Spotty results in our *Sw-7* tomato spotted wilt virus research

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The tomato spotted wilt virus (TSWV) resistance gene *Sw-7* derived from *S. chilense* accession LA1938 was named in a Tomato Genetics Cooperative report in 2007. Also in 2007 a field experiment was conducted that consisted of resistant control Fla. 8516, susceptible control Fla. 8153 (Tasti-Lee™), and 102 F_3 lines derived from crosses of Fla. 8516 with susceptible parents where the F_2 selection was done in the absence of TSWV infection. Fla. 8516 and Fla. 8153 had 3.5% and 67.0% disease incidence, respectively. The F_3 lines were tested for their fit to a single dominant gene 3:1 ratio and categorized as homozygous resistant if there was an unacceptable fit due to an excess of resistant plants, heterozygous resistant if the fit was a acceptable, and homozygous susceptible if there was an unacceptable fit due to an excess of susceptible plants. There were 26, 39, and 37 plants in the three above groups, respectively, and this did not fit the expected 1:2:1 ratio (χ^2=8.019, p=0.025-0.01) due to a deficiency of heterozygotes and an excess of susceptible lines. Disease incidences in 2009 hybrid trials were; 42-60% for susceptible hybrids, 13.6-13.8% for *Sw-7/+* hybrids, and 0.0-5.2% for *Sw-5/+* hybrids with differences being significant between the three groups. Two subsequent experiments to clarify the level of dominance for *Sw-7* failed due to a lack of disease infection. Mechanical inoculation of seedlings is not an option for lines with *Sw-7* because they appear to be susceptible. In 2009 *Sw-7* was mapped to the 45-58 cM region of chromosome 12. In 2011 67 lines with recombination in this region were grown in the field. Although disease incidence was low, all 14 RILs with the introgression spanning the markers CT100 andTG360 had no disease; of 11 lines that had no introgression in this region, 7 (64%) had some disease incidence; of 42 lines with recombination between these two markers, 14 (33%) had some disease incidence. Thus, there is some evidence that the gene is between the above markers near the 48cM region. Additional molecular markers have been developed to further delimit the target region.