Inheritance of Partial Resistance to Tobacco Etch Virus and Tobacco Vein Mottling Virus in Burley Tobacco Cultivar Sota 6505

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ABSTRACT

Tobacco etch virus (TEV) and tobacco vein mottling virus (TVMV) are serious diseases of burley tobacco and are controlled most effectively by use of resistant cultivars. The inheritance of resistance to TEV and TVMV in the burley tobacco cultivar Sota 6505 and allelism with other sources of potyvirus resistance were evaluated. Crosses were made between Sota 6505 and two susceptible burley tobacco cultivars, Ky 14 and Va 528. Parental genotypes, F₁, F₂, and backcross generations to each of the parents were evaluated in randomized complete block designs for TEV resistance at Waynesville, North Carolina, and at Las Varas, Nayarit, Mexico, and for TVMV resistance at Laurel Springs, North Carolina, during 1988 and 1989. Crosses were also made between Sota 6505 and TEV- and TVMV-resistant cultivars Virgin A Mutant and Havana 307 to test for allelism. Resistant × resistant crosses were evaluated for TEV resistance in a greenhouse in Raleigh, North Carolina. Plants were mechanically inoculated in the field approximately 1 mo after transplanting and in the greenhouse 1 wk after transplanting. Disease severity data were collected at topping time in the field and 1 mo after transplanting in the greenhouse. Chi-square goodness of fit tests were conducted, but simple Mendelian inheritance ratios did not fit the data for expression of resistance with either virus, possibly because of environmental effects. Generation means analysis showed that a simple additive-dominance model adequately described the data. Additivity was the major genetic effect, and there was no evidence of epistasis. The gene(s) controlling TEV resistance in Sota 6505 appears to be allelic to the virus resistance gene(s) found in resistant cultivars Virgin A Mutant and Havana 307.

The potyviruses tobacco etch virus (TEV) and tobacco vein mottling virus (TVMV) cause yield losses of over 60% in susceptible burley tobacco (Nicotiana tabacum L.) (3–5,13,14,17,19). These nonpersistent viruses overwinter in perennial weeds and are transmitted by several species of aphids (5,13). Losses are variable because disease incidence varies (4,12). Currently, these viruses are controlled through use of resistant cultivars, since controlling perennial weeds and aphids to reduce virus incidence is neither practical nor economical under conditions in North Carolina (2,7,9).

Virgin A Mutant (VAM) and Havana 307 have been used as sources of resistance to TEV and TVMV (6,11). Resistance to TEV and TVMV in VAM is controlled by a single recessive gene (11). However, VAM possesses poor yield and quality and is highly susceptible to both chewing insects and tobacco blue mold, caused by Peronospora tabacina D.B. Adam (15,18). Havana 307 is a low-yielding cultivar of cigar wrapper tobacco, and resistance to TEV is postulated to be conditioned by a few genes with additive effects (16). Sota 6505, a moderately yielding burley cultivar from Switzerland, also has resistance to TEV and TVMV (15).

The objectives of this research were to study the inheritance of partial resistance to TEV and TVMV in Sota 6505 in field and greenhouse experiments and to determine whether the gene(s) conditioning virus resistance in Sota 6505 is the same (allelic) as, or independent of, those found in VAM and Havana 307. If the source of resistance in Sota 6505 differs from that of VAM and Havana 307, Sota 6505 could be used to add genetic diversity to a breeding program.

MATERIALS AND METHODS
Field experiments. Burley tobacco cultivar Sota 6505 was crossed with two TEV- and TVMV-susceptible burley tobacco cultivars, Kentucky 14 (Ky 14) and Virginia 528 (Va 528). Parental (P₁ = Sota 6505, P₂ = Ky 14 or Va 528), F₁, F₂, and backcross (F₁ × P₁ = BC₁P₁, F₁ × P₂ = BC₂P₁) generations were grown in field experiments for evaluation of resistance to TEV and TVMV.

Experiments with TEV were conducted at the Mountain Research Station, Waynesville, North Carolina, and at Las Varas, Nayarit, Mexico, in 1988 and 1989. A randomized complete block design was used. There were five replicates in 1988 and four replicates in 1989 at Waynesville and three replicates at Las Varas in both years. Lines (entries) in the Sota 6505 × Ky 14 family were evaluated at both locations each year. Lines (entries) in the Sota 6505 × Va 528 family were evaluated at Waynesville in 1989 only. The experimental unit consisted of one-row plots containing 22 plants per row at Waynesville and four-row plots containing 13 plants per row at Las Varas. Data were collected on bordered (competitive) plants only. Plants were spaced 46 cm apart within rows and 122 cm between rows.

In North Carolina, tobacco seed was sown in plant beds in March and transplanted to the field in June of each year. In Mexico, seed was sown in September and transplanted in November of each year. Recommended cultural practices for burley tobacco were used at all locations.

Experiments with TVMV were conducted at the Upper Mountain Research Station, Laurel Springs, North Carolina. A randomized complete block design was again used, with five replicates in 1988 and four in 1989. Both the Sota 6505 × Ky 14 and Sota 6505 × Va 528 families were evaluated both years in Laurel Springs in the same manner as the experiments conducted with TEV in North Carolina. All experiments included noninoculated controls.

Isolate NC-191 of TEV was used in the Waynesville experiments (16), and isolate NC-148 of TVMV was used in Laurel Springs. For experiments conducted in North Carolina, stock cultures of the viruses were maintained in plants of burley tobacco cultivar Burley 21 grown in screen cages to prevent contamination from other viruses. Inoculum was prepared and inoculations were performed with an artist’s airbrush as previously reported (15), with the addition of 1% sodium sulfate per volume of buffer and 1.5 g of 22-μm Carbordum per 100 ml of inoculum. In Mexico, tobacco leaves naturally infected with a local TEV isolate, identified by serology, were collected from surrounding fields, homogenized, and rubbed onto the leaves with cotton-tipped applicators, using the same ratios of buffer, tissue,
and Carborundum as in North Carolina.

Plant inoculations with TEV and TVMV were performed at the seven- to 10-leaf stage (approximately 1 mo after transplanting). Individual plant reactions to viral infection (based on visual symptoms) were assessed approximately 45 days after inoculation according to the Horstfall-Barrat scale (8). The disease index scale was: 0 = no symptoms, 1 = 0–3%, 2 = 3–6%, 3 = 6–12%, 4 = 12–25%, and 5 = 25–50% of the leaf area damaged. Symptoms produced by both viruses included etching along the leaf veins and lamina, leaf mottling, chlorosis, and mild leaf and vein necrosis.

**Greenhouse experiments.** TEV resistance could be evaluated in the greenhouse, but TVMV-infected plants showed very mild symptoms. Seeds were sown in plastic pots containing Metro-Mix 220 (Grace Horticultural Products, Cambridge, MA), covered with a clear polyethylene film, and placed in a growth chamber at 24°C, with 16 hr photo-periods having a light intensity of 400 ± 50 μE·m⁻²·s⁻¹. Seedlings at the two- to three-leaf stage were transplanted into 5 × 5 cm peat pots containing Metro-Mix 220, then transferred to the greenhouse. The experimental design was a randomized complete block with four replicates: three inoculated and one noninoculated control. A replicate consisted of one tray of 12 plants for the resistant and susceptible parents and the F₁ generation. Five trays per replicate were used for the F₂ generation and both backcrosses. Segregating generations (F₂, BC₁P₁, and BC₁P₂) were represented by larger populations to gain a more precise estimate of their disease means.

When plants were established and the two lower leaves to be inoculated were 8–10 cm long (usually 2–3 wk after transplanting), plants were inoculated with TEV isolate NC-191. Approximately 3 wk after inoculation, plants were rated individually by the same scale used in the field experiments.

**Genetic analyses.** Disease severity data for both greenhouse and field experiments were analyzed for simple Mendelian inheritance using chi-square goodness of fit tests. Subsequently, the generation means analysis procedure outlined by Mather and Jinks (10) was used to test a simple additive-dominance genetic model for virus resistance. The model can be described in terms of \( m \) = the midparent value or the midpoint between the homozygous parents, \( d \) = the additive effect of the genes or the sum over loci of all \( d \)'s that measure the departure of each homozygote from the midparent \( m \), and \( h \) = the dominance effects of the genes or the sum over loci of all \( h \)'s that measure the departure of the heterozygote from the midparent. The procedure utilizes disease severity means from the six generations evaluated per cross: \( P₁, P₂, F₁, F₂, BC₁P₁, \) and \( BC₁P₂ \). Because the number of individuals varied in each generation, the six means were weighted by the reciprocal of their corresponding variance as suggested by Mather and Jinks (10), and three equations were used to estimate the model parameters \( m, d, \) and \( h \). From the model's parameters, predicted disease values for the six generations were calculated. A joint scaling test as proposed by Cavalli (1) was used to test the fit of the additive-dominance model using a chi-square goodness of fit test with three degrees of freedom. In addition, individual scaling tests as defined by Mather and Jinks (10) were computed and compared with the results of the joint scaling test. The individual scaling tests using disease severity means were:

\[
A = 2BC₁P₁ - P₁ - F₁, \quad B = 2BC₁P₂ - P₂ - F₁, \quad \text{and} \quad C = 4F₁ - 2F₂ - P₁ - P₂,
\]

where \( A, B, \) and \( C \) should equal to zero (as determined by a \( t \) test) if the additive-dominance model adequately describes the genetic variance (10).

**Test of allelism.** A second greenhouse experiment was conducted to determine whether Sota 6505 possesses the same or different gene(s) for TEV resistance as those found in VAM and Havana 307. A randomized complete block design was used with four replicates: three inoculated and one noninoculated control. Parental \( (P₁ = \text{Sota 6505 and } P₂ = \text{VAM or Havana 307}) \), \( F₁ \), and \( F₂ \) generations were evaluated. Plants of \( Ky \) 14 were included as a susceptible control. A replicate included one tray of 12 plants each of the two parents and \( F₁ \) progeny and 15 trays of the \( F₂ \) generation. Plants were inoculated with TEV isolate NC-191 when the two lower leaves were 8–10 cm long. Individual plants were evaluated for disease symptoms approximately 3 wk later, and a random sample of plants was assayed using the Ouchterlony double-diffusion test to detect the presence of virus in asymptomatic plants.

**RESULTS AND DISCUSSION**

**Field experiments.** Analyses of variance revealed highly significant differences among families in all trials. Year

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**Fig. 1.** Disease index means of parental (Sota 6505 and Ky 14), \( F₁, F₂ \), and backcross generations for reaction to tobacco etch virus under field conditions in (A) Waynesville, North Carolina, and (B) Las Varas, Nayarit, Mexico. Data represent means for 2 yr, 1988 and 1989. Disease index scale: 0 = no symptoms, 1 = 0–3%, 2 = 3–6%, 3 = 6–12%, 4 = 12–25%, and 5 = 25–50% of the leaf area damaged.
effects were generally significant except for the TEV trials with Sota 6505 × Ky 14 conducted at Waynesville. Year × family interactions were not significant except for the Sota 6505 × Ky 14 cross inoculated with TVMV at Laurel Springs. This interaction was due to differences in the magnitude of disease observed over years and not to differences in family rankings. Therefore, data were pooled across years.

Average disease severity data for all generations from the cross Sota 6505 × Ky 14 in the TEV experiments conducted at Waynesville showed that Sota 6505 was consistently the most resistant parent, with an average disease index of 1.6 over both years (Fig. 1A). Most Sota 6505 plants (70%) had disease indexes of 0–2, whereas the remaining plants (30%) were scored in the 3–4 range; no Sota 6505 plants received a disease index of 5. Ky 14 plants were the most susceptible, with an average disease index of 4.9; 97% of the Ky 14 plants showed a disease index of 5, and none was scored lower than 4. F1 plants had an average disease rating of 3.6, slightly higher than the midparent value of 3.3. As with the parental lines, the F1 generation had varying reactions to TEV, with most plants (99%) classified in the 3–5 disease indexes. Variation in the resistant and susceptible parental lines and in the F1 generation may have been due to environmental factors (particularly temperature), as genotypes were genetically uniform.

The F2 generation had a disease severity index mean of 3.5, although scores were distributed toward susceptibility. In the F2, 88% of the plants had disease indexes of 3–5. The BC1P1 generation had a disease severity index mean of 2.3 and was skewed in the direction of the resistant parent (Sota 6505); 84% of the plants in the BC1P1 generation had disease indexes of 0–3. The BC1P2 generation had a disease index mean of 4.0 and was skewed in the direction of the susceptible parent (Ky 14); all BC1P2 plants had indexes of 3–5.

Mean disease severity indexes for all generations from the cross Sota 6505 × Ky 14 in the TEV experiments conducted at Las Varas, Nayarit, Mexico, showed the same general trend as in the Waynesville experiments (Fig. 1B), although a different isolate of TEV was used. Sota 6505 had an average disease rating of 0.9, with 96% of the plants having disease indexes of 0–2. Ky 14 had an average disease index of 4.2, with 94% of the plants scoring in the 3–5 range; 3% of the Ky 14 plants received a rating of 0, indicating a lack of virus infection (escapes), possibly resulting from poor

**Fig. 2.** Disease index means of parental (Sota 6505 and Ky 14), F1, F2, and backcross generations for reaction to tobacco vein mottling virus under field conditions in Laurel Springs, North Carolina. Data represent means for 2 yr, 1988 and 1989. Disease index scale: 0 = no symptoms, 1 = 0–3%, 2 = 3–6%, 3 = 6–12%, 4 = 12–25%, and 5 = 25–50% of the leaf area damaged.

**Fig. 3.** Disease index means of parental (Sota 6505 and Va 528), F1, F2, and backcross generations for reaction to tobacco viruses under field conditions in 1989. Reaction to (A) tobacco etch virus, Waynesville, North Carolina, and (B) tobacco vein mottling virus, Laurel Springs, North Carolina. Disease index scale: 0 = no symptoms, 1 = 0–3%, 2 = 3–6%, 3 = 6–12%, 4 = 12–25%, and 5 = 25–50% of the leaf area damaged.
inoculation technique. The F\textsubscript{1} generation score of 3.6 was again higher than the midparent value of 2.5. The F\textsubscript{2} generation had a disease index of 3.0, between the midparent value and the F\textsubscript{1} generation score. In the F\textsubscript{2} generation, 30% of the plants had disease indexes of 0–2. The BC\textsubscript{1}P\textsubscript{1} and BC\textsubscript{1}P\textsubscript{2} generations had average indexes of 2.5 and 3.8, respectively, and both were skewed toward their respective parent.

In the TVMV experiments conducted at Laurel Springs with the Sota 6505 × Ky 14 cross, Sota 6505 had an average disease index of 0.2, with 99% of the plants in the 0–1 indexes (Fig. 2). Ky 14 had an average disease index of 4.3, and all plants were in the 3–5 range. The F\textsubscript{1} generation disease index of 3.0 was higher than the midparent value of 2.2. The F\textsubscript{2} generation average disease index of 2.8 was slightly lower than that of the F\textsubscript{1}. The BC\textsubscript{1}P\textsubscript{1} generation had a mean disease index of 1.8 and was skewed toward its recurrent parent; 70% of BC\textsubscript{1}P\textsubscript{1} generation plants received a score of 0–3. The BC\textsubscript{1}P\textsubscript{2} generation had an average disease rating of 3.6 and was skewed in the direction of susceptibility, with 100% of the plants receiving a value of 3–5.

Lines from the cross Sota 6505 × Va 528 were evaluated for TEV resistance in 1989 at Waynesville. As in the other tests, Sota 6505 had the lowest average disease rating, 0.1, and all plants were scored in the 0–2 disease indexes (Fig. 3A). The susceptible parent, Va 528, had an average disease value of 2.8, and all plants were rated in the 2–4 indexes.

### Table 1. Joint scaling test (chi-square) and individual scaling tests (A, B, C) of the fit of the additive-dominance genetic model for the inheritance of resistance to tobacco etch virus (TEV) and tobacco vein mottling virus (TVMV) in two crosses of burley tobacco under greenhouse and field conditions

<table>
<thead>
<tr>
<th>Test</th>
<th>Virus</th>
<th>Location</th>
<th>Sota 6505 × Ky 14</th>
<th>Sota 6505 × Va 528</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint scaling(^a)</td>
<td>TEV</td>
<td>Greenhouse</td>
<td>5.2</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Field</td>
<td>12.4</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td>TVMV</td>
<td>Field</td>
<td>17.3</td>
<td>48.2</td>
</tr>
<tr>
<td>Individual scaling(^b)</td>
<td>TEV</td>
<td>Greenhouse</td>
<td>0.10 ± 2.5 NS(^c)</td>
<td>0.15 ± 2.4 NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Field</td>
<td>0.06 ± 2.8 NS</td>
<td>0.21 ± 2.5 NS</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>Greenhouse</td>
<td>−0.24 ± 1.6 NS</td>
<td>0.05 ± 1.3 NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Field</td>
<td>−0.30 ± 1.7 NS</td>
<td>0.00 ± 1.5 NS</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>Greenhouse</td>
<td>−0.14 ± 4.2 NS</td>
<td>0.18 ± 4.5 NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Field</td>
<td>−0.03 ± 4.6 NS</td>
<td>−0.24 ± 4.5 NS</td>
</tr>
<tr>
<td></td>
<td>TVMV</td>
<td>A</td>
<td>0.41 ± 2.0 NS</td>
<td>−0.80 ± 2.0 NS</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.14 ± 1.3 NS</td>
<td>−1.00 ± 1.5 NS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>0.78 ± 4.2 NS</td>
<td>−0.03 ± 3.7 NS</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)Calculated chi-square values compared with tabular value obtained by equating observed family means to their expectations. Probability of a fit = 0.50–0.95, indicating adequacy of the additive-dominance genetic model.

\(^b\)Where \(A = 2BC\textsubscript{1}P\textsubscript{1} - P\textsubscript{1} - F\textsubscript{1} = 0 ± SE\), \(B = 2BC\textsubscript{1}P\textsubscript{2} - P\textsubscript{2} - F\textsubscript{1} = 0 ± SE\), and \(C = 4F\textsubscript{2} - 2F\textsubscript{1} - P\textsubscript{1} - P\textsubscript{2} = 0 ± SE\).

\(^c\)NS = Not significantly different from zero, indicating agreement with the joint scaling test (\(\chi^2\)), i.e., adequacy of the additive-dominance genetic model.

### Table 2. Estimates of genetic effects in two crosses of burley tobacco evaluated for resistance to tobacco etch virus (TEV) and tobacco vein mottling virus (TVMV) under greenhouse and field conditions

<table>
<thead>
<tr>
<th>Virus</th>
<th>Genetic effect(^a)</th>
<th>Location</th>
<th>Sota 6505 × Ky 14</th>
<th>Sota 6505 × Va 528</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEV</td>
<td>(m)</td>
<td>Greenhouse</td>
<td>2.58</td>
<td>1.32</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Field</td>
<td>2.86</td>
<td>1.42</td>
</tr>
<tr>
<td>(d)</td>
<td>Greenhouse</td>
<td>−1.47</td>
<td>−1.10</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Field</td>
<td>−1.61</td>
<td>−1.33</td>
<td></td>
</tr>
<tr>
<td>(h)</td>
<td>Greenhouse</td>
<td>0.46</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Field</td>
<td>0.66</td>
<td>−0.03</td>
<td></td>
</tr>
<tr>
<td>TVMV</td>
<td>(m)</td>
<td>Field</td>
<td>2.28</td>
<td>2.11</td>
</tr>
<tr>
<td>(d)</td>
<td>Field</td>
<td>−2.02</td>
<td>−1.17</td>
<td></td>
</tr>
<tr>
<td>(h)</td>
<td>Field</td>
<td>0.70</td>
<td>0.40</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\)\(m\) = Midparent value, \(d\) = the sum over loci of the additive effects, and \(h\) = the sum over loci of the dominance effects.

Disease severity was lower in this cross than in the Sota 6505 × Ky 14 cross. The susceptible parent (Va 528) did not show severe symptoms during the 1989 growing season, i.e., no plant received a score of 5. The F\textsubscript{1} generation average disease mean of 2.1 was higher than the midparent value of 1.4. The F\textsubscript{2} generation average disease index was 1.7, the BC\textsubscript{1}P\textsubscript{1} mean was 1.2, and the BC\textsubscript{1}P\textsubscript{2} mean was 2.4. The same pattern occurred as with the Sota 6505 × Ky 14 cross, i.e., each backcross was skewed toward its recurrent parent and the F\textsubscript{2} generation had a lower value than the F\textsubscript{1} generation. The score for 23% of the plants in the F\textsubscript{2} generation was 0.

Disease severity data from the cross Sota 6505 × Va 528 in the TVMV experiments conducted at Laurel Springs, North Carolina, showed average disease values of 1.0 for Sota 6505 and 3.4 for Va 528 (Fig. 3B). As with the TEV test in Waynesville, plants in this cross did not show severe symptoms and only 8% received a rating of 5. Mean disease values were 2.7 for plants in the F\textsubscript{1} generation and 2.4 for those in the F\textsubscript{2} generation. Both scores were slightly above the midparent value of 2.2. The BC\textsubscript{1}P\textsubscript{1} generation had an average disease value of 1.4 and was skewed toward the resistant parent, with all plants receiving indexes of 0–3. The BC\textsubscript{1}P\textsubscript{2} generation average disease value was 2.6, with 87% of the plants having an index of 3–4.

### Genetic analyses

An effort was made to fit simple Mendelian gene models to the field and greenhouse data, but observed values differed significantly from expected values for one and two gene models (both dominant and recessive) as determined by chi-square goodness of fit tests. Environmental effects on expression of resistance may prevent fit of a simple gene model.

Individual scaling tests and joint scaling tests for the crosses Sota 6505 × Ky 14 and Sota 6505 × Va 528 for both TEV and TVMV experiments were found to be nonsignificant, indicating that a simple additive-dominance model adequately explains the variation with no evidence of epistasis (Table 1).

Estimates of \(m\), \(d\), and \(h\) were calculated from the joint scaling test (Table 2). The midparent values (\(m\)) in the TEV experiments ranged from 1.32 to 2.86. The values of \([d]\) (the sum over loci of the additive effects) ranged from −1.10 to −1.61, and the values of \([h]\) (the sum over loci of the dominance effects) ranged from −0.03 to 0.66. In the TVMV experiments, the midparent values (\(m\)) ranged from 2.11 to 2.28, the \([d]\) values ranged from −1.17 to −2.02, and the \([h]\) values ranged from 0.40 to 0.70. The values of \([d]\) were negative because the disease scale measured disease severity (i.e., resistance values are obtained by measuring in the negative direction or corresponding smaller disease values).
Table 3. Tobacco etch virus incidence in parental (P₁ and P₂), F₁, and F₂ generations derived from resistant × resistant crosses \( (n = 684) \)

<table>
<thead>
<tr>
<th>Cross</th>
<th>P₁</th>
<th>P₂</th>
<th>F₁</th>
<th>F₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sota 6505 × Virgin A Mutant</td>
<td>0/48</td>
<td>0/48</td>
<td>0/48</td>
<td>17/540</td>
</tr>
<tr>
<td>Sota 6505 × Havana 307</td>
<td>0/48</td>
<td>0/48</td>
<td>0/48</td>
<td>0/540</td>
</tr>
</tbody>
</table>

The additive component \([d]\) was the major genetic effect in these systems, ranging from 3 to 44 times greater than the dominance component \([h]\) (Table 2). Further support of additivity is derived from the F₁ generation disease means, which were close to the midparental values. This is not surprising, as tobacco is a self-pollinated species where additive genetic effects are usually most important. Since additivity for resistance is the major component, incorporating the resistance found in Sota 6505 into other breeding material should not be a problem. In the field, F₂ generation plants were found with a rating of 0, indicating that resistance lines can be developed easily through selection and selfing.

**Test of allelism.** A greenhouse study was conducted to determine whether the gene(s) for TEV resistance in Sota 6505 is the same or different from those present in Havana 307 and VAM. The two resistant parents of each cross, as well as the F₁ and F₂ generations, were evaluated. In the Sota 6505 × VAM cross, neither parent nor F₁ showed any symptoms (Table 3). Seventeen plants from the F₂ generation were infected with TEV, as determined by the Ouchterlony test, but symptoms were not as severe as those on the susceptible check cultivar Ky 14. The presence of a few symptomatic plants was not unexpected, as occasionally a plant of Sota 6505 will show mild TEV symptoms in the greenhouse as well as in the field. However, the occurrence of symptomatic plants is rare and not considered evidence of segregation but is thought to be variation in expression of partial resistance due to environmental effects. In the cross Sota 6505 × Havana 307, no visually infected plants were detected from parental, F₁, or F₂ generations. Furthermore, none of the F₂ plants reacted positively to the Ouchterlony test. The lack of segregation in F₂ generations indicates the gene(s) coding for partial resistance to TEV in Sota 6505 must be allelic to those present in VAM or Havana 307.

Although allelic to VAM and Havana 307, Sota 6505 possesses partial resistance to several different isolates of TEV and to at least one isolate of TVMV and therefore would make a valuable contribution to a breeding program. Sota 6505 has several advantages over the currently used sources of virus resistance in breeding programs, i.e., VAM and Havana 307. Sota 6505 is a commercial burley cultivar, whereas Havana 307 is a cigar wrapper tobacco and chemically quite different from burley tobacco. VAM is a burley tobacco whose plant type, yield, and quality are inferior to those of Sota 6505. In addition, Sota 6505 does not appear to possess the extreme susceptibility to chewing insects and blue mold that are serious problems with VAM (18).

During the years of evaluating Sota 6505, the incidence of potato virus Y (PVY) has increased in the field. Because the reaction of Sota 6505 to PVY is unknown, it would be valuable to test Sota 6505 with different PVY isolates, as this virus could become a threat to burley tobacco growers in the future.

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**Literature Cited.**