buss et al., 1989; Chen et al., 1991; Bowers et al., 1992; Wang et al., 1998, 2004; Liao et al., 2002; Zheng et al., 2006; Liao et al., 2011). Other studies showed that resistance to SMV was governed by either a single recessive gene (Kwon and Oh, 1980; Sun et al., 1990) or two complementary genes (Koshimizu and Lizuka, 1963; Liao et al., 1994; Ma et al., 1995, 2002; Chen et al., 1999). Concerning the inheritance of seed-coat resistance, one SMV virulence factor limited the normal expression of the alleles I and i to the hilum color pigment (Wilcox and Laviolette, 1968). Bowers and Goodman (1979) reported that the Merit cultivar was not resistant to the SMV, as they found infectious virus in juvenile seeds. One study showed that the resistance to seed coat mottling was controlled by a single dominant gene (Hu et al., 1995; Li et al., 2008; Wang et al., 2010). The number of resistant genes affected the severity of resistance, the strong resistant parent was only controlled by one single dominant resistance gene, its genetic model was simple, and the weak resistance parent was controlled by one to three resistance genes in the different crosses. This gene model included the gene accumulation and gene interaction. Three independent SMV resistant loci have been identified and reported, which named *Rsv1*, *Rsv3* and *Rsv4* (Kiihl and Hartwig, 1979; Buzzell and Tu, 1989; Chen et al., 1991; Buss et al., 1997, 1999; Hayes et al., 2000). Nine resistant alleles at the *Rsv1* locus have been reported (Chen et al., 1991, 1994, 2001, 2002; Kiihl and Hartwig 1979; Ma et al., 1995, 2003), and two alleles have been identified at the *Rsv3* locus (Buss et al., 1999; Buzzell and Tu 1989). The *Rsv4* locus (Hayes et al., 2000) was identified in a breeding line, LR2, which was released as V94-5152 (Buss et al., 1997). In addition, molecular marker assisted mapping of these resistant genes has been reported. Two RFLP markers (PA186 and PK644a) and one SSR marker (SM176) had been identified (Yu et al., 1994), which were closely linked to *Rsv1*, on the linkage group (LG) F. Subsequently, *Rsv3* and *Rsv4* were also mapped to LG B2 (Jeong et al., 2002) and LG D1b (Hayes et al., 2000), respectively. These molecular markers identified for SMV resistant genes could have the potential to facilitate both marker-assisted selection (MAS) and map-based cloning of resistant genes. In China, seven resistant genes such as *Rsa*, *Rn-1*, *Rn-3*, *Rsc-7*, *Rsc-8*, *Rsc-9* and *Rsc-13*, were localized to LG D1b by using a recombinant inbred lines (RILs) population (Wang et al., 2004; Zhan et al., 2006; Guo et al., 2007). To date, there were no reports to record *Rsc-4* and *Rsc-17*, two resistant genes had the same resistance to SMV3. Two resistant genes named *Rsc-14Q* and *Rsc-11* from Qihuang No.1 were mapped on LG F, respectively (Li et al., 2006; Bai et al., 2009). After the initial gene mapping, the *Rsv1* region was saturated with 38 loci detected by 24 markers using 1056 F_2 individuals (Gore et al., 2002; Hayes et al., 2004). A comparative mapping strategy was used to define an approximately 5 cM region containing *Rsv4* (Hwang et al., 2006), which was later limited to a 1.3 cM region by using the whole-genome shotgun sequence (Saghai-Maroof et al., 2010). Although Northeastern China is the main production area of soybean, there is no information that can be used for the mapping of resistant genes in local soybean cultivars. The objectives of this study were: (i) to determine the inheritance of resistance to SMV in strain SMV3, (ii) to locate the resistant gene R_{SMV3} on the soybean genetic linkage map, which revealed the relationship of adult-plant resistance and resistance to seed coat mottling, and (iii) to identify molecular markers linked to R_{SMV3}.

Results

Inheritance of the adult-plant resistance to SMV3 in two populations

The phenotypic values of the two F_2 populations from the normal and reverse crosses were obtained when inoculated with SMV3 in 2008, respectively, showed in Table 1. Total 288 individual plants were obtained from the normal cross of 3C624 (S) × Dongnong8143 (R), of them 160 individual plants were resistant (R), 42 individual plants were necrotic (N), and 86 individual plants were susceptible (S), they exhibited a ratio of 3(R+N):1S when inoculated with SMV3, the heterogeneity test of this F_2 population showed a good fit to the 3(R+N):1S ratio, the result clearly indicated that Dongnong 8143 showed the inherited mode of the single independent dominant gene for adult-plant resistance to SMV3. Total 300 individual plants were obtained from the reverse cross of Dongnong 8143 (R) ×3C624 (S), of them 128 individual plants were resistant (R), 63 individual plants were necrotic (N), and 109 individual plants were susceptible (S). The ratio did not show as 3(R+N):1S when inoculated with SMV3, the heterogeneity test of this F_2 population also did not exhibit a good fit to the 3(R+N):1S ratio, the result indicated that Dongnong 8143 did not show the inherited mode of adult-plant resistance to SMV3, which controls by the single independent dominant gene. Therefore, these two results showed that the adult-plant resistance to SMV3 showed by Dongnong 8143 could be controlled by nuclear and cytoplasmic genes in common. To identify the inherited mode of the adult-plant resistance to SMV3, progeny testing was carried out on the F_3 lines in 2009. The seeds of the individual plants in F_2 populations from the two crosses were planted and advanced to F_3 lines when inoculated with SMV3, and phenotypic values were obtained, respectively, showed in Table 2. Total 210 lines were obtained from the normal cross of 3C624 (S) × Dongnong8143 (R), of them 50 lines were resistant (R), 100 lines were segregative (Seg.), and 60 individual plants were susceptible (S), they showed a ratio of 1R:2Seg.:1S when inoculated with SMV3, the heterogeneity test of this F_3 population showed a good fit to the 1R:2Seg.:1S ratio, the result clearly demonstrated that the results from F_2 and F_3 populations were consistent basically, and Dongnong 8143 showed the inherited mode of the single independent dominant gene for adult-plant resistance to SMV3. Total 198 lines were obtained from the reverse cross of Dongnong8143 (R) ×3C624 (S), of them 36 lines were resistant (R), 104 lines were segregative (Seg.), and 58 lines were susceptible (S), they showed a ratio of 1R:2Seg.:1S when inoculated with SMV3, the heterogeneity test of this F_3 population also showed a good fit to the 1R:2Seg.:1S ratio, but the result clearly demonstrated that the results from F_2 and F_3 populations were not consistent basically. The reason was that the necrotic plants of F_2 population did not seed, and which could lead to partial segregation. The number of the necrotic plants in F_2 population was more enough, so the situation of the partial segregation was very serious. Therefore, if the necrosis was looked as homozygous resistance in F_3 population, the segregative ratio of resistance and susceptibility in F_3 population from 3C624 (S) × Dongnong8143 (R) was 92 (42+50):100:60, and the segregative ratio of resistance and susceptibility in F_3 population from Dongnong8143 (R) ×3C624 (S) was 99 (63+36):104:58. So the ratios of the normal and the reverse populations tended to be consistent, but they did not fit to the segregative ratio of 1R:2Seg.:1S, these results could demonstrate that the inherited mode of adult-plant resistance to SMV3 from Dongnong8143 was controlled by complicated nuclear genes, and not controlled by cytoplasmic genes.

Table 1. χ^2 analysis of adult-plant resistance to SMV3 in two groups of F_2 population in 2008

Parents and crosses	No. of Plants (lines)†				Expected ratio	χ^2	$\chi^2_{0.05(1)}$
	Total	R	N	S			
3C624	20	0	0	20			
Dongnong8143	20	20	0	0			
Hengfeng No.25 (control)	20	0	0	20			
3C624×Dongnong8143	288	160	42	86	3(R+N):1S	3.19	3.84
Dongnong8143×3C624	300	128	63	109	3(R+N):1S	20.55	3.84

†R, resistant (symptomless); N, necrotic; S, susceptible (mosaic); R+N, plants were combined in segregating rows.

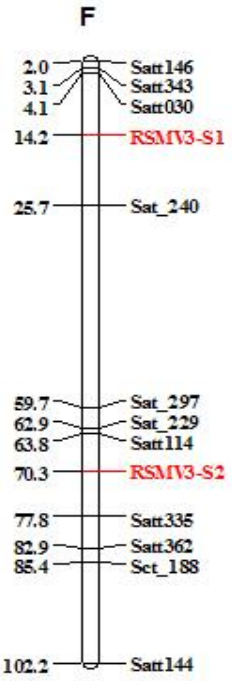

Fig. 1 Positions of $R_{SMV3-S1}$ and $R_{SMV3-S2}$ on soybean linkage group F

Inheritance of the seed coat mottling resistance to SMV3 in two populations

The phenotypic values of the $F_{2:3}$ seeds in the two populations were obtained when harvested in 2008, showed in Table 3. Total 225 shares of seeds were obtained from the normal cross of 3C624 (S) × Dongnong 8143 (R), of them 193 shares were resistant (R), 32 shares susceptible (S), they showed a ratio of 13R:3S, the heterogeneity test of this $F_{2:3}$ population showed a good fit to the 13R:3S ratio, the result indicated that Dongnong 8143 exhibited the seed coat mottling resistance to SMV3 was controlled by two major genes, and inhibitory effect existed between the two genes. Total 213 shares of seeds were obtained from the reverse cross of Dongnong 8143 (R) ×3C624 (S), of them 164 shares were resistant (R), 49 shares susceptible (S), they showed a ratio of 13R:3S, the heterogeneity test of this $F_{2:3}$ population showed a good fit to the 13R:3S ratio, the result showed that Dongnong 8143 exhibited the seed coat mottling resistance to SMV3 was controlled by two genes, and inhibitory effect existed between the two major genes. To identify the inherited mode of the seed coat mottling resistance to SMV3, progeny testing was carried out on the $F_{3:4}$ seeds in 2009 (Table 4). Total 192 shares of seeds were obtained from the normal cross of 3C624 (S) × Dongnong 8143 (R), of them 160 shares were resistant (R), 32 shares susceptible (S), they showed a ratio of 13R:3S, the heterogeneity test of this $F_{2:3}$ population showed a good fit to the 13R:3S ratio, the result indicated that Dongnong 8143 exhibited the seed coat mottling resistance to SMV3 was controlled by two major genes, and inhibitory effect existed between the two genes. Total 178 shares of seeds were obtained from the reverse cross of Dongnong 8143 (R) ×3C624 (S), of them 137 shares were resistant (R), 41 shares susceptible (S), they showed a ratio of 13R:3S, the heterogeneity test of this $F_{2:3}$ population showed a good fit to the 13R:3S ratio, the result showed that Dongnong 8143 exhibited the seed coat mottling resistance to SMV3 was controlled by two major genes, and inhibitory effect existed between these two genes. Therefore, the results in 2008 and 2009 showed the same result that the seed coat mottling resistance to SMV3 showed by Dongnong 8143 could be controlled by two nuclear genes but cytoplasmic genes, the inherited modes of these genes were nuclear inheritance, the two major genes had the inhibitory effect by each other.

Conjoint analysis for the resistance of adult-plant and seed coat mottling to SMV3

The adult-plant resistance of the two F_2 populations and the seed coat mottling resistance of the two $F_{2:3}$ populations were analyzed conjointly between the normal and reverse crosses (Table 5). In the cross of 3C624 (S) × Dongnong 8143 (R), 149 shares exhibited the two kinds of resistant types, 24 shares exhibited the susceptibility of adult-plant and seed coat mottling, 49 shares exhibited different reactions to SMV3; In

Table 2. χ^2 analysis of adult-plant resistance to SMV3 in two groups of F_3 population in 2009.

Parents and crosses	No. of Plants (lines)†				Expected ratio	χ^2	$\chi^2_{0.05(1)}$
	Total	R	Seg.	S			
3C624	20	0	0	20			
Dongnong8143	20	20	0	0			
Hengfeng No.25 (control)	20	0	0	20			
3C624×Dongnong8143	210	50	100	60	1R:2Seg.:1S	1.43	5.99
Dongnong8143×3C624	198	36	104	58	1R:2Seg.:1S	5.39	5.99

†R, resistant (symptomless); N, necrotic; S, susceptible (mosaic); R+N, plants were combined in segregating rows.

Table 3. χ^2 analysis of seed coat mottling resistance to SMV3 in $F_{2:3}$ lines in 2008.

Parents and crosses	No. of Plants (lines)†			Expected ratio	χ^2	$\chi^2_{0.05(1)}$
	Total	R	S			
3C624	20	0	20			
Dongnong8143	20	20	0			
Hengfeng No.25 (control)	20	0	20			
3C624×Dongnong8143	225	193	32	13R:3S	0.40	3.84
Dongnong8143×3C624	213	164	49	13R:3S	2.49	3.84

†R, resistant (symptomless); S, susceptible (mosaic).

Table 4. χ^2 analysis of seed coat mottling resistance to SMV3 in $F_{3:4}$ lines in 2009.

Parents and crosses	No. of Plants (lines)†			Expected ratio	χ^2	$\chi^2_{0.05(1)}$
	Total	R	S			
3C624	20	0	20			
Dongnong8143	20	20	0			
Hengfeng No.25 (control)	20	0	20			
3C624×Dongnong8143	192	160	32	13R:3S	0.55	3.84
Dongnong8143×3C624	178	137	41	13R:3S	2.14	3.84

†R, resistant (symptomless); S, susceptible (mosaic).

the cross of Dongnong 8143 (R) ×3C624 (S), 146 shares exhibited the two kinds of resistant types, 30 shares exhibited the susceptibility of adult-plant and seed coat mottling, 33 shares exhibited different reactions to SMV3. The recombination rats of the two genotypes were 22.07% and 15.79%, respectively. The results indicated that the phenotypes of the two crosses were consistent basically, the adult-plant resistance and the seed coat mottling resistance were controlled by different major genes, and these genes were linkage inheritance.

Molecular mapping of seed coat mottling resistant genes

The principle: seed coat mottling resistance was controlled by two major genes through genetic analysis, and in order to identify the genetic distance of the two major genes associated with SSR markers, the band types of SSR markers were looked as genotypes to be analyzed in pairs, and the interactions between genes were considered to calculate the genotypic rates. If the genotypic rates could match the theoretic genotypic rates by calculating the interactions between some kinds of genes, and the coincidence rates were highest between the phenotypic values and the genotypic values after comparing in pairs, so the two SSR markers could be considered as associated with the two major genes closely. 1000 SSR markers were used to screen the polymorphism between the two parents of the two crosses as well as R/S BSA. Among these SSR markers, Satt343, Sat_240, Sat_297, Sat_229, Sct_188, Satt114, Satt146, Satt030, Satt335, Satt362, and Satt144 were screen to be polymorphic and be associated with the seed mottling resistance genes in Dongnong 8143. These SSR markers located on linkage group (LG) F, and the order and the genetic distance could be expressed as follows (Figure 1):
Satt146-1.1cM-Satt343-1.0cM-Satt030-21.6cM-Sat_240-34.0cM-Sat_297-3.2cM-Sat_229-0.9cM-Satt114-14.0cM-Satt335-5.1cM-Satt362-2.5cM-Sct_188-16.8cM- Satt144. The band types of these 11 SSR markers could be looked as the genotypes, and used the software of Gene Interaction V1.0 to analysis, the results were exhibited in Table 6. The marker pair of Satt030 (4.0cM)-Satt114 (63.7cM) had the highest coincidence rate between the genotypic values and the phenotypic values in all the primer groups under the condition of inhibitory effect, the heterogeneity test of R/S showed a good fit to the 13R:3S ratio. The primer groups in or out of the interval of the primer group of Satt030 (4.0cM)-Satt114 (63.7cM) had either the lower coincidence rate or the bad heterogeneity test values. So it was confirmed that the primers of Satt030 (4.0cM) and Satt114 (63.7cM) were the SSR markers tightly associated with seed coat mottling resistant genes to SMV3 in Dongnong 8143, $R_{SMV3\text{-}S1}$ (resistant gene of seed coat mottling to SMV3) was named as the resistant gene associated with Satt030 (4.0cM), and $R_{SMV3\text{-}S2}$ was named as the resistant gene associated with Satt114 (63.7cM). The genetic distance between $R_{SMV3\text{-}S1}$ and Satt030 (4.0cM) was obtained by calculating the coincidence rates between the markers near Satt030 (4.0cM) and Satt114 (63.7cM), it was 10.1cM; and the genetic distance between $R_{SMV3\text{-}S2}$ and Satt114 (63.7cM) was obtained by calculating the coincidence rates between the markers near Satt114 (63.7cM) and Satt030 (4.0cM), it was 6.5cM. The two genes of $R_{SMV3\text{-}S1}$ and $R_{SMV3\text{-}S2}$ were mapped on LG F, the positions and genetic distances of them were showed as follows (Figure 1): Satt030-10.1cM-$R_{SMV3\text{-}S1}$-11.5cM-Sat_240 and Satt114-6.5 cM-$R_{SMV3\text{-}S2}$-7.5cM- Satt335.

Discussion

Genetic regularity of the resistance to SMV3

The resistance to SMV was classified into two styles, adult-plant resistance and seed coat mottling resistance, which

Table 5. Conjoint analysis for adult-plant and seed coat mottling resistance to SMV3.

Crosses	Genotypes†				Recombination rate (%)
	RR	SS	RS	SR	
3C624×Dongnong8143	149	24	42	7	22.07
Dongnong8143×3C624	146	30	17	16	15.79

†RR: adult-plant resistance and seed coat mottling resistance.
SS: adult-plant susceptibility and seed coat mottling susceptibility.
RS: adult-plant resistance and seed coat mottling susceptibility.
SR: adult-plant susceptibility and seed coat mottling resistance.

Table 6. Analysis of gene interaction with selected SSR markers.

Primer groups	Coincidence rate	Resistance genotypes	Susceptible genotypes	R/S ratio	χ^2	$\chi^2_{0.05(1)}$
Satt030-Satt114	85.3%	163	28	13R:3S	2.19	3.84
Satt343-Satt114	81.2%	167	24	13R:3S	4.93	3.84
Sat_240-Satt114	84.7%	169	20	13R:3S	8.24	3.84
Satt030-Sat_229	69.1%	149	32	13R:3S	0.14	3.84
Satt030-Satt335	68.6%	151	37	13R:3S	0.11	3.84
Satt343-Satt335	76.2%	152	37	13R:3S	0.08	3.84
Sat_240-Sat_229	69.3%	149	30	13R:3S	0.52	3.84

was controlled by different genes (Hu et al., 1995; Wang et al., 2010). Most studies had researched the inheritance of the adult-plant resistance to SMV, and the results demonstrated that the inheritance of resistance to SMV was controlled by a single dominant gene (Roane et al., 1983; Buzzell and Tu, 1989; Lim, 1985; Bowers et al., 1992; Wang et al., 2003; Zhan et al., 2006; Guo et al., 2007; Bai et al., 2009; Wang et al., 2010; Liao et al., 2011). But due to the specificity of genetic materials and the diversity of SMV strains, different results had been reported by lots of researchers using various SMV strains and different soybean cultivars. The inheritance of resistance to SMV has been reported to be controlled by a single recessive gene (Sun et al., 1990), two dominant suppressive genes (Liao et al., 1994), two dominant complementary genes (Luan, 1997; Chen, 1999), or by two recessive complementary genes (Liao et al., 1994). In this study, the inheritance of the adult-plant resistance to SMV strain SMV3 was also controlled by a single dominant gene in Dongnong8143, which was complicated nuclear gene, it was same as the known studies.

Classification of stem tip necrosis

The standards of different classification for the symptoms had undoubtably influenced the phenotypic ratios and genetic interpretations. The classification of necrosis as R or S has been a controversial issue. Some studies had classified stem tip necrosis as an S reaction (Pu et al., 1982; Lim, 1985; Hu et al., 1995), and some reports had classified stem tip necrosis as a hypersensitive R reaction (Kiihl and Hartwig, 1979; Buzzell and Tu, 1989), some studies classified stem tip necrosis as a single style and inheritance was calculated by 3(R+N):1S ratio (Fu et al., 2006; Zheng et al., 2006). In this study, stem tip necrosis was classified as R reaction, like the latter researchers, and the inheritance of resistance to SMV3 was controlled by single dominant gene, so different classification could obtain different results (Sun et al., 1990; Liao et al., 1994; Luan, 1997; Chen, 1999). No matter how to classify stem tip necrosis as R or S reactions, the inheritance of resistant genes can rely on the types that stated on the above, so these results could provide us useful examples when the study about the inheritance of resistance to SMV can be started to research.

Resistant genes associated with molecular markers

The inheritance of seed coat mottling resistance to SMV had already been studied (Bowers and Goodman, 1992; Hu et al., 1995; Domier et al., 2007, 2011; Wang et al., 2010), and most researcher focused on the adult-plant resistance to SMV, but confirming the mechanism of the inheritance of seed coat mottling resistance to SMV could also be a very meaningful work for soybean breeders, because the outside quality of soybean seeds can influence the commercial value directly. Therefore, in China, especially in northeast China, many researchers have started to research the inheritance of seed coat mottling resistance to SMV. In this study, two resistant gene named $R_{SMV3\text{-}S1}$ and $R_{SMV3\text{-}S2}$ were mapped on LG F (chromosome 13), linkage with Satt030 and Satt114, respectively. Many SMV resistant genes had been mapped on LG F, Yu et al. (1994) found that two RFLP markers (PA186 and PK644a) and one SSR marker (SM176) were closely linked to *Rsv1*, on the molecular linkage group (MLG) F; R_{SC14Q} and R_{SC11} from Qihuang No.1 were mapped to LG F (chromosome 13) by Li et al. (2006) and Bai et al. (2009), respectively. Wang et al. (2010) found two SSR markers closely linkage with seed coat mottling resistant genes *DSRSMV1*-4.6cM-Sat_317 and Satt516-2.0cM-*TSRSMV1*, these markers were located on LG F. Ma et al. (2010) identified the resistance gene $R_{SC\text{-}12}$ from Qihuang 22 was located on linkage group F between the SSR markers Satt334 and Sct_033. Hayes et al. (2000) reported that resistant genes to SMV always existed in clusters, this opinion could be examined by the studies from above. Therefore, $R_{SMV3\text{-}S1}$ and $R_{SMV3\text{-}S2}$ could be confirmed to be two novel resistant genes to SMV, and these resistant genes were not at the same locus. Research using uniform strains would clarify whether there is allelism between these genes.

MAS for resistant breeding to SMV3

The ideal markers used in MAS are based on PCR by considering experimental cost and technical feasibility. In the previous reports, most of the markers used to locate the resistant genes to SMV were RAPD and RFLP markers (Yu et al., 1994; Hayes et al., 2000; Zheng et al., 2001; Jeong et al., 2002; Wang et al., 2004; Zhang et al., 2006), which were applied limitedly in breeding program for their poor repeatability, complicated operation and high labor and time consumed. The co-dominant SSR markers Satt030 and Satt114 identified in this research, closely linked to the seed coat mottling resistant genes to SMV3, could be used to screen the

homozygous resistant plants in the early generations by convenient experimental operation. Furthermore, the SSR markers used in MAS showed high efficiency. Therefore, it is feasible for these two SSR markers to be used as a tool in SMV3 resistant breeding program.

Materials and Methods

Plant genetic materials

The SMV3-susceptible soybean cultivar, 3C624, and the SMV3-resistant cultivar, Dongnong8143, were obtained from Northeast Agricultural University in Harbin, China. The normal cross, 3C624 × Dongnong8143, and the reverse cross, Dongnong8143×3C624, were made in the field in 2006. F_1 plants for producing an F_2 population were grown without virus inoculation in an aphid-free net-house in the field at the Crop Research and Breeding, Land-Reclamation Base of Heilongjiang Province, China, in 2008, 288 F_2 individual plants and 300 F_2 individual plants were obtained for the two crosses, respectively. All F_2 plants were harvested individually for developing $F_{2:3}$ seeds, 225 $F_{2:3}$ share of seeds and 213 $F_{2:3}$ share of seeds were obtained for the two crosses, respectively. The $F_{2:3}$ seeds from the two crosses were planted in the field at the Crop Research and Breeding, Land-Reclamation Base of Heilongjiang Province, China, in 2009, 222 F_3 individual plants and 211 F_3 individual plants were obtained for the two crosses, respectively. All F_3 plants were harvested individually for developing $F_{3:4}$ seeds, 197 $F_{3:4}$ share of seeds and 180 $F_{3:4}$ share of seeds were obtained for the two crosses, respectively. The phenotype of seeds from $F_{2:3}$ and $F_{3:4}$ lines were considered to be influenced by F_2 and F_3 generation, respectively, so the adult-plant resistance and resistance to seed coat mottling were investigated in F_2 and F_3 plants, $F_{2:3}$ and $F_{3:4}$ seeds (Table 1-Table 4). The cultivar, Hengfeng No.25, as a control and the parents were also planted with the two populations in two years.

SMV strain and inoculation identification

The SMV strain SMV3 was preserved on the SMV susceptible cultivar Hefeng No.25 in an aphid-free greenhouse and conserved by soybean research institute of Northeast Agriculture University and soybean research institute of Heilongjiang Academy of Agricultural Sciences. The inoculum was prepared by grinding young symptomatic leaves with a mortar and pestle at a ratio of 1:2 (w/v) in 0.1 M sodium phosphate buffer, pH 7.0. A small amount of 600-mesh carborundum was added to the inoculum (Roane et al., 1983). Young plants in aphid-proof net house were inoculated by gently rubbing the newly unfolded primary leaves with inoculum at V1 stage, then by re-inoculation on the first trifoliate leaf at V2 stage (Fehr and Caviness, 1977). The inoculated leaves were gently rinsed with tap water after inoculation. Twenty plants of the control and the parents were inoculated and other twenty plants were not inoculated as controls. Pesticides were sprayed 7 days interval to exterminate aphides. For the field inoculation, about 0.2 mL of inoculum was applied to the underside of a single leaflet per plant (stages V1-V3) by using an artist's airbrush for 1 s from a distance of 1 to 2 cm. Air pressure was maintained at 4.2 kg to 5.6 kg cm^{-2} (60-80 p.s.i.) by a gasoline-powered portable compressor. The temperature was maintained at 20 ^{o}C to 30 ^{o}C for the duration of the test. The first observations were taken 15-30 days after the first inoculation; at the same time the plants with symptoms were marked to avoid disturbance from latency of symptoms. Observations were made again 3 days later. Plants were classified as resistant (R): no symptoms appeared on leaves above those inoculated, no matter what happened to inoculated leaves; as susceptible (S): mosaic or necrotic symptoms occurred on leaves above those inoculated no matter how severe the symptoms were; as necrotic (N): stem tip necrosis. In this study, (R+N) was looked as the resistant style, and S was looked as the susceptible style. Seed-coat mottling resistance identification for parents was to calculate the mottled ratio of the seed surface. For the two populations, samples of 100 seeds were evaluated from the individual plants to determine the percentage of seed coat discoloration for a soybean seed sample. Any seed showing brown or black seed coat discoloration was counted as mottled. In $F_{2:3}$ population, 100 seeds were selected in the individual plants randomly, and the average mottled ratio of 100 seeds were looked as the phenotypic values of the individual plants according to calculate the mottled ratio of single seed. In $F_{3:4}$ population, 100 seeds were selected in the individual planting lines randomly, and the average mottled ratio of 100 seeds were looked as the phenotypic values of the individual planting lines according to calculate the mottled ratio of single seed. The mottled ratio of the resistant style was smaller than 5%, and the mottled ratio of the susceptible style was bigger than 5%. Chi-square tests were used to determine the goodness-of-fit of observed segregations to expected genetic ratios and the homogeneity of different populations from the same type crosses. A Chi-square test for heterogeneity was also performed to examine whether different populations from the same type of cross displayed similar genetic behavior. Genetic models were proposed according to inheritance of single, independent and dominant genes, *Rsv1*, *Rsv3*, and *Rsv4*, at three separate loci. Determination of the inheritance of each locus was based on the results of F_2, F_3, $F_{2:3}$ and $F_{3:4}$ segregation ratios observed after inoculation with SMV3 in 2008 and 2009.

DNA extraction and SSR marker analysis

Genomic DNAs of the RIL individuals and the two parents were extracted by using the CTAB (cetyltrimethyl-ammonium bromide) method (Saghai-Maroof et al., 1984). 1000 SSR primers were synthesized according to sequences published on the SOYBASE website (http://soybase.agron. iastate. edu/ssr.html). PCR was conducted in a total reaction mixture of 10 μl including approximately 50 ng of genomic DNA, 10× PCR buffer (500 mmol/L KCl, 100 mmol/L Tris-HCl, pH 8.0, 0.1% gelatin), 0.25 μmol/L of each primer, 0.2 mmol/L dNTPs and 1 U Taq polymerase in double distilled water. PCR reaction conditions were as follows: denaturation at 94 ºC for 5 min; 30 cycles of denaturation at 94 ºC for 30 s, annealing at 55 ºC for 50 s, extension at 72 ºC for 50 s; and a final extension at 72 ºC for 10 min before cooling to 4 ºC. PCR products were visualized after electrophoresis on an 6% polyacrylamide gel followed by silver staining; or on a 1% agarose gel followed by ethidium bromide staining. SSR primers were screened by Bulked Sergeants Analysis (BSA) in the resistant (R) and susceptible (S) pools from each F_2 and F_3 population with their resistant parents and susceptible parents (Michelmore et al. 1991). Resistant parents and susceptible parents were screened by SSR primers at first, then, polymorphic primers were chosen to screen in R and S pools. R pool consisted of equal amounts of genomic DNA from 20 plants which are homozygous resistant (R) and S pool consisted of equal amounts of genomic DNA from 20 plants which are homozygous susceptible (S). Those SSR primers, showed polymorphism between the parents and also between the R and S pools, were further used to amplify the F_2 and F_3 population to map the resistance genes.

Linkage analysis

Soybean lines for which gel band profiles of the PCR products were consistent with those of the resistant parent (Dongnong8143) were designated as "1", and those consistent with the susceptible parent (3C624) were designated as "2". the hybrid band type was designated as "3", and unclear or deleted bands were designated as "0". Individual resistant to the SMV3 strain was designated as "1", and susceptible to the SMV3 strain as "2". The genetic map of SSR molecular markers and the resistant loci was constructed from the above "1" and "2" data by the MAPMAKER/EXP 3.0b computer program (Lander et al. 1987). Map distances were obtained by the Kosambi function. The molecular map was constructed by the MapChart 2.1 software. In order to map the resistant genes, which had the highest rate of coincidence, the phenotypic values and the genotypic values should be compared and calculated by using the software of Gene Interaction V1.0 (self-created by the lab, national patent authorized), according to the results of the resistance of adult-plant and seed coat mottling to SMV3.

Acknowledgment

This work were Supported by Doctoral Fund of Ministry of Education of China, Trans-Century Training Programme Foundation for the Talents by the State Education Commission, Supported by China Postdoctoral Science Foundation, National Natural Science Foundation of China (General Program; Key Program; Major Program);

References

Bai L, Li H, Ma Y, Wang D, Liu N, Zhi H (2009) Inheritance and gene mapping of resistance to soybean mosaic virus strain SC-11 in soybean. Soybean Sci. 28:1-6.

Bowers GR, Goodman RM (1979) Soybean mosaic virus: Infection of soybean seed parts and seed transmission. Phytopathology. 69:569-572.

Bowers GR, Paschal ES, Bernard RL, Goodman RM (1992) Inheritance of resistance to soybean mosaic virus in 'Buffalo' and HLS soybean. Crop Sci. 32:67-72.

Buss GR, Roane CW, Tolin SA (1985) Breeding for resistance to virus in soybean. *In* R. Shibles (ed.) Proc. World Soybean Res. Conf. III, Ames, IA. 12-17 Aug. 1984. Westview Press, Boulder and London.

Buss GR, Roane CW, Tolin SA, Chen P (1989) Inheritance of reaction to soybean mosaic virus in two soybean cultivars. Crop Sci. 29:1439-1441.

Buss GR, Ma G, Chen P, Tolin SA (1997) Registration of V94-5152 soybean germplasm resistant to soybean mosaic potyvirus. Crop Sci. 37:1987-1988.

Buss GR, Ma G, Kristipati S, Chen P, Tolin SA (1999) A new allele at the Rsv3 locus for resistance to soybean mosaic virus. In: Proceedings of the World Soybean Research Conference VI (Kauffman HE, ed). Champaign, IL: Superior Printing; 490.

Buzzell RI, Tu JC (1989) Inheritance of a soybean stem-tip necrosis reaction to soybean mosaic virus. J Hered. 80:400-401.

Chen P, Buss GR, Roane CW, Tolin SA (1994) Inheritance in soybean of resistant and necrotic reactions to soybean mosaic virus strains. Crop Sci. 34:414-422.

Chen P, Ma G, Buss GR, Gunduz I, Roane CW, Tolin SA (2001) Inheritance and allelism tests of Raiden soybean for resistance to soybean mosaic virus. J Hered. 92:51-53.

Chen P, Buss GR, Roane CW, Tolin SA (1991) Allelism among genes for resistance to soybean mosaic virus in straindiff erential soybean cultivars. Crop Sci. 31:305-309.

Chen P, Buss GR, Tolin SA, Gunduz I, Clcek M (2002) A valuable gene in Suweon 97 soybean for resistance to soybean mosaic virus. Crop Sci. 42:333-337.

Chen Y (1999) Inheritance of resistance to SMV strain no.1 in the soybean germplasms. Heilongjiang Agri Sci. 1:4-6.

Chen Y, Du W, Man W, Zhang G (1999) An evaluation and screening of soybean germplasms resistant to soybean mosaic virus. Soybean Sci. 18:32-36.

Cho EK, Goodman RM (1979) Strains of soybean mosaic virus: Classification based on virulence in resistant soybean cultivars. Phytopathology. 69: 467-470.

Cho EK, Goodman RM (1982) Evaluation of resistance in soybeans to soybean mosaic virus strains. Crop Sci. 22:1133-1136.

Choi BK, Koo JM, Ahn HJ, Yum HJ, Choi CW, Ryu KH, Chen PY, Tolin SA (2005) Emergence of Rsv-resistance breaking soybean mosaic virus isolates from Korean soybean cultivars. Virus Res. 112:42-51.

Conover RA (1948) Studies of two viruses causing mosaic disease of soybean. Phytopathology. 58:724-734.

Domier LL, Hobbs HA, McCoppin NK, Bowen CR, Steinlage TA, Chang S, Hartman GL (2011) Multiple loci condition seed transmission of soybean mosaic virus (SMV) and SMV-induced seed coat mottling in soybean. Phytopathology. 101:750-756.

Domier LL, Steinlage TA, Hobbs HA, Wang Y, Herrera-Rodriguez G, Haudenshield JS, McCoppin NK, Hartman GL (2007) Similarities in seed and aphid transmission among Soybean mosaic virus isolates. Plant Dis. 91:546-550.

Fehr WR, Caviness CE (1977) Stages of soybean development. Iowa State University Special Report. 80:1-12.

Fu SX, Zhan Y, Zhi HJ, Gai JY, Yu DY (2006) Mapping of SMV resistance gene Rsc-7 by SSR markers in soybean. Genetica. 128:63-69.

Gore MA, Hayes AJ, Jeong SC, Yue YG, Buss GR, Saghai-Maroof MA (2002) Mapping tightly linked genes controlling potyvirus infection at the Rsv1 and Rpv1 region in soybean. Genome. 45:592-599.

Guo DQ, Wang YW, Zhi HJ, Gai JY, Li HC, Li K (2007) Inheritance and gene mapping of resistance to SMV strain group SC-13 in soybean. Soybean Sci. 26:21-24.

Guo DQ, Zhi HJ, Wang YW, Gai JY, Zhou XA, Yang CL, Li K, Li HC (2005) Identification and distribution of Soybean mosaic virus strains in Middle and Northern Huang Huai Region of China. Chin J Oil Crop Sci. 27:64-68.

Guo JQ, Zhang MH (1987) Study on the important vectors of soybean mosaic virus and their transmission efficiencies. Soybean Sci. 8:55-63.

Hartman GL, Sinclair JB, Rupe JC (1999) Compendium of soybean diseases, 4th ed. St Paul, MN: Am Phytopathology Soc.

Hayes AJ, Jeong SC, Gore MA, Yu YG, Buss GR, Tolin SA, Saghai-Maroof MA (2004) Recombination within a nucleotide-bindingsite/leucine-rich-repeat gene cluster produces new variants conditioning resistance to soybean mosaic virus in soybeans. Genetics. 166:493-503.

Hayes AJ, Ma G, Buss GR, Saghai-Maroof MA (2000) Molecular marker mapping of Rsv4, a gene conferring resistance to all known strains of soybean mosaic virus. Crop Sci. 40:1434-1437.

Hill JH, Lucas BS, Benner HI, Tachibana H, Hammond RB, Pedigo LP (1980) Factors associated with the epidemiology of soybean mosaic virus in Iowa. Phytopathology.

70:536-540.
Hu GH, Wu ZP, Gao FL (1995) Studies for genic effect of resistance to seed coat mottling on soybean. Acta Genetica Sin. 22:133-141.
Hunst PL, Tolin SA (1982) Isolation and comparison of two strains of soybean mosaic virus. Phytopathology. 72:710-713.
Hwang TY, Moon JK, Yu S, Yang K, Mohankumar S, Yu YH, Lee YH, Kim HS, Kim HM, Saghai-Maroof MA (2006) Application of comparative genomics in developing molecular markers tightly linked to the virus resistance gene Rsv4 in soybean. Genome. 49:380-388.
Jeong SC, Kristipati S, Hayes AJ, Maughan PJ, Noffsinger SL, Gunduz I, Buss GR, Saghai-Maroof MA (2002) Genetic and sequence analysis of markers tightly linked to the soybean mosaic virus resistance gene, Rsv3. Crop Sci. 42:265-270.
Kiihl RAS, Hartwig EE (1979) Inheritance of reaction to soybean mosaic virus in soybean. Crop Sci. 19:372-375.
Koshimizu Y, Lizuka T (1963) Studies on soybean virus diseases in Japan. Nat Agric Exp Stn. 27:1-104.
Kwon SH, Oh JH (1980) Resistance to a necrotic strain of soybean mosaic virus in soybean. Crop Sci. 20:403-404.
Lander ES, Green P, Abrahamson J, Barlow A, Daley M, Lincoln S, Newburg L (1987) MAPMAKER, an interactive computer package for constructing primary genetic linkage maps of experimental and natural populations. Genomics. 1:174-181.
Li HC, Zhi HJ, Gai JY, Guo DQ, Wang YW, Li K, Bai L, Yang H (2006) Inheritance and gene mapping of resistance to soybean mosaic virus strain SC14 in soybean. J Integr Plant Biol. 48:1466-1472.
Li K, Yang QH, Zhi HJ, Gai JY (2010) Identification and distribution of soybean mosaic virus strains in southern china. Plant Dis. 94:351-357.
Li WF, Liu CY, Gao YL, Li CD, Jiang HW, Wang K, Chen QS, Hu GH (2008) Genetic analysis and mapping of resistance gene to seed coat mottle in soybean. Acta Agron Sin. 34:1544-1548.
Liao L, Chen P, Rajcan I, Buss GR, Tolin SA (2011) Genetic analysis of "8101" soybean containing three genes for resistance to soybean mosaic virus. Crop Sci. 51:503-511.
Liao L, Chen P, Buss GR, Yang Q, Tolin SA (2002) Inheritance and allelism of resistance to soybean mosaic virus in Zao 18 soybean from China. J Hered. 93:447-452.
Liao L, Liu Y, Sun D, Zhao Y, Tian P (1994) Inheritance of soybean resistance to soybean mosaic virus: I. Inheritance of several resistance varieties introduced resistance to SMV No. 2. Acta Genet Sin. 21:403-408.
Lim SM (1985) Resistance to soybean mosaic virus in soybeans. Phytopathology. 75:199-201.
Luan XY (1997) Inheritance of soybean plant resistant to no III (173) strain of soybean mosaic virus. Soybean Sci. 16:223-226.
Ma G, Chen P, Buss GR, Tolin SA (1995) Genetic characteristics P of two genes for resistance to soybean mosaic virus in PI 486355 soybean. Theor Appl Genet. 91:179-184.
Ma G, Chen P, Buss GR, Tolin SA (2002) Complementary action of two independent genes in Columbia soybean for resistance to soybean mosaic virus. J Hered. 93:179-184.
Ma G, Chen P, Buss GR, Tolin SA (2003) Genetic study of a lethal necrosis to soybean mosaic virus in PI 507389 soybean. J Hered. 94: 205-211.
Ma Y, Li H, Wang D, Liu N, Zhi H (2010) Molecular mapping and marker assisted selection of soybean mosaic virus resistance gene RSC12 in soybean. Legum Genomics Genet. 8:41-46.
Michelmore RW, Paran I, Kesseli RV (1991) Identification of markers linked to disease-resistance genes by bulked segregant analysis: a rapid method to detect markers in specific genomic regions using segregating populations. Proc Natl Acad Sci. 88:9828-9832.
Pu ZC, Cao Q, Fang DC, Xie BT, Fang ZD (1982) Classification of soybean mosaic virus strains. Acta Phytophylacica Sin. 9:15-20.
Ren Q, Pfeiffer TW, Ghabrial SA (1997) Soybean mosaic virus incidence level and infection time: Interaction effects on soybean. Crop Sci. 37:1706-1711.
Roane CW, Tolin SA, Buss GR (1983) Inheritance of reaction to two viruses in the soybean cross 'York'×'Lee 68'. J Hered. 74:289-291.
Ross JP (1983) Effect of soybean mosaic virus on component yields from blends of mosaic resistant and susceptible soybean lines. Crop Sci. 17:869-872.
Saghai-Maroof MA, Tucker DM, Skoneczka JA, Bowman BC, Tripathy S, Tolin SA (2010) Fine mapping and candidate gene discovery of the soybean mosaic virus resistance gene, Rsv4. Plant genome. 3:14-22.
Saghai-Maroof MA, Soliman KM, Jorgenson RA, Allard RW (1984) Ribosomal DNA spacer-length polymorphisms in barley: Mendelian inheritance, chromosomal location and population dynamics. Proc Natl Acad Sci. 81: 8014-8018.
Sun Zh, Liu Y, Sun D, Liao L (1990) Inheritance of resistance to SMV No. 1, No. 2, and No. 3 in soybean. Chin J Oil Crop Sci. 2:20-24.
Takahashi K, Tanaka T, Iida W, Tsuda Y (1980) Studies on virus disease and causal viruses of soybean in Japan. Natl Agric Exp Stn. 62:1-130.
Wang K, Li WF, Zhang L, Liu CY, Zhu XS, Che QS, Hu GH (2010) Genetic Analysis and Molecular Mapping of Two SMV-Resistance Traits in Soybean: Adult-Plant Resistance and Resistance to Seed Coat Mottling. Agr Sci China. 9:11-18.
Wang XQ, Gai JY, Pu ZQ (2003a) Classification and distribution of strains of soybean mosaic virus in middle and lower Huanghuai and Changjiang river valleys. Soybean Sci. 22:102-107.
Wang XQ, Gai JY, Yu DY (2003) Inheritance of resistance to the SMV strain group SC-8 in Kefeng No.1. Soybean Sci. 22: 190-192.
Wang Y, Nelson RL, Hu Y (1998) Genetic analysis of resistance to soybean mosaic virus in four soybean cultivars from China. Crop Sci. 38:922-925.
Wang Y, Yang DF, Wang X, Yang Y, Yu D, Gai J, Wu X, He Ch, Zhang J, Chen Sh (2004) Mapping of five genes for resistance to SMV strains in Soybean. Acta Genet Sin. 31:87-90.
Wilcox JR, Laviolette FA (1968) Seed coat mottling response of soybean genotypes to infection with soybean mosaic virus. Phytopathology. 58:1446-1447.
Yu GY, Saghai-Maroof MA, Buss GR, Maughan P, Tolin S (1994) RFLP and microsatellite mapping of a gene for soybean mosaic virus resistance. Phytopathology. 84:60-64.
Zhan Y, Yu DY, Chen SY, Gai JY (2006) Inheritance and gene mapping of resistance to SMV strain SC-7 in soybean. Acta Agron Sin. 32:936-938.
Zheng CM, Chang RZ, Qiu LJ (2001) Inheritance of resistance to SMV3 and identification of RAPD marker linked to the resistance gene in soybean. Sci Agri Sin. 34:1-4.
Zheng C, Chen P, Gergerich R (2006). Genetic analysis of resistance to soybean mosaic virus in J05 soybean. J Hered. 97:429-437.