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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, Acyrthosiphon 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, ‘Ouhoju’ and ‘Adams’, were found (Tanimura et al. 1982). ‘Ouhoju’ 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
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 Sat217 and Sat211 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 Sat217 and Sat211 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 F3 (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 soybean genome sequence Glyma01 (Schmutz et al. 2010) and selected SSRs which showed polymorphism between ‘Chukei-413’ and ‘Shokukou-0001 F3’, the parents of the mapping population. PCR was performed according to Suzuki et al. (2012) with the following modifications: M13 primer (5′-CACGACGTGTAAAACGAC-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...
Fine mapping of the Soybean dwarf virus resistance gene Rsdv1

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

| Marker | Location (kb) | Forward primer (5’ to 3’) | Reverse primer (5’ to 3’) | Reference
|--------|---------------|---------------------------|---------------------------|-----------|
| CSTS11 | 38,497        | ACGGTTGACCTCACTA          | GCACACTGCTTGCTA           | Xia et al. 2007 |
| Sat_217| 38,670        | GCGAAAAATTGCAATATGATGATGTAAG | GCGGTCTGATGAAATATGCTTGTAA | Song et al. 2004 |
| Sat_06 | 38,683        | AAAGTTGAAATATAAGCCTTTT    | GCACTATACAAATATTATAACCTTCA |           |
| Sat_11 | 38,704        | TCTTGTTCCTCTTTTCT         | AAAAGCCCATATTTTCCA        |           |
| CA782298 | 38,735    | ATTCGTTGACCTCACTA          | GCGGTCTGATGAAATATGCTTGTAA | Xia et al. 2007 |
| Sat04  | 38,740        | GACTTCTGAGCTATGTA          | GATAACAAAGCCCTTGTG        |           |
| Sat14  | 38,744        | TAGGCTGACCTGATAGCAC        | ATATTAGTGTGGGCAAG         |           |
| Set_13 | 38,747        | CATCTTCAATATAATGCTTCGAC   | TCTGGAATTCTAATACTG      |           |
| Sat_09 | 38,755        | GACTTCTGAGCTATGTA          | GATAACAAAGCCCTTGTG        |           |
| Sat_02 | 38,798        | CATCTTTCATAAGCCTT          | AGTAAAGAAGAAACCTTTAAAA   |           |
| Stta01 | 38,816        | AGGGAGGAGAAAAGAAGAGAAG    | ATAAAAGCACGCGTGGTGTTG |           |
| Sat_271| 38,832        | GCGTACTTAAATCATTACAATGACAA | GCGGTCTGAGCCTCCTAAAAACACAA | Song et al. 2004 |
| GM038  | 39,079        | CATTTTCTAGCTGATGAAG        | CCTTCTGAGAGGGGAAC         |           |
| Stat211| 39,926        | GAAAAGGCACCACATTTCAA       | CATGCGCATGCAGTAACA        | Cregan et al. 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.

Maturity date | Plant height (cm) | 100-seed weight (g) | Hilum color | Initial symptom appearance (DAI) | Field tests (%SP) | Genotype
---|-----------------|---------------------|-------------|-------------------------------|-----------------|---------|
Rsdv1-NIL-1 | 20 Sep bc      | 47.3 b              | 35.0 de     | Yellow                        | 33.7 a           | R       |
Rsdv1-NIL-2 | 22 Sep c       | 51.0 cd             | 35.8 e      | Yellow                        | 33.9 a           | R       |
Rsdv1-NIL-3 | 22 Sep c       | 53.4 d              | 34.8 cde    | Yellow                        | 33.6 a           | R       |
Rsdv1-NIL-4 | 16 Sep a       | 48.7 bc             | 33.2 bc     | Yellow                        | 35.3 a           | R       |
Rsdv1-NIL-5 | 18 Sep ab      | 47.6 b              | 33.5 bcd    | Yellow                        | 35.3 a           | R       |
Wilis        | –              | 114.0 e             | –           | Yellow                        | 50.7 b           | R       |
Shokukei-32  | 22 Sep c       | 36.7 a              | 22.3 a      | Brown                         | –               | R       |
Toyokomachi  | 18 Sep a       | 48.6 bc             | 32.8 b      | Yellow                        | 27.0 a           | 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.
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 F$_6$ 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 F$_3$ 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 F$_5$’, 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).

### 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 3. Graphical genotypes and SbDV resistance of ‘Shokukou-0001’ (‘Toyokomachi’ × ‘Wilis’) recombinants |
| %SP (2006)$^a$ | Phenotype | Genotype$^b$ |
|----------------|-----------|-------------|
| Shokukou-0001-13 0 | R | R |
| Shokukou-0001-31 0 | R | R |
| Shokukou-0001-41 0 | R | R |
| Shokukou-0001-44 0 | R | S |
| Shokukou-0001-53 10 | R | R |
| Shokukou-0001-7 40 | S | S |
| Shokukou-0001-52 40 | S | S |
| Shokukou-0001-27 50 | S | S |
| Shokukou-0001-68 50 | S | S |
| Shokukou-0001-26 60 | S | S |
| Wilis 0 | R | R |
| Toyokomachi 90 | S | S |

$^a$ Percentage of symptomatic plants.

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

| Table 4. Graphical genotypes and SbDV resistance of ‘Chukou-1640’ (‘Chukei-413’ × ‘Shokukou-0001 F$_5$’) recombinants |
| Initial symptom appearance (Days)$^a$ | Phenotype | Genotype$^b$ |
|----------------|-----------|-------------|
| Chukou-1640-581 14.4** | R | R |
| Chukou-1640-1208 7.1* | R | R |
| Chukou-1640-125 2.8 | S | S |
| Chukou-1640-1196 4.1 | S | S |
| Chukou-1640-334 0.7 | S | S |
| Chukou-1640-298 2.2 | S | S |
| Chukou-1640-242 0.0 | S | S |
| Wilis 20.0** | R | R |
| Chukei-413 1.2 | S | S |
| Toyokomachi 1.0 | 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 F$_5$’ type) and S (‘Chukei-413’ type), lines homozygous for the respective alleles.
Table 5. Predicted genes in the Rsdv1 region (based on ‘Williams 82’ genome)

| Locus         | Predicted gene product/putative function | Location (kb) |
|---------------|------------------------------------------|---------------|
| Glyma05g34320 | 26S Proteasome regulatory subunit RN2/PSMD1 (transcription- and export-related) | 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 |

*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 RN2/PSMD1. In Human cytomegalovirus-infected human cells, RN2 accumulates in the viral DNA replication center (Tran et al. 2010). In Nicotiana benthamiana, RN9 (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.

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