Relationship Between Seed Infection by Barley Stripe Mosaic Virus and Yield Loss

F. W. Nutter, Jr., V. D. Pederson, and R. G. Timian

Former graduate research assistant, professor, and research plant pathologist (USDA, ARS), respectively, Department of Plant Pathology, North Dakota State University, Fargo 58105.

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ABSTRACT


Field experiments were conducted in 1981 and 1982 to quantify the effect of barley stripe mosaic virus (BSMV) seed infection levels on grain yield, yield components, kernel protein, and percent seed transmission in barley cultivar Dickson. Barley seed infected with BSMV (strain ND18) was blended with feed free of BSMV to produce infected seed levels of 0, 0.1, 1, 5, 15, 30, 45, and 60%. The number of heads per meter of row and seed weight decreased as percent seed infected with BSMV increased, whereas the number of seeds per head was not greatly affected by BSMV. Reduction in yield per plot, heads per meter of row, and 500-seed weight in response to increasing levels of seed infection by BSMV was best described by a quadratic model in 1981, but by a linear model in 1982. To make yield and yield component loss comparisons between years, the linear model \( y = b_0 + b_1x \), in which \( y \) is the predicted response of any yield factor and \( x \) is the level (percentage) of seed infection by BSMV, was the most adequate. Regression coefficients relating levels of seed infection by BSMV to reduction in yield per plot, heads per meter of row, seeds per head, and 500-seed weight were greater in 1981 than in 1982. This was most likely the result of environmental conditions favoring greater plant-to-plant spread of BSMV in 1981. The apparent infection rate was greater in 1981 than in 1982 and percent seed transmission of BSMV (an indirect measure of field spread) was also greater in 1981. In 1981, percent kernel protein increased as the proportion of seed infected by BSMV was increased.

Barley stripe mosaic virus (BSMV) is one of the few seedborne viruses of grasses (16). Seed infection is epidemiologically important because it ensures that the virus will be associated with the planted crop, infected seeds are randomly dispersed in the field, and the infected seedlings serve as sources of inoculum to initiate secondary spread because the virus is also mechanically transmitted from plant to plant (1,7,13-15).

Artificial inoculations of barley field plots with BSMV have provided information on how the relative yield losses incurred when all plants in a plot are infected at one specified growth stage (16). Using this method, the greatest reduction in yield occurred when plants were inoculated in the late tillering stage (3,14,16). However, under natural conditions, simultaneous infection of all plants in a field is most unlikely. There may be yield compensation by healthy plants that are adjacent to infected plants. Also, plants that are infected late in the season suffer less yield loss (5,16). Loss in yield should be a function of the initial proportion of infected seed and the rate of secondary spread early in the growing season.

Knowledge of the relationship between disease severity and yield loss is essential for effective disease management. Although seed certification programs have reduced the prevalence of BSMV, the virus is still found in many barley-producing areas (3-5,14).

The objective of the research reported here was to determine more precisely the effect of seed infection by BSMV and its subsequent spread in the field on barley yield.

MATERIALS AND METHODS

Planting. Field plots were located at North Dakota State University, Fargo. Barley (Hordeum vulgare L.) cultivar Dickson was sown in plots with a drill on 5 May 1981 and 8 May 1982. The plots were arranged in a randomized complete block design. Seed infected with BSMV strain ND18 (cereal virus 52) was produced in 1978 by inoculating field plots of Dickson barley at the two-leaf stage. Sixty-four percent of the harvested seed was infected with BSMV as determined by greenhouse tests and serological latex tests on seed embryos (9). This seed lot was blended with BSMV-free seed to produce known initial percentages (0, 0.1, 1, 5, 15, 30, and 60%) of seed infected with BSMV in 1981. In 1982, the 0.1% level in the series was replaced with 45%. Seeding rates were adjusted for differences in percent germination and seed weight to minimize differences in stands among treatments. Stand counts were made at the two-leaf stage and there were no significant differences in stands among treatments in either year. Each plot (seed infection level) was 1.8 m wide (15-cm row spacing) and 6.1 m long and there were twelve replicates. Each plot was surrounded by six rows of oats (Avena sativa L. 'Moore') to prevent the spread of BSMV between plots. Pathogenicity tests revealed that Moore was immune to strain ND18 of BSMV.

Disease assessment. The percentage of plants infected by BSMV was determined at the tillering, jointing, and boot stages of growth. Four groups of 25 successive plant samples from each plot were assessed visually (nondestructively) for symptoms caused by BSMV. The four assessments were averaged to estimate the percentage of plants infected by BSMV in each plot. Percent estimates were converted to disease proportions, transformed to logit, \( \log \left(\frac{X}{(1-X)}\right) \), and apparent infection rates were calculated by regressing logit on time (17).

Harvest. The heads per meter of row in each plot were counted just before harvest. Also, seeds per head from 40 randomly selected heads per plot were counted. Plots were harvested on 12 August in 1981 and on 8 August in 1982. After harvest, the seeds from each plot were air-dried, cleaned, and weighed. The 500-seed weight was also obtained for each plot. Percent seed transmission (percent harvested seeds infected with BSMV) was determined by planting 300 seeds from each plot in seed rolls of 50 seeds each (2). Seeds were germinated in a growth chamber maintained at a constant temperature of 30 °C and fluorescent light (350 µE·m⁻²·sec⁻¹) for a 16-hr photoperiod. Seedlings were examined for symptoms of BSMV 7 days later. Total protein of barley kernels (percent dry basis) was determined by the Department of Cereal Chemistry and Technology, North Dakota State University, Fargo, using the Kjeldahl technique (12).

The influence of levels of infection by BSMV in seeds and secondary spread in the field on yield, yield components, percent kernel protein, and percent seed transmission was analyzed by regression with levels of seed infection by BSMV as the independent variable. Comparisons of differences in yield variables in the absence of BSMV seed infection (y-intercept) and in response
to increasing levels of BSMV seed infection (regression coefficient) between years were conducted by adding 'year' as an independent classification variable to the linear model.

RESULTS

Grain yield, heads per meter of row, and 500-seed weight decreased as the proportion of planted seed infected with BSMV was increased in both years (Fig. 1). Reduction in yield, number of heads per meter of row, and seed weight (Y) in response to increasing levels (percent) of seed infection by BSMV (X) was best described by a linear model in 1982, but by a quadratic model in 1981. Although the quadratic term in the models for reduction in grain yield per plot, number of heads per meter of row, and seed weight explained a significant part of the variation in 1981, comparisons between years could only be made with one model, and we chose the linear model since this model adequately described both years. Comparisons of the linear regression models relating BSMV seed infection to yield (dependent) variables in 1981 and 1982 are given in Table I.

The variable 'year' was added to the linear model as an independent classification variable to test if the intercepts (b0) and

Fig. 1. Relationship between percent BSMV-infected seed of barley cultivar Dickson planted and: A, grain yield per plot; B, number of heads per meter of row; C, seed weight; D, number of seeds per head; E, kernel protein (percent dry basis); and F, percent seed transmission in 1981 and 1982.
TABLE 1. Relationship between yield variables ($Y$) and percent BSMV infected seed planted ($x_0$) in 1981 and 1982

<table>
<thead>
<tr>
<th>Dependent Variable ($Y$)</th>
<th>1981 Intercept</th>
<th>Regression coefficient</th>
<th>Coefficient of determination</th>
<th>1982 Intercept</th>
<th>Regression coefficient</th>
<th>Coefficient of determination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yield per plot (kg)</td>
<td>3.28</td>
<td>-0.031***</td>
<td>0.72</td>
<td>3.57</td>
<td>-0.024***</td>
<td>0.68</td>
</tr>
<tr>
<td>500 seed wt. (g)</td>
<td>14.38</td>
<td>-0.040**</td>
<td>0.53</td>
<td>18.29</td>
<td>-0.018**</td>
<td>0.48</td>
</tr>
<tr>
<td>Number of heads</td>
<td>68.08</td>
<td>-0.259**</td>
<td>0.54</td>
<td>47.01</td>
<td>-0.141**</td>
<td>0.49</td>
</tr>
<tr>
<td>meter of row</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of kernels per head</td>
<td>54.80</td>
<td>-0.112**</td>
<td>0.35</td>
<td>43.80</td>
<td>-0.001</td>
<td>0.01</td>
</tr>
<tr>
<td>Transmission (%)</td>
<td>-0.58</td>
<td>+0.590**</td>
<td>0.91</td>
<td>-0.55</td>
<td>+0.235**</td>
<td>0.87</td>
</tr>
<tr>
<td>Kernel protein (%)</td>
<td>12.68</td>
<td>+0.050**</td>
<td>0.75</td>
<td>14.04</td>
<td>+0.003</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*The number of observations in each analysis was 84 (7 treatments x 12 replications).

**F-test significant at $P = 0.01$.

***Percent BSMV infected seed in harvested plots.

**Percent protein on dry weight basis.

regression coefficients ($b$) for yield variables in 1981 were significantly different from those of 1982 ($b_1$). The null hypothesis that the $y$-intercept for grain yield per plot in 1981 was equal to the $y$-intercept in 1982 was accepted (ie, intercepts were not significantly different), but the hypothesis that the regression coefficients were equal was rejected. Grain yield per plot was reduced more in 1981 by BSMV than in 1982 (Fig. 1A).

Effect of BSMV on components of yield. The number of heads per meter of row decreased in both years as the level of seed infected by BSMV increased (Fig. 1B). The regression coefficient and the $y$-intercept was significantly greater in 1981 than in 1982. Seed weight was reduced more by BSMV in 1981 than in 1982 as indicated by the significantly higher regression coefficient in 1981 (Fig. 1C). The $y$-intercept for 1981 was significantly lower than in 1982. The number of kernels per head was reduced by BSMV in 1981, but not in 1982 (Fig. 1D).

Effect of BSMV on kernel protein. Kernel protein increased significantly as the proportion of seed infected by BSMV planted in 1981 was increased (Fig. 1E). Kernel protein was high for all treatments in 1982, and no effect of BSMV seed infection was evident.

Effect of BSMV seed infection level on percent transmission and rate of secondary field spread. Percent transmission of BSMV in the seed of the harvested crop indirectly indicates the degree of early plant-to-plant spread in the field. The intercepts for percent transmission in 1981 and 1982 were not significantly different, but the regression coefficients were (Fig. 1F).

A more direct measure of the rate of plant-to-plant field spread is the apparent infection rate (AIR). The level of seed infection by BSMV did not affect the rate of secondary spread. The average AIR in 1981 was 0.07 per unit per day which was significantly higher than the average AIR of 0.02 per unit per day in 1982 (Table 2). The $y$-intercepts were not significantly different between years.

**DISCUSSION**

Yield in barley is the product of heads per unit area, number of seeds per head, and seed weight (6). Reduction in grain yield per plot in response to increasing levels of BSMV seed infection was largely caused by a reduction in the number of heads per meter of row and lower seed weight. The incidence of BSMV in seed had a greater effect on grain yield, the number of heads per meter of row, and seed weight in 1981 compared to 1982. The $y$-intercept for seed weight was higher in 1982, but this may have been due in part to the significantly lower $y$-intercept for number of heads per meter of row and lower number of seeds per head in 1982; there may have been some compensation for increased seed weight because of both fewer heads and kernels per head competing for nutrients. The number of seeds per head was not greatly affected by infection of seed by BSMV in 1981 as indicated by the rather low regression coefficient in 1981 ($r = 0.11$) and the lack of response in 1982.

Comparison of regression coefficients relating level of seed infection by BSMV to percent seed transmission suggests that greater plant-to-plant spread of BSMV occurred in 1981. There was more than twice as much seed transmission in 1981 than in 1982. Timian (16) reported that the level of seed transmission of BSMV in barley depended on the age of the plants at the time they became infected. No transmission occurred in plants infected 7 days prior to anthesis and thereafter. Thus, percent seed transmission should be greater in years favoring plant-to-plant spread before anthesis, as was the case in 1981. In 1981 adequate moisture was available for the development of tillers, whereas in 1982 moisture was a limiting factor prior to anthesis and resulted in reduced tillering and leaf contact. More direct evidence of greater plant-to-plant spread in 1981 is the fact that the apparent infection rate was more than three times higher in 1981 than in 1982.

In 1981, kernel protein increased as levels of seed infection by BSMV increased. High protein content in malting barley is undesirable, and seed will be heavily discounted if it is above 13.5% (11). Using the model in Table 1 and solving for $x_0$, 16.5% seed infection by BSMV would have rendered the harvested crop unacceptable for malting. Moisture stress may have been the cause of high protein levels in 1982 in the absence of infection by BSMV. O'Hare (10) found that drought stress increased protein in the barley cultivar 'Trophy'.

This experiment indicates that the initial level of infection by BSMV seed planted and the rate of secondary infection determines the reduction in barley yield caused by a reduction in heads per meter of row and in seed weight. Apparently, if little spread of BSMV occurs in the field, reduction in grain yield per plot, heads per meter of row, and seed weight is nearly linear in response to increasing levels of seed infection by BSMV; but as the rate of secondary field spread increases, greater yield reduction occurs, and the response is more accurately represented by a quadratic model. Infection before or at boot stage severely reduces yield of infected plants because of the shock reaction from virus infection that occurs in newly infected plants (12,16). In our experiments, a higher proportion of plants were infected at a critical stage of growth in 1981 than in 1982.

**TABLE 2. Effect of BSMV seed infection level on the apparent infection rate of barley stripe mosaic in cultivar Dickson barley in 1981 and 1982**

<table>
<thead>
<tr>
<th>Initial level of BSMV-infected seed planted (%)</th>
<th>1981</th>
<th>1982</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>0.09</td>
<td>...</td>
</tr>
<tr>
<td>1.0</td>
<td>0.08</td>
<td>0.02</td>
</tr>
<tr>
<td>5.0</td>
<td>0.07</td>
<td>0.02</td>
</tr>
<tr>
<td>15.0</td>
<td>0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>30.0</td>
<td>0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>45.0</td>
<td>...</td>
<td>0.02</td>
</tr>
<tr>
<td>60.0</td>
<td>0.06</td>
<td>0.02</td>
</tr>
<tr>
<td>Average</td>
<td>0.07</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Apparent infection rate, per unit per day, between tillering and boot growth stages.
The use of levels of seed infection by BSMV offers several advantages over the method of inoculating entire plots at different growth stages to determine yield loss. The effect of levels of seed infection by BSMV on components of yield can be evaluated as well as cultivar effects on field spread and yield compensation. For instance, the use of known seed infection levels may be useful in making comparisons of different strains of BSMV in a single cultivar or as a means of studying the effect of a single strain of BSMV in different barley cultivars to find those in which field spread is restricted and seed transmission is low. Analysis of yield components may be useful in identifying cultivars which are resistant or tolerant to BSMV.

LITERATURE CITED