Fine mapping of the major *Soybean dwarf virus* resistance gene *Rsdv1* of the soybean cultivar ‘Wilis’

Yoko Yamashita^{*1)}, Toru Takeuchi¹⁾, Shizen Ohnishi²⁾, Jun Sasaki²⁾ and Akiko Tazawa³⁾

¹⁾ Hokkaido Research Organization Central Agricultural Experiment Station, Higashi 6 Kita 15, Naganuma, Hokkaido 069-1395, Japan

²⁾ Hokkaido Research Organization Kitami AES, 52 Yayoi, Kunneppu, Hokkaido 099-1496, Japan

³⁾ Hokkaido Research Organization Tokachi AES, S9-2 Shinsei, Memuro, Hokkaido 082-0071, Japan

Soybean dwarf virus (SbDV), a *Luteoviridae* family member, causes dwarfing, yellowing and sterility of soybean (*Glycine max*), leading to one of the most serious problems in soybean production in northern Japan. Previous studies revealed that the Indonesian soybean cultivar ‘Wilis’ is resistant to SbDV and that the resistance can be introduced into Japanese cultivars. A major QTL for SbDV resistance has been reported between SSR markers Sat_217 and Satt211 on chromosome 5. In this study, we named this QTL *Rsdv1* (resistance to SbDV) and developed near-isogenic lines incorporating *Rsdv1* (*Rsdv1*-NILs) using Sat_217 and Satt211 markers. The *Rsdv1*-NILs were resistant to SbDV in greenhouse inoculation and field tests, indicating that *Rsdv1* alone is sufficient for the resistance phenotype. We fine-mapped *Rsdv1* within the 44-kb region between Sat_11 and Sct_13. None of the six genes predicted in this region was closely related to known virus resistance genes in plants. Thus, *Rsdv1* may confer resistance by a previously unknown mechanism. We suggest that *Rsdv1* may be a useful source for the Japanese soybean breeding program to introduce SbDV resistance.

Key Words: disease resistance, *Glycine max*, near-isogenic lines, SSR markers.

Introduction

Soybean dwarf virus (SbDV), a member of the family *Luteoviridae*, causes one of the most serious diseases of soybean (*Glycine max*) in Hokkaido and the northern Tohoku region of Japan (Tamada 1975, Tamada *et al.* 1969). SbDV has also spread in most soybean-producing states in the USA (Damsteegt *et al.* 2011). SbDV infection in legumes other than soybean has been reported in Australia, Ethiopia, Germany, New Zealand and Tunisia (Abraham *et al.* 2007, Johnstone and McLean 1987, Najar *et al.* 2003, Tadesse *et al.* 1999, Wilson and Close 1973).

On the basis of the disease symptoms and vector specificity, SbDV is subdivided into four strains (Terauchi *et al.* 2001), with SbDV-DS and SbDV-YS being prevalent in Hokkaido (Tamada *et al.* 1969). SbDV-DS-infected soybeans become dwarfed, with shortened internodes and petioles and dark, brittle leaves curling downward. The yellowing strain, SbDV-YS, causes interveinal chlorosis and thickened, brittle mature leaves. Both DS and YS strains are transmitted by the foxglove aphid, *Aulacorthum solani* (Tamada 1970), whereas the less prevalent DP and YP strains are transmitted by the pea aphid, *Acyrtosiphon*

pisum (Honda *et al.* 1999). SbDV causes not only a considerable decrease in yield but also low quality of the harvested seeds, because infected plants remain green and seedless, staining seeds during harvest. The most effective way to control SbDV is through breeding and growing resistant cultivars.

Approximately 3100 soybean germplasms were collected and screened for SbDV resistance from 1966 to 1981; two moderately field-resistant cultivars, ‘Ouhouju’ and ‘Adams’, were found (Tanimura *et al.* 1982). ‘Ouhouju’ has been used to develop moderately field-resistant cultivars such as ‘Tsurukogane’, ‘Tsurumusume’ and ‘Iwaikuro’, which develop milder symptoms (Banba *et al.* 1985, Nakamura *et al.* 1991, Shirai *et al.* 2000) but which still suffer yield loss (Tanimura and Banba 1987). ‘Adams’ exhibits antibiosis to the foxglove aphid (Jinno *et al.* 1997). However, the QTL from ‘Adams’ conferring aphid resistance is insufficient for SbDV tolerance and pesticide application is required to prevent yield loss (Kamiya *et al.* 2008, Ohnishi *et al.* 2012).

Additional screening of East and Southeast Asian germplasms in the 1990s revealed that the Indonesian cultivar ‘Wilis’ is resistant to SbDV. ‘Wilis’ develops mild symptoms at a very late stage in the field (Tazawa *et al.* 2008) and in greenhouse inoculation tests (Uchibori *et al.* 2009). However, it does not reach maturity in northern Japan, and continues vigorous vegetative growth during the harvest of local cultivars. To test whether the resistance from ‘Wilis’ can be

Communicated by M. Ishimoto

Received June 12, 2013. Accepted October 24, 2013.

*Corresponding author (e-mail: yamashita-yoko@hro.or.jp)

introduced into Japanese cultivars, Tazawa *et al.* (2008) crossed 'Wilis' and an early-maturing Japanese cultivar, 'Karafuto-1', to develop 'Shokukei-32', which is resistant to SbDV and matures early. This indicates that resistance derived from 'Wilis' is not closely linked to nor results from a pleiotropic effect of the gene(s) controlling the maturity date.

Analysis of recombinant inbred lines (RILs) from a cross between 'Wilis' and a susceptible Japanese cultivar, 'Toyokomachi', detected a single QTL for SbDV resistance in the 7.3-cM region between Sat_217 and Satt211 on chromosome 5; this QTL had a LOD score of 23.8 and accounted for 79% of the resistance phenotype (Uchibori *et al.* 2009). These data suggest that a single major gene derived from 'Wilis' confers SbDV resistance.

Here, we named this QTL *Rsdv1* (resistance to *Soybean dwarf virus*) and investigated whether *Rsdv1* alone is sufficient to confer the resistance phenotype. We also fine-mapped the gene within a 44-kb region on chromosome 5, in which six genes are predicted.

Materials and Methods

Plant materials

Resistant cultivars 'Wilis' and 'Shokukei-32' (Tazawa *et al.* 2008) and susceptible Japanese cultivars 'Toyokomachi' and 'Chukei-413' were used in resistance tests. Near-isogenic lines carrying *Rsdv1* in the 'Toyokomachi' background (*Rsdv1*-NILs) were obtained by five backcrosses of 'Toyokomachi' (the recurrent parent) and 'Wilis' (*Rsdv1* donor). The SSR markers Sat_217 and Satt211 were used for *Rsdv1* introduction. For fine mapping, 'Shokukou-0001' F₆ lines derived from a cross between 'Toyokomachi' and 'Wilis', and 'Chukou-1640' F₃ lines from a cross between 'Chukei-413' and 'Shokukou-0001' F₅ (resistant to SbDV) were used.

Agronomic trait analysis

The *Rsdv1*-NILs were grown in an experimental field of the Hokkaido Central Agricultural Experiment Station in Takikawa-shi. Seeds were sown on 19 May 2009 at 15-cm intervals in rows 66 cm apart. Each line was grown in five 1.98 m² plots, with 20 plants per plot. Maturity date, plant height, 100-seed weight and hilum color of 15 plants from each of the five plots were evaluated and means were determined.

Greenhouse inoculation test for SbDV resistance

The greenhouse inoculation test was performed as previously described (Uchibori *et al.* 2009), except that inoculation with SbDV-YS-infested aphids was 3 days. Resistance to SbDV was evaluated on the basis of the time between virus inoculation and initial symptom appearance (days after inoculation, DAI).

Field tests for SbDV resistance

Field tests were performed in 2006, 2009 and 2010 in an experimental field of the Hokkaido Central Agricultural Experiment Station in Date-shi, a city with a severe endemic infection of SbDV-DS and SbDV-YS. No other virus diseases were observed in soybean in this region. Seeds were sown on 16 May 2006, 7 May 2009 and 7 May 2010 at 20-cm intervals in rows 66 cm apart. For evaluation of the SbDV resistance of 'Shokukou-0001', each line was grown in two randomly allocated 0.66 m² plots, with five plants per plot, in 2006. For evaluation of the resistance of the *Rsdv1*-NILs, each *Rsdv1*-NIL was grown in four randomly allocated 2.64 m² plots, with 20 plants per plot, in 2009 and 2010. Plants with dwarfing or yellowing symptoms were counted and the percentage of symptomatic plants (%SP) was calculated on 15 August 2006, 27 August 2009 and 28 August 2010.

DNA extraction and marker analysis

DNA was extracted from young leaves by a modified CTAB method (Suzuki *et al.* 2012). Both published and newly designed SSR markers were used (Table 1). To design SSR markers, we identified SSRs from the Williams 82 soybean genome sequence Glyma01 (Schmutz *et al.* 2010) and selected SSRs which showed polymorphism between 'Chukei-413' and 'Shokukou-0001' F₅, the parents of the mapping population. PCR was performed according to Suzuki *et al.* (2012) with the following modifications: M13 primer (5'-CACGACGTTGTAAAACGAC-3') fluorescently labeled with either 6-FAM, VIC, NED or PET was added to a final concentration of 0.1 µM; the final concentration of SSR primers was 0.2 µM. A 19-nucleotide 5' M13 tail (as above) was added to each forward primer according to Schuelke (2000). PCR products were analyzed in an ABI Prism 310 Genetic Analyzer (Applied Biosystems) using the GeneScan software and GeneScan-500 LIZ as the size standard.

Results

SbDV resistance and agronomic traits of the *Rsdv1*-NILs

In the greenhouse test, five *Rsdv1*-NILs developed the initial symptoms at 33.5–35.3 DAI, or 6.5–8.3 days later than 'Toyokomachi' (Fig. 1), whereas 'Wilis' developed the initial symptoms at 50.7 DAI. In the two field tests (2009 and 2010), the average %SP was 0.5% for the resistant cultivar 'Shokukei-32' and 25.9% for the susceptible cultivar 'Toyokomachi'. The average %SP of the *Rsdv1*-NILs (1.7%–7.5%) was significantly lower than that of 'Toyokomachi' and did not differ significantly from that of 'Shokukei-32' (Table 2). We considered these NILs to be resistant to SbDV because of the ~7-day delay in the initial symptom appearance and low %SP. Agronomic traits of the *Rsdv1*-NILs were similar to those of 'Toyokomachi'. The hilum color in all NILs and 'Toyokomachi' was yellow (not brown as in 'Shokukei-32'). The maturity date, plant height

Table 1. The markers on chromosome 5 of soybean cultivar ‘Williams 82’ and primer sequences

Marker	Location (kb) ^a	Forward primer (5' to 3')	Reverse primer (5' to 3')	Reference ^b
CSTS11	38,497	ACGCGTCCGCTCACTA	GCACCACTCATGTCTTGGA	Xia <i>et al.</i> 2007
Sat_217	38,670	GCGAAAAATTGTCAATGATATGATCAGTAAG	GCGGTCCTAGATGAAAAATGCTTTGTAA	Song <i>et al.</i> 2004
Sat_06	38,683	AAAGTGTGAAAAATAAATCCTTTCT	GTTACTATTACACAATTAATAACTTCA	
Sat_11	38,704	TCTTTGTTTACCTTCTTTGTGA	AAAAAGCCACATATTTTCA	
CA782298	38,735	ATTCGCGTTCCTCTTCTCTCT	GCTGTGATTGAGGGAGAAGCTT	Xia <i>et al.</i> 2007
Stat04	38,740	GAGTTGGCAGATTGATCGTA	GAATACAAAAGCCCATTGTG	
Sact14	38,744	TAGCCGAGCTTGTATAGCAC	ATATTAGATGTGGGGCGAAG	
Sct_13	38,747	CATCTTCAACTAAATGTTTGCAC	TCTGGGAATTCATAACTGGT	
Sat_08	38,755	GAAACTCACAAATCAAAGTATGG	CCTGACACATAGTAAAGGGAAC	
Sat_02	38,798	CAGATTTTATAAGTCCCTGGAA	ACGTAAGAGAAAAGCTTAAATAAAAA	
Stta01	38,816	AGGGAGGACAAAGAAACAAG	ATAAAGCACGGTTGTTGTGT	
Sat_271	38,832	GCGTCACTTTAATCATTACAATAAAACAACA	GCGACTTAGGCACACTCTAATATAACCA	Song <i>et al.</i> 2004
GM038	39,079	CATTATAAATATAGTTGACG	CTCATTATTTTGTGGATGTTG	Xia <i>et al.</i> 2007
Satt211	39,926	GAAAAAGCCCACATCCAA	CATGGGCATGCAGTAACA	Cregan <i>et al.</i> 1999

^a Data from the Phytozome database (<http://www.phytozome.net/soybean.php>, Schmutz *et al.* 2010).

^b Markers without references are newly designed in this study.

Table 2. Characteristics of *Rsdv1*-NILs

	Maturity date	Plant height (cm)	100-seed weight (g)	Hilum color	Initial symptom appearance (DAI) ^a	Field tests (%SP) ^b			Genotype ^c	
						2009	2010	Average	Sat_217	Satt211
<i>Rsdv1</i> -NIL-1	20 Sep bc	47.3 b	35.0 de	Yellow	33.7 a	8.1	3.6	5.9 a	R	R
<i>Rsdv1</i> -NIL-2	22 Sep c	51.0 cd	35.8 e	Yellow	33.9 a	1.1	5.1	3.1 a	R	R
<i>Rsdv1</i> -NIL-3	22 Sep c	53.4 d	34.8 cde	Yellow	33.6 a	11.5	3.5	7.5 a	R	R
<i>Rsdv1</i> -NIL-4	16 Sep a	48.7 bc	33.2 bc	Yellow	33.5 a	2.3	5.0	3.7 a	R	R
<i>Rsdv1</i> -NIL-5	18 Sep ab	47.6 b	33.5 bcd	Yellow	35.3 a	2.3	1.1	1.7 a	R	R
Wilis	–	114.0 e	–	–	50.7 b	–	–	–	R	R
Shokukei-32	22 Sep c	36.7 a	22.3 a	Brown	–	0.0	0.9	0.5 a	R	R
Toyokomachi	18 Sep a	48.6 bc	32.8 b	Yellow	27.0 a	21.9	29.9	25.9 b	S	S

Values with the same letter (a–e) within each column are not significantly different (Tukey–Kramer, $P < 0.05$).

^a Days after virus inoculation.

^b Percentage of symptomatic plants.

^c R (‘Wilis’ type) and S (‘Toyokomachi’ type), lines homozygous for the respective alleles.

Fig. 1. Soybeans 28 days after inoculation with SbDV-YS. While the resistant line ‘*Rsdv1*-NIL-1’ (A) remained symptomless, the susceptible cultivar ‘Toyokomachi’ (B) developed interveinal chlorosis and thick, embrittled leaves.

Table 3. Graphical genotypes and SbDV resistance of ‘Shokukou-0001’ (‘Toyokomachi’ × ‘Wilis’) recombinants

	%SP (2006) ^a	Phenotype	Genotype ^b					
			CSTS11	Sat_217	CA782298	Sat_271	GM038	Satt211
Shokukou-0001-13	0	R	R	R	R	R	R	S
Shokukou-0001-31	0	R	R	R	R	R	R	S
Shokukou-0001-41	0	R	R	R	R	R	R	S
Shokukou-0001-44	0	R	S	S	R	R	R	R
Shokukou-0001-53	10	R	R	R	R	R	S	S
Shokukou-0001-7	40	S	S	S	S	S	S	R
Shokukou-0001-52	40	S	S	S	S	S	S	R
Shokukou-0001-27	50	S	S	S	S	S	R	R
Shokukou-0001-68	50	S	S	S	S	S	S	R
Shokukou-0001-26	60	S	S	S	S	S	S	R
Wilis	0	R	R	R	R	R	R	R
Toyokomachi	90	S	S	S	S	S	S	S

^a Percentage of symptomatic plants.^b R (‘Wilis’ type) and S (‘Toyokomachi’ type), lines homozygous for the respective alleles.

and 100-seed weight of NIL-4 and NIL-5 were also similar to those of ‘Toyokomachi’ (Table 2).

Fine mapping of Rsdv1 on chromosome 5

Recombinations between Sat_217 and Satt211 were observed in 10 out of 289 F₆ lines of ‘Shokukou-0001’. The recombinants were genotyped with four additional markers, and their phenotypes were evaluated by the field test. The %SP of the resistant cultivar was 0%, and that of the susceptible cultivar was 90%. Five recombinants, with a %SP of 0%–10%, were considered resistant, and the other five, with %SP >10%, susceptible. Comparison of the phenotypes and graphical genotypes revealed that *Rsdv1* lies between markers Sat_217 and GM038 (Table 3).

To further elucidate the position of *Rsdv1*, we screened 1213 F₃ lines of ‘Chukou-1640’ for recombinants between Sat_217 and GM038. Seven informative lines were genotyped with 10 additional markers. In the greenhouse inoculation test, ‘Wilis’ was used as the resistant control because it was the *Rsdv1* donor for ‘Shokukou-0001 F₅’, the resistant parent of ‘Chukou-1640’. The symptoms first appeared in ‘Chukou-1640-242’, followed by the other lines within 14.4 days (Table 4). The time of the initial symptom appearance was distributed continuously among the recombinants. Because *Rsdv1* incorporation delays the initial symptom appearance by approximately 7 days (Table 2), we considered lines that showed symptoms at >7 days later than ‘Chukou-1640-242’ to be resistant to SbDV. Two resistant lines, ‘Chukou-1640-581’ and ‘Chukou-1640-1208’ (but not the other five), developed symptoms significantly more slowly than ‘Chukei-413’. Comparison of the phenotypes and graphical genotypes suggests that *Rsdv1* lies between markers Sat_11 and Sct_13 (Table 4), within a 44-kb region. Six protein-coding sequences, *Glyma05g34320–34370*, are predicted within this region according to the Phytozome database (Table 5).

Table 4. Graphical genotypes and SbDV resistance of ‘Chukou-1640’ (‘Chukei-413’ × ‘Shokukou-0001 F₅’) recombinants

	Initial symptom appearance (Days) ^{a,b}	Phenotype	Genotype ^c									
			Sat_217	Sat_06	Sat_11	CA782298	Satt04	Satt14	Sct_13	Sat_08	Satt_02	Satt01
Chukou-1640-581	14.4**	R	R	R	R	R	R	R	R	R	S	S
Chukou-1640-1208	7.1*	R	R	R	R	R	R	R	R	R	R	S
Chukou-1640-125	2.8	S	S	S	S	S	S	S	R	R	R	R
Chukou-1640-1196	4.1	S	S	S	S	S	S	S	S	R	R	R
Chukou-1640-334	0.7	S	S	S	S	S	S	S	S	S	R	R
Chukou-1640-298	2.2	S	R	S	S	S	S	S	S	S	S	S
Chukou-1640-242	0.0	S	R	R	R	S	S	S	S	S	S	S
Wilis	20.0**	R	R	R	R	R	R	R	R	R	R	R
Chukei-413	1.2	S	S	S	S	S	S	S	S	S	S	S
Toyokomachi	1.0	S	S	S	S	S	S	S	S	S	S	S

^a Days from the initial symptom appearance in ‘Chukou-1640-242’.^b Significant differences from ‘Chukei-413’ by Tukey–Kramer test at *5%, **1%.^c R (‘Shokukou-0001 F₅’ type) and S (‘Chukei-413’ type), lines homozygous for the respective alleles.

Discussion

The initial symptoms appeared in *Rsdv1*-NILs approximately 7 days later than in ‘Toyokomachi’ and 17 days earlier than in ‘Wilis’. Although both the NILs and ‘Wilis’ start developing symptoms at similar growth stages, the NILs mature earlier than ‘Wilis’, which may explain why they showed the symptoms earlier. In order to eliminate the effects of early maturation on resistance evaluation, we used ‘Shokukei-32’ (which matures similarly to the NILs) as the resistant control in the field tests. The %SP of the NILs was similar to that of ‘Shokukei-32’. Both the greenhouse and field test results led us to conclude that the *Rsdv1*-NILs are

Table 5. Predicted genes in the *Rsdv1* region (based on ‘Williams 82’ genome)

Locus	Predicted gene product/putative function	Location (kb) ^a
Glyma05g34320	26S Proteasome regulatory subunit RPN2/PSMD1	38,695–38,702
Glyma05g34330	THO complex subunit 2 (transcription- and export-related)	38,709–38,729
Glyma05g34340	Clathrin assembly protein (phospholipid-binding)	38,729–38,730
Glyma05g34350	Ribosomal protein S9	38,731–38,733
Glyma05g34360	Mitochondrial import receptor subunit TOM40	38,734–38,740
Glyma05g34370	PLATZ transcription factor (zinc ion binding)	38,744–38,746

^a Data from the Phytozome database (Schmutz *et al.* 2010).

resistant to SbDV. We speculate that the 7-day delay in the greenhouse test contributed to the low %SP in the field tests (Table 2). The *Rsdv1*-NILs in the background of another Japanese cultivar (‘Toyomusume’) also showed low %SP in field tests (our unpublished work). These results clearly indicate that *Rsdv1* alone is necessary and sufficient for SbDV resistance. The NILs developed in this study have better agronomic traits (such as yellow hilum color and larger seed size) than ‘Shokukei-32’ (Table 2) and find more extensive use in our SbDV-resistant soybean breeding program.

Rsdv1 was located between Sat_217 and Satt211 on chromosome 5 (Uchibori *et al.* 2009). We further mapped this region by using ‘Shokukou-0001’ and ‘Chukou-1640’ recombinants between Sat_11 and Sct_13 (Tables 3, 4), where six genes are predicted (Table 5). One of the predicted genes, *Glyma05g34320*, encodes the 26S proteasome regulatory subunit RPN2/PSMD1. In *Human cytomegalovirus*-infected human cells, RPN2 accumulates in the viral DNA replication center (Tran *et al.* 2010). In *Nicotiana benthamiana*, RPN9 (another 26S proteasome subunit) inhibits the systemic transport of *Tobacco mosaic virus* and *Turnip mosaic virus* (Jin *et al.* 2006). Thus, *Glyma05g34320* may play a role in SbDV infection in soybean.

Another *Rsdv1* candidate gene, *Glyma05g34360*, is predicted to encode the mitochondrial import receptor subunit TOM40. TOM40 is required for replication of *African swine fever virus* in cultured human cells (Chang *et al.* 2006). No specific relation has been suggested between TOM40 and virus replication in plants. The other four genes have not been reported to be specifically associated with resistance to viruses.

In plants, the genes that control gene-for-gene resistance encode proteins with coiled-coil (CC) domains, nucleotide-binding sites (NBS) and C-terminal leucine-rich repeats (LRRs) or serine/threonine kinases (reviewed by Bent 1996). *Rpg-1b*, a soybean gene for resistance to *Pseudomonas syringae*, encodes a protein of the CC-NBS-

LRR class (Ashfield *et al.* 2003). NBS-LRR type resistance genes are also likely to mediate resistance to *Soybean mosaic virus* at the *Rsv1* locus (Hayes *et al.* 2004). No such genes are predicted within the 44-kb *Rsdv1* region in the Phytozome database, which is based on ‘Williams 82’. This region may be different in ‘Wilis’ and ‘Williams 82’. As none of the six candidate genes has been directly linked to resistance to plant viruses, further studies are required to identify *Rsdv1* and to reveal the details of its role in SbDV resistance.

Acknowledgements

This research was supported by the Ministry of Agriculture, Forestry and Fisheries of Japan (Genomics for Agricultural Innovation DD-3211) and in part by JSPS KAKENHI Grant 23780007.

Literature Cited

- Abraham, A.D., W. Menzel, H.J. Vetten and H. Saucke (2007) First report of *Soybean dwarf virus* (genus *Luteovirus*) infecting faba bean and clover in Germany. *Plant Dis.* 91: 1059.
- Ashfield, T., A. Bocian, D. Held, A.D. Henk, L.F. Marek, D. Danesh, S. Penuela, K. Meksem, D.A. Lightfoot, N.D. Young *et al.* (2003) Genetic and physical localization of the soybean *Rpg1-b* disease resistance gene reveals a complex locus containing several tightly linked families of NBS-LRR genes. *Mol. Plant Microbe Interact.* 16: 817–826.
- Banba, H., Y. Tanimura, I. Matsukawa, T. Ushirogi, Y. Mori and I. Chiba (1985) A new soybean variety “Tsuru-kogane”. *Bull. Hokkaido Prefect. Agric. Exp. Stn.* 52: 53–64.
- Bent, A.F. (1996) Plant disease resistance genes: function meets structure. *Plant Cell* 8: 1757–1771.
- Chang, A.C.Y., L. Zsak, Y. Feng, R. Mosseri, Q. Lu, P. Kowalski, A. Zsak, T.G. Burrage, J.G. Neilan, G.F. Kutish *et al.* (2006) Phenotype-based identification of host genes required for replication of African swine fever virus. *J. Virol.* 80: 8705–8717.
- Cregan, P.B., T. Jarvik, A.L. Bush, R.C. Shoemaker, K.G. Lark, A.L. Kahler, N. Kaya, T.T. VanToai, D.G. Lohnes, J. Chung *et al.* (1999) An integrated genetic linkage map of the soybean genome. *Crop Sci.* 39: 1464–1490.
- Damsteegt, V.D., A.L. Stone, M. Kuhlmann, F.E. Gildow, L.L. Domier, D.J. Sherman, B. Tian and W.L. Schneider (2011) Acquisition and transmissibility of U.S. *Soybean dwarf virus* isolates by the soybean aphid, *Aphis glycines*. *Plant Dis.* 95: 945–950.
- Hayes, A.J., S.C. Jeong, M.A. Gore, Y.G. Yu, G.R. Buss, S.A. Tolin and M.A. Saghai Maroof (2004) Recombination within a nucleotide-binding site/leucine-rich-repeat gene cluster produces new variants conditioning resistance to soybean mosaic virus in soybeans. *Genetics* 166: 493–503.
- Honda, K., S. Kanematsu and Y. Mikoshiba (1999) Dwarfing strain of soybean dwarf luteovirus transmitted by *Neuraphis bakeri* and *Acyrtosiphon pisum*. *Ann. Phytopathol. Soc. Jpn.* 65: 387.
- Jin, H., S. Li and A. Villegas Jr. (2006) Down-regulation of the 26S proteasome subunit RPN9 inhibits viral systemic transport and alters plant vascular development. *Plant Physiol.* 42: 651–661.
- Jinno, H., K. Araki, K. Kira and Y. Shinada (1997) Breeding highly tolerant lines to Soybean dwarf virus; with foxglove aphid resistance.

- Report of the Hokkaido Branch, the Japanese Society of Breeding and Hokkaido Branch, the Crop Science Society of Japan 38: 112–113.
- Johnstone, G.R. and G.D. McLean (1987) Virus diseases of subterranean clover. *Ann. Appl. Biol.* 110: 421–440.
- Kamiya, M., J. Sasaki, M. Saitou, O. Kanehira, Y. Hashimoto, T. Miyoshi, Y. Tanaka, F. Kousaka, S. Ohnishi, A. Tazawa *et al.* (2008) QTL analysis and marker development of resistance in soybean to *Aulacorthum solani*, the aphid vector of *Soybean dwarf virus* (SbDV). *Breed. Res.* 10 (Suppl. 1): 146.
- Najar, A., S.G. Kumari, K.M. Makkouk and A. Daaloul (2003) A survey of viruses affecting faba bean (*Vicia faba*) in Tunisia includes first record of *Soybean dwarf virus*. *Plant Dis.* 87: 1151.
- Nakamura, S., H. Banba, I. Matsukawa, Y. Tanimura, T. Adachi and K. Suzuki (1991) A new soybean variety “Tsurumusume”. *Bull. Hokkaido Prefect. Agric. Exp. Stn.* 63: 71–82.
- Ohnishi, S., N. Miyake, T. Takeuchi, F. Kousaka, S. Hiura, O. Kanehira, M. Saito, T. Sayama, A. Higashi, M. Ishimoto *et al.* (2012) Fine mapping of foxglove aphid (*Aulacorthum solani*) resistance gene *Rasol* in soybean and its effect on tolerance to *Soybean dwarf virus* transmitted by foxglove aphid. *Breed. Sci.* 61: 618–624.
- Shirai, K., S. Hagihara, F. Kousaka, H. Banba, S. Nakamura, K. Murata, K. Suzuki, Y. Takamiya, I. Matsukawa and T. Adachi (2000) A new black seeded soybean variety “Iwaikuro.” *Bull. Hokkaido Prefect. Agric. Exp. Stn.* 78: 39–58.
- Schuelke, M. (2000) An economic method for fluorescent labeling of PCR fragments. *Nat. Biotechnol.* 18: 233–234.
- Schmutz, J., S.B. Cannon, J. Schlueter, J. Ma, T. Mitros, W. Nelson, D.L. Hyten, Q. Song, J.J. Thelen, J. Cheng *et al.* (2010) Genome sequence of the palaeopolyploid soybean. *Nature* 463: 178–183.
- Song, Q.J., L.F. Marek, R.C. Shoemaker, K.G. Lark, V.C. Concibido, X. Delannay, J.E. Specht and P.B. Cregan (2004) A new integrated genetic linkage map of the soybean. *Theor. Appl. Genet.* 109: 122–128.
- Suzuki, T., M. Sato and T. Takeuchi (2012) Evaluation of the effects of five QTL regions on *Fusarium* head blight resistance and agronomic traits in spring wheat (*Triticum aestivum* L.). *Breed. Sci.* 62: 11–17.
- Tadesse, N., K. Ali, D. Gorfu, A. Abraham, A. Lencho, M. Ayalew, A. Yusuf, K.M. Makkouk and S.G. Kumari (1999) First report of soybean dwarf virus infecting lentil and beet western yellows virus infecting lentil and chickpea crops in Ethiopia. *Plant Dis.* 83: 589.
- Tamada, T. (1970) Aphid transmission and host range of soybean dwarf virus. *Ann. Phytopathol. Soc. Japan* 36: 266–274.
- Tamada, T. (1975) Studies on the soybean dwarf disease. *Rep. Hokkaido Pref. Agric. Exp. Stn.* 25: 1–144.
- Tamada, T., T. Goto, I. Chiba and T. Suwa (1969) Soybean dwarf, a new virus disease. *Ann. Phytopathol. Soc. Japan* 35: 282–285.
- Tanimura, Y. and H. Banba (1987) Breeding for resistance to soybean dwarf virus (SDV) V. Degree of resistance of a new variety “Tsurukogane”. *Bull. Hokkaido Prefect. Agric. Exp. Stn.* 56: 83–92.
- Tanimura, Y., I. Matsukawa, I. Chiba and H. Banba (1982) The research of resistant varieties to soybean dwarf virus disease (SDVD). Miscellaneous Publication of Hokkaido Prefectural Agricultural Experiment Stations No. 13.
- Tazawa, A., H. Jinno, M. Tezuka, T. Miyoshi, F. Kousaka and Y. Tanaka (2008) Development of soybean breeding line Shokukei-32 with high resistance to soybean dwarf virus disease derived from WILIS. *Bull. Hokkaido Prefect. Agric. Exp. Stn.* 92: 51–60.
- Terauchi, H., S. Kanematsu, K. Honda, Y. Mikoshiba, K. Ishiguro and S. Hidaka (2001) Comparison of complete nucleotide sequences of genomic RNAs of four *Soybean dwarf virus* strains that differ in their vector specificity and symptom production. *Arch. Virol.* 146: 1885–1898.
- Tran, K., J.A. Mahr and D.H. Spector (2010) Proteasome subunits re-localize during human cytomegalovirus infection, and proteasome activity is necessary for efficient viral gene transcription. *J. Virol.* 84: 3079–3093.
- Uchibori, A., J. Sasaki, T. Takeuchi, M. Kamiya, A. Tazawa, T. Inukai and C. Masuta (2009) QTL analysis for resistance to *Soybean dwarf virus* in Indonesian soybean cultivar Wilis. *Mol. Breed.* 23: 323–328.
- Xia, Z., Y. Tsubokura, M. Hoshi, M. Hanawa, C. Yano, K. Okamura, T.A. Ahmed, T. Anai, S. Watanabe, M. Hayashi *et al.* (2007) An integrated high-density linkage map of soybean with RFLP, SSR, STS, and AFLP markers using a single F₂ population. *DNA Res.* 14: 257–269.
- Wilson, J. and R.C. Close (1973) Subterranean clover red leaf virus and other legume viruses in Canterbury. *N.Z. J. Agric. Res.* 16: 305–310.