Marker-assisted Selection for Coupling Phase Resistance to Tomato spotted wilt virus and Phytophthora infestans (Late Blight) in Tomato

Matthew D. Robbins
Department of Horticulture and Crop Science, The Ohio State University, Ohio Agricultural Research and Development Center, 1680 Madison Avenue, Wooster, OH 44691

Mohammed A.T. Masud
Vegetable Division, Horticulture Research Centre, Bangladesh Agricultural Research Institute, Joydebpur, Gazipur-1701, Bangladesh

Dilip R. Panthee
Department of Horticultural Science, North Carolina State University, Mountain Horticultural Crops Research and Extension Center, 455 Research Drive, Mills River, NC 28759

Randolph G. Gardner
Department of Horticultural Science, North Carolina State University, Mountain Horticultural Crops Research and Extension Center, 455 Research Drive, Mills River, NC 28759

David M. Francis
Department of Horticulture and Crop Science, The Ohio State University, Ohio Agricultural Research and Development Center, 1680 Madison Avenue, Wooster, OH 44691

Mikel R. Stevens
Department of Plant and Wildlife Sciences, Brigham Young University, 287 WIDB, Provo, UT 84602

Abstract. Tomato spotted wilt virus (TSWV) and Phytophthora infestans (late blight) in tomato (Solanum lycopersicum) have a worldwide distribution and are known to cause substantial disease damage. Sw-5 (derived from S. peruvianum) and Ph-3 (derived from S. pimpinellifolium) are, respectively, TSWV and late blight resistance genes. These two genes are linked (within 5 cM on several maps) in repulsion phase near the telomere of the long arm on chromosome 9. The tomato lines NC592 (Ph-3) and NC946 (Sw-5) were crossed to develop an F2 population and subsequent inbred generations. Marker-assisted selection (MAS) using three polymerase chain reaction-based codominant markers (TG32, TG591, and SCAR421) was used in F2 progeny with the goal of selecting for homozygous coupling-phase recombinant lines. From 1152 F2 plants, 11 were identified with potential recombination events between Ph-3 and Sw-5; of those, three were male sterile (ms-10). F3 progeny were generated from the remaining eight F2 recombinants, and resistance to both pathogens, or Ph-3 and Sw-5 in the coupling phase, was confirmed in three of those. Recombination was suppressed fivefold in our F2 population to 1.11 cM between genes when compared with published maps of the same region. However, MAS was an efficient tool for selecting the desirable recombination events for these two pathogen resistance genes.

Although cultivar development for multiple pathogen resistance in tomato (Solanum lycopersicum L.) is a desirable goal, the process is often challenging. This is largely the result of the need for large-scale screening and the lack of available resistance genes in a cultivated genetic background. It is often further complicated by linkage drag of unacceptable characteristics tightly linked with resistance, emergence of new disease pathogens or new races of existing pathogens, and the necessity of selecting for resistance to multiple pathogens (Yang and Francis, 2005). Marker-assisted selection (MAS) offers an opportunity to overcome some of the problems associated with phenotypic selection and facilitates combining multiple resistance genes.

Tomato spotted wilt virus (TSWV) and Phytophthora infestans (Mont.) de Bary (late blight) are responsible for substantial tomato crop losses worldwide (Foolad et al., 2008; Fry and Goodwin, 1997; Kim and Mutschler, 2005, 2006; Mumford et al., 1996; Roselli et al., 1996). Several resistance genes have been identified for both pathogens; however, two genes have been found to be especially valuable for their broad level of resistance to each of these pathogens (Foolad et al., 2008; Gordillo et al., 2008). Both genes, Sw-5 for resistance to TSWV and Ph-3 for resistance to P. infestans, were originally introgressed from wild relatives and confer high levels of pathogen resistance.

The TSWV resistance gene Sw-5 was introgressed from S. peruvianum (previously Lycopersicon peruvianum Mill.; accession unknown) (Stevens, 1964; Stevens et al., 1992; van Zijl et al., 1986). This gene confers resistance to the Tospovirus species TSWV, Groundnut ringspot virus, and Tomato chlorotic spot virus (Biotex et al., 1993; Stevens et al., 1992; van Zijl et al., 1986). The P. infestans resistance gene Ph-3, derived from S. pimpinellifolium L. [formerly L. pimpinellifolium (Jusl). Mill. accession L3707], is of incomplete dominant inheritance; however, it confers strong resistance to a spectrum of P. infestans isolates, even when plants are heterozygous for this gene (AVRDC, 1998; Black et al., 1996a, 1996b; Chwungonge et al., 1998, 2002; Foolad et al., 2008; Gardner and Panthee, 2010). Sw-5 and Ph-3 are tightly linked to the restriction fragment length polymorphism markers CT220 and TG591A, respectively (Brommonschenkel et al., 2000; Chwungonge et al., 1998, 2002; Folkertsmia et al., 1999; Stevens et al., 1995). These two markers are near the telomere of the long arm of chromosome 9 and are separated by 5 cM or less on the EXPEN 2000 and the EXPEN 1992 tomato maps (http://solgenomics.net; Tanksley et al., 1992). Because these two genes originate from different wild relatives of tomato, they are naturally linked in the repulsion phase. The phase and short genetic distance between them provide a potential challenge to recombining them into a coupling phase, a step needed for developing homozygous parent lines for hybrids and open-pollinated cultivars. An additional challenge is that a number of tomato breeding programs and studies have identified recombination suppression in chromosomal regions derived from wild relatives (Canady et al., 2005, 2006; Ganal and Tanksley, 1996; Lihras et al., 1996).

Because both TSWV and P. infestans can be devastating pathogens, the development of tomato germplasm homozygous for Sw-5 and Ph-3 enables the possibility of pyramiding Ph-1, Ph-2, and Ph-3 with Sw-5 and Sw-7 for broader resistance than is presently available.
BREEDING, CULTIVARS, ROOTSTOCKS, AND GERMPLASM RESOURCES

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Atriplas inoculation of the ‘HR-1’ isolate was described (Canady et al., 2001; Gordillo et al., 2003), eight F2 recombinants were evaluated in the field in Wooster, OH, in 2000; Gardner and Panthee, 2010] in 2000, we transplanted 40 individual F3 plants for confirmation of the original F2 recombinants (Table 1).

Phytophthora infestans resistance was evaluated using a detached leaf assay and field tests. Isolate Ph2-OC, originating in tomato in North Carolina, overcomes Ph-2, but not Ph-3, and was maintained on tomato plants. Lines NC 2CELBR (Ph-3/Ph-3; resistant) and NC 30P (Ph-3/Ph-3; susceptible) were used as controls. Fully expanded young leaves with five leaflets were excised from each of six plants per line using a single-edge razor blade from greenhouse-grown plants. Each leaf was placed into 120 mL distilled water in a snap seal plastic sample container with a 1-cm hole in the center of the lid. Containers were placed in a clear plastic box and hand sprayed to atomize a sporangia suspension (5000 sporan- gia/mL of distilled water) of Ph2-OC until the upper surface of each excised tomato leaf was completely covered. The boxes were closed by a polyethylene bag and placed at 16°C for 12-h photoperiods. Leaves of each line were evaluated after 1 week using a score from 1 to 5 (1 for no lesions and 5 for completely covered with lesions or dead). Fifteen resistant lines identified from the detached leaf tests were planted in the field in the summer in two locations (Milledgeville and Waynesville, NC) under natural inoculum without any fungicide spray. Individual plants were scored from 0 to 5 (0 for no lesions and 5 for completely covered with lesions or dead).

Molecular marker genotyping. Marker-assisted selection for Ph-3 and Sw-5 was accomplished using previously identified markers. Ph-3 was indirectly selected using the cleaved amplified polymorphic sequences (CAPS) markers TG328 and TG591 (M. Mutschler, personal communication). TG328 was detected using the primers (5’ to 3’) TG328F (GGTGATCTGCTTATAGACTTG and TG328R (AAGGTCTAAAGAAG CGACTGTCG and the restriction endonuclease BstNI. For TG591, the primers TG591F (AAGGCAAAGAAGTTGGAGGTCA) and TG591R (AGAGTTGCAACTCTGTT GATTGAG) were used with AcI. Fragment sizes for TG328/BstNI were 500 bp (undigested) for the NC946 allele (T) and 260/240-bp doublet for the NC592 allele (P). For the TG591/AcI digest, a multiple band phenotype was observed with the diagnostic polymorphism as a 150-bp fragment in NC946 (T) and a 160-bp fragment in NC592 (P).

To indirectly select for Sw-5, the sequenced characterized amplified region (SCAR) marker SCAR421 was used (Anfoka et al., 2006; Folkertsma et al., 1999; Stevens et al., 1996). SCAR421 amplified a 940-bp fragment in NC946 (T allele associated with Sw-5) and a 920-bp fragment in NC592 (P allele). Each individual within all populations evaluated using markers was initially genotyped and then selections were subsequ-}

 territorio as homozgyous-susceptible (Sw-5'/Sw-5'). Seven to 20 individual F2 or F3 progeny were rated for resistance to TSWV as R or S and the percentage of susceptible (S) progenies was calculated. For the segregation evaluations were performed in 2008 and 2010 using progeny from different F2 recombinants (Table 1).

Materials and Methods

Plant material. An F2 population, segre- gating for Ph-3 and Sw-5, was derived from NC 946-1(2004)-11 × NC 0592-8-10 and was used to identify coupling phase Ph-3/Sw-5 recombinants. NC 0592-8-10, designated as NC92, is a Ph-3/Ph-3 inbred line and was derived from NC 0483 × NC 25P (Gardner and Panthee, 2003), whereas NC946 (2004)-11, designated as NC946, is a Sw-5/Sw-5 inbred line derived from NC EBR-7 × ‘Ame- lia’. A total of 1152 F2 plants were subjected to DNA marker analysis to identify recombinants between Ph-3 and Sw-5. Eleven recombinant F2 plants were identified with the gene combination in the coupling phase and were self-pollinated to create F3 progeny. Because three were male sterile [ms-10 gene derived from the NC952 parent (Gardner, 2000; Gardner and Panthee, 2010)], eight F2 recombinants produced seed in the greenhouse. For each of these eight recombinants, we transplanted 40 individual F3 plants for evaluation in the field in Wooster, OH, in 2008. The F3 plants were also genotyped by molecular markers to identify 65 individuals potentially homozgyous for Sw-5 and Ph-3. These plants were visually inspected and 30 were retained and self-pollinated based on desirable characteristics of wine type, heavy fruit load, uniform fruit color, fruit shape, and fruit size. Among the 30 selected F3 plants were at least three representatives from each of the eight original F2 recombinants (Table 1). Additionally, F3 progeny were created for confirmation of the original F2 recombinants 08-5401, 08-5403, and 08-5408.

Evaluation of disease resistance. TSWV maintenance and evaluation of resistant toma- toes was performed as previously de- scribed (Canady et al., 2001; Gordillo et al., 2003; Stevens et al., 1992). Briefly, mechanical inoculation of the ‘HR-1’ isolate was performed at least twice, 1 week apart, with controls [near isogenic tomato lines 989 (Sw-5/Sw-5; resistant) and 89S (Sw-5'/Sw-5'; susceptible)] included. HR-1 is an isolate derived from TSWV-infected field tomatoes in Hawaii. This isolate readily infects Sw-5'/ Sw-5' lines but does not infect Sw-5/Sw-5 or Sw-5'/Sw-5' genotypes. Plants with stunted growth, necrotic patches, or chlorotic ring- spots were scored as resistant. We used the literature in combination with our data and the available tomato genome sequence to lay the foundation for cloning Ph-3.

Results and Discussion

Selection of coupling phase recombinants using molecular markers. A total of 1152 F2 plants were genotyped with TG328, TG591, and SCAR421 to identify 24 recombinants with 11 potentially having both resistances in coupling phase. Of these 11, eight produced F3 progeny (Table 1). The observed recombi- nation events suggested a marker order of TG328, TG591, and SCAR421, consistent with previous maps and the physical order of the markers on BAC clones for chromosome 9 (Folkertsma et al., 1999). However, re- combination distances of 0.33 cm between TG328 and TG591 and 0.78 cm between TG591 and SCAR421 suggest that recombination was suppressed between the S. peruvianum introgression (Sw-5) and the S. pimpinellifolium introgression (Ph-3) seg- ments. In contrast, markers TG328 and TG591 are separated by 1 cm on the reference maps, whereas TG591 and SCAR421 (physically linked to CT220) are separated by 5.0 cm (Pillen et al., 1996; Tanksley et al., 1992) on the EXPEN 2000 map (http://www. solgenomics.net). Suppression of recombin- ation is not unexpected in introgressions (Canady et al., 2005, 2006; Ganal and Tanksley, 1996; Liharska et al., 1996; Zamir and Tadmor, 1986).

Confirmation of resistances of F4 and F5 progeny. For phenotypic confirmation, F3 and F4 progeny derived from each of the eight F2 coupling phase recombinants were sequentially resampled and genotyped for confirmation. To determine marker genotypes, genomic DNA was isolated in 96-well format follow- ing the modified CTAB method described by Kablekova et al. (2002) and subjected to poly- merase chain reaction (PCR). Conditions for PCR reactions were 10 mm Tris-HCl (pH 8.3), 50 mm KCl, 1.5 mm MgCl2, 50 μM of each dNTP, 0.1 μM of each forward and reverse primers, 20 ng of template DNA, and 1 unit of Taq DNA polymerase in a total volume of 10 to 20 μL. PCR amplification was performed using the cycling parameters of 30 at 94°C followed by 35 cycles of 20 s at 94°C, 20 s at 55°C, and 2 min at 72°C followed by an extended incubation for 7 min at 72°C. For CAPS markers, PCR amplicons were digested with the appropriate restriction enzyme by adding 0.6 to 0.8 units of enzyme in 1x digestion buffer for each microliter of PCR product. Markers were visualized as size polymorphisms by agarose gel electrophoresis. Genotype frequencies for all markers were tested for deviation from expected segregation ratios using the goodness-of-fit test. Linkage distances between markers were calculated from the genotypic data of the original F2 population using JoinMap 3.0 (Van Ooijen and Voorrips, 2001). The markers were first grouped with a LOD of at least 10.0 and then the Kosambi mapping function was used to calculate the distance between markers in centiMorgans.
challenged with *P. infestans* and TSWV. Progeny derived from 08-5401, 08-5404, and 08-5406 were significantly more susceptible to *P. infestans* than the resistant control. These progeny were also more resistant than susceptible controls; thus, we considered the phenotype ambiguous. Progeny from at least five of the F₂ recombinants were unambiguously resistant to *P. infestans*. The progeny derived from 08-5402 appeared to segregate or show less than complete resistance to TSWV, whereas families derived from 08-5407 were fully susceptible. Families derived from 08-5403, 08-5405, and 08-5408 showed resistance to both pathogens (Table 1).

**Table 1. Genotype (F₂ and F₃) and phenotype (F₄ and F₅) of individuals selected as coupling phase recombinants for Ph-3 (*P. infestans*) and Sw-5 (TSWV) resistance genes in tomato.**

| F₂ or F₃ recombinant | Genotype | Resistance rating to *P. infestans* (Ph-3) | Percent susceptible to TSWV (Sw-5) |
|----------------------|----------|------------------------------------------|---------------------------------|
|                      | TG328    | TG591 | SCAR421 | Detached leaf | Field | 2008 | 2010 |
| 08-5401 (F₂)         | H        | H     | T       |              |       | 0.0  |      |
| 08-5401-04           | P        | P     | T       | 1.2          |       |      |      |
| 08-5401-25           | P        | P     | T       | 3.9          |       |      |      |
| 08-5401-31           | P        | P     | T       | 1.3          |       |      |      |
| 08-5402 (F₂)         | H        | H     | T       |              |       |      |      |
| 08-5402-03           | P        | P     | T       | 1.0          |       | 57.1 |      |
| 08-5402-20           | P        | P     | T       | 0.6          |       | 46.1 |      |
| 08-5402-22           | P        | P     | T       | 1.0          |       | 22.2 |      |
| 08-5402-31           | P        | P     | T       | 1.3          |       | 66.7 |      |
| 08-5403 (F₂)         | P        | P     | T       |              |       |      |      |
| 08-5403-05           | P        | P     | T       | 1.0          |       |      |      |
| 08-5403-07           | P        | P     | T       | 1.3          |       | 7.1  |      |
| 08-5403-13           | P        | P     | T       | 1.0          |       | 0.0  |      |
| 08-5403-25           | P        | P     | T       | 0.9          |       |      |      |
| 08-5404 (F₂)         | H        | T     | T       |              |       |      |      |
| 08-5404-01           | P        | T     | T       | 1.3          |       | 9.1  |      |
| 08-5404-14           | P        | T     | T       | 2.2          |       | 6.7  |      |
| 08-5404-15           | P        | T     | T       | 3.9          |       |      |      |
| 08-5404-38           | P        | T     | T       | 2.4          |       |      |      |
| 08-5405 (F₂)         | H        | H     | T       |              |       |      |      |
| 08-5405-01           | P        | P     | T       | 2.5          |       |      |      |
| 08-5405-09           | P        | P     | T       | 1.5          |       |      |      |
| 08-5405-11           | P        | P     | T       | 1.0          |       |      |      |
| 08-5405-18           | P        | P     | T       | 1.1          |       |      |      |
| 08-5406 (F₂)         | H        | T     | T       |              |       |      |      |
| 08-5406-20           | P        | T     | T       | 1.0          |       | 3.0  |      |
| 08-5406-22           | P        | T     | T       | 2.7          |       |      |      |
| 08-5406-27           | P        | T     | T       | 0.0          |       |      |      |
| 08-5407 (F₂)         | H        | P     | T       |              |       |      |      |
| 08-5407-05           | P        | P     | T       | 1.0          |       | 100.0               |
| 08-5407-17           | P        | P     | T       | 1.0          |       | 100.0               |
| 08-5407-19           | P        | P     | T       | 0.3          |       | 100.0               |
| 08-5407-26           | P        | P     | T       | 0.5          |       | 100.0               |
| 08-5408 (F₂)         | H        | T     | T       |              |       |      |      |
| 08-5408-01           | P        | T     | T       | 0.8          |       | 5.6  |      |
| 08-5408-07           | P        | T     | T       | 1.2          |       |      |      |
| 08-5408-09           | P        | T     | T       | 0.9          |       |      |      |
| 08-5408-16           | P        | T     | T       | 1.0          |       |      |      |

Controls

| Genotype | Resistance rating to *P. infestans* (Ph-3) | Percent susceptible to TSWV (Sw-5) |
|----------|------------------------------------------|---------------------------------|
| NC 2CELB (R) | 1.0 |       | 0.0 |
| 89R (R) | 10.0 | 10.0 | 0.0 |
| NC 30P (S) | 4.4 | 5.0 | 100.0 |
| 89S (S) | 100.0 | 100.0 | 0.0 |

Note: F₂ progeny from 08-5401-25 were not tested for TSWV because of lack of seed. F₂ progeny from 08-5401-04, 8-5403-07, and 08-5408-07 were evaluated for TSWV in 2010. All were 0.00% susceptible and matched the genotype of their respective F₂ parent.

F₂ individuals were derived from a cross between resistant parents NC592 (Ph-3/Ph-3) and NC946 (Sw-5/Sw-5) and were selected for coupling phase recombination based on molecular markers. All other recombinants are F₃ individuals selected from the F₂ by markers for homozygous coupling phase recombination.

Genotype of the F₂ or F₃ individual. TG328 and TG591 are linked to Ph-3 and SCAR421 is linked to Sw-5. H = heterozygous; T = homozygous for the NC946 (Sw-5) allele; P = homozygous for the NC592 (Ph-3) allele.

Mean ratings of individual F₄ progeny scored for resistance to *P. infestans* based on 1–5 (detached leaf ratings) or 0–5 (field ratings) scale (0 or 1 for no lesions and 5 for completely covered with lesions or dead).

Individual F₄ or F₅ progeny were rated for resistance to TSWV as R or S and the percentage of susceptibility (S) progenies was calculated.

P from analysis of variance for the test of the main effect of entries (F₄ or F₅ progeny).

TSWV = Tomato spotted wilt virus; R = resistant; S = susceptible; LSD = least significant difference.
The marker and resistance gene order suggested by recombinant progeny is consistent with the physical organization of the Sw-5 region of chromosome 9. Sequence data for bacterial artificial chromosomes (BACs) are available from Folkertsma et al. (1999) and from the international tomato genome sequencing effort. Marker TGS28 is not yet anchored to a BAC. Marker TGS91 is anchored to a 110,571-bp BAC clone, C09Hba109D11 (GenBank EF647605; GI: 149930467), and the 113,581-bp BAC clone C09Hba226D21 (GenBank EU139072; GI: 157649058). The 3' end of C09Hba109D11 overlaps with the 5' end of clone C09Hba226D21 suggesting a possible contiguous sequence. CT202 is anchored to a clone C09Hba109D11 overlaps with the 5' end of BAC, C09HBa109D11 (GenBank EF647605; GI: 149930467), and the 113,581-bp BAC clone C09Hba226D21 (GenBank EU139072; GI: 157649058). The 3' end of C09Hba109D11 overlaps with the 5' end of clone C09Hba226D21, suggesting a possible contiguous sequence. CT202 is anchored to a clone C09Hba109D11 overlapping with the 5' endpoint of BAC, C09HBa109D11 (GenBank EF647605; GI: 149930467). The 114,526-bp BAC Hba0059I05 (SGN only, be-}

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