Reduction of Disease Incidence in Small Field Plots by Isolate-Specific Resistance to Barley Yellow Dwarf Virus

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ABSTRACT

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Epidemics of two isolates, RMV and MAV, of barley yellow dwarf virus (BYDV) were studied in three spring oat genotypes. Two of the genotypes previously were found to possess varying levels of resistance expressed as a reduction in the accumulation of virus antigen titer in plants. The reduced virus antigen titer contributed to reduced virus acquisition and transmission efficiencies by aphid vectors. The third genotype was susceptible to the two BYDV isolates. Resistance was expressed at high levels against the RMV isolate; and in 1990 and 1991, the final disease incidence of RMV and rate of disease progress were significantly reduced in the resistant genotypes relative to the susceptible genotype. The resistance reduced both rate of disease progress and final disease incidence of the MAV isolate during both years of the study. The magnitude of the reduction in final MAV incidence relative to the susceptible genotype varied between the 2 yr and could be related to vector population intensity and environmental factors. Resistance expressed as reduced or suppressed virus accumulation in plants can be easily identified by serological or nucleic acid-based assays. The lower virus titer may reduce disease impact on plant growth and can reduce the efficiency with which aphid vectors acquire and transmit virus among resistant plants.

Transmission efficiency of various barley yellow dwarf luteoviruses (BYDV) is differentially influenced by several factors, including aphid vector, length of acquisition and inoculation times, and physiological age of source tissue (9,14). In addition, virus titer can have a strong influence on acquisition and transmission efficiency of some BYDV isolates by their aphid vectors (9). Recently, we described a resistance in a spring oat genotype that was BYDV isolate specific and that was expressed as a reduction in virus antigen accumulation (10). The reduced virus antigen titer in the resistant oat genotype was also shown to play a significant role in reducing the transmission efficiency of some BYDV isolates by their aphid vectors. A similar type of resistance in potato to potato leafroll luteovirus (PLRV) has also been shown to reduce the acquisition and transmission efficiency of PLRV by an aphid vector (2,3). Furthermore, the spread of PLRV to PLRV-susceptible potato plants was decreased when resistant plants were the source of virus for aphid vectors (4). In general, PLRV titer in the source plant was positively correlated with final disease incidence within a row of susceptible plants flanking the source plant.

Reduced virus titer can affect vector transmission efficiency by reducing the overall number of vectors acquiring virus and/or by reducing the amount of virus taken up by the vector. Both mechanisms have been demonstrated for the uptake of PLRV by *Myzus persicae* (3). However, increasing acquisition access periods on low-titer virus sources can increase both the amount of luteovirus acquired by a vector (18) and the transmission efficiency (9). Therefore, the epidemiological significance of a resistance that is expressed as a reduction in virus content of a persistently transmitted virus will depend not only on the quantitative and qualitative traits of virus accumulation but also on the feeding behavior of the vector. The objective of this study was to quantify the development of epidemics induced by two isolates of BYDV in a BYDV-susceptible cultivar of spring oats and two spring oat genotypes that possess varying levels of resistance manifested as a reduction in titer.

MATERIALS AND METHODS

Virus isolates, aphid vectors, and host genotypes. The BYDV isolates used in these studies included New York MAV and RMV (NY-MAV and NY-RMV, respectively) previously characterized by Rochow (16). Isolates were maintained in Coast Black oats (*Avena byzantina* K. Koch) as described by Rochow (16).

The aphids used in all experiments were clonally propagated from aphids originally collected and described by Rochow, and his methods and rearing conditions were followed (16). In all the studies, NY-MAV was transmitted by *Sitobion avenae* (Fabricius) and NY-RMV by *Rhopalosiphum maidis* (Fitch).

Plant genotypes included two spring oat cultivars, Astro, which is susceptible to five New York isolates of BYDV, and Ogle, which is reported to be resistant to BYDV (7,10). In addition, a breeding accession, IL86 5262, developed at the University of Illinois and kindly provided by C. Brown was included. IL86 5262 had been field tested at Cornell University in 1987 and 1988 and found to possess excellent field resistance to the NY-PAV isolate of BYDV (S. M. Gray, *unpublished*). Coast Black, a winter oat cultivar that is susceptible to five New York isolates of BYDV, had been used as a susceptible oat cultivar in initial laboratory and greenhouse experiments to characterize the resistance in Ogle and IL86 5262 (10) but could not be used in subsequent field experiments because of the vernalization requirements. Astro was used as the susceptible oat cultivar in all field experiments.

Oat is not a preferred host of our biotype of *R. maidis*. Adult longevity and reproductive rate are reduced when the aphid is confined on oat, but the aphid will efficiently inoculate Coast Black and Astro plants with NY-RMV (S. M. Gray, *unpublished*). Ogle and IL86 5262 plants are more difficult to infect with NY-RMV, suggesting that these plants may possess some resistance to inoculation of NY-RMV by *R. maidis* in addition to the resistance to NY-RMV expressed as reduced virus antigen accumulation (10).

Experimental trials. Field experiments were conducted during 1990 and 1991 at the Caldwell field plots on the Cornell University campus. Experiments were established on 2 May 1990 and 19 April 1991 by hand planting seeds in rows spaced 18 cm apart with seeds spaced 5 cm apart within rows. A 3 × 3 Latin square with oat genotypes as treatments was used as the experimental design. Each of the nine plots was approximately 1 m^2 and contained six rows of 20 plants. Plots were separated by 1 m of fallow ground, and a 20-cm-wide, densely seeded border planted in IL86 5262 surrounded the nine plots to assist in preventing aphid immigration into the test plots. The entire 3 × 3 design, including the border, was duplicated. The two experimental arenas were separated by 4 m of fallow ground. One of the nine-plot arenas was used to study epidemics induced by NY-MAV and the other to study epidemics induced by NY-RMV.

When plants were at the two- to three-leaf stage, NY-RMV and *R. maidis* or NY-MAV and *S. avenae* were introduced into the nine-plot arenas by transplanting a hill of five BYDV-infected, aphid-infested plants at the center of each 1-m^2 plot. Each five-plant hill was infested with 20 adult aphids 1 wk prior to transplanting. The source plants were the same genotype as the remainder of the plants in the plot. These two isolates were chosen because Ogle and IL86 5262 were shown to possess various levels of resistance to these isolates (10). In addition, these isolates were not identified as being prominent local isolates infecting spring oats in surveys in previous years (S. M. Gray, *unpublished*). PAV is the most prevalent naturally occurring BYDV biotype or serotype infecting spring oats in the test plot area; therefore, plant tissue was tested by enzyme-linked immunosorbent assay (ELISA) with antibodies that recognize the NY-PAV, NY-MAV, and NY-RMV isolates. The results indicated the extent of virus dissemination between the two nine-plot arenas (NY-MAV and NY-RMV) or into the arenas from local sources (PAV serotypes).

Weekly sampling was conducted to determine disease incidence in each of the plots. The oat plants in each plot were subdivided into three concentric ring categories dependent upon their distance from the virus-aphid source plants; ring 1 = plants within 16 cm of the source (n = 12); ring 2 = plants 17–33 cm from the source (n = 32); and ring 3 = plants 34–50 cm from the source (n = 56). All plants were visually inspected for symptoms to assess disease incidence in the Astro plots. Confirmation of BYDV infection and isolate determination was done by using a previously described ELISA (9) on randomly selected samples of leaf tissue collected from symptomatic Astro plants. Infections of Ogle and IL86 5262 are often symptomless (10), and ELISA was used to determine infection status of individual plants. On each sampling date, samples consisting of 2- to 4-cm pieces of tissue from two to three leaves per plant were collected from 28–33% of the oat plants randomly selected from each of the three concentric rings (i.e., 4 plants in ring 1, 10 plants in ring 2, and 16 plants in ring 3). Each sample was individually tested for BYDV by ELISA. Differences in final disease incidence among genotypes were analyzed for each isolate independently by analysis of variance (ANOVA) by the Minitab procedure (12). Pairwise comparison of treatment means was done by the Minitab ONEWAY and Tukey procedures (12).

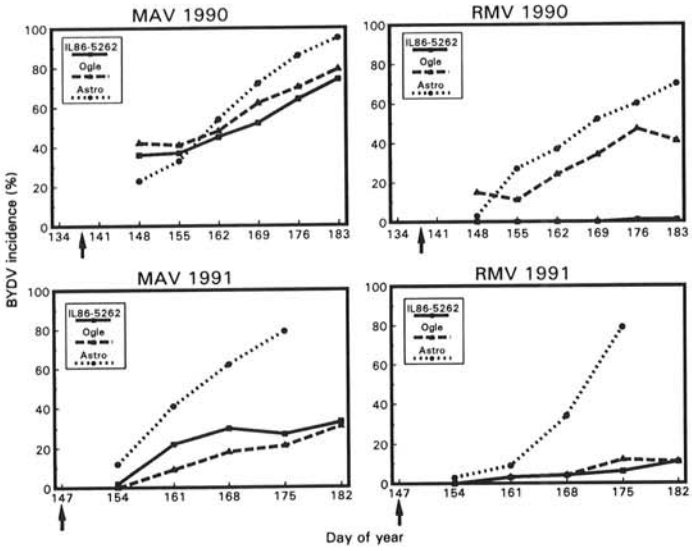

Fig 1. Disease progress curves for virus epidemics caused by two New York isolates (RMV and MAV) of barley yellow dwarf virus occurring in plots of three spring oat genotypes. Data are averages of disease incidence data collected from three replicate plots of each genotype during 1990 and 1991. Arrows indicate the dates a hill of five virus-infected, vector-infested plants was transplanted into the center of each test plot.

Analysis of disease progress. Disease progress was analyzed for each plot separately. Untransformed data or data transformed appropriately for the monomolecular, Gompertz, and logistic models (5,11) were analyzed by ordinary least squares regression techniques to estimate parameters of the linear model. Goodness of fit of the models to the data was tested by the Minitab REGRESS procedure (12). Coefficients of determination (R^2), coefficients of variation, and subjective evaluation of plots of standardized residuals vs. predicted values were used to indicate the appropriateness of a given model.

All epidemics did not reach an asymptote, because they were still in the increasing phase of the development when the crop became senescent. To obtain reliable estimates of the rate parameter (13), two empirical estimates of the asymptote were used in conjunction with the nonlinear models: 1.0 and the treatment maximum + 0.01. To compare rate of disease progress among epidemics for each of the different nonlinear models, the Richards rate parameter (15) was calculated and used in ANOVA, or the estimate of the slope (b_1) of the linear regression model for each treatment replicate was used in ANOVA.

Virus accumulation in field-grown plants. To determine whether resistance to MAV and RMV was expressed in field-grown plants of the three oat genotypes at levels similar to those that had been observed in greenhouse studies (10), weekly samples were collected and analyzed from the virus source plants transplanted

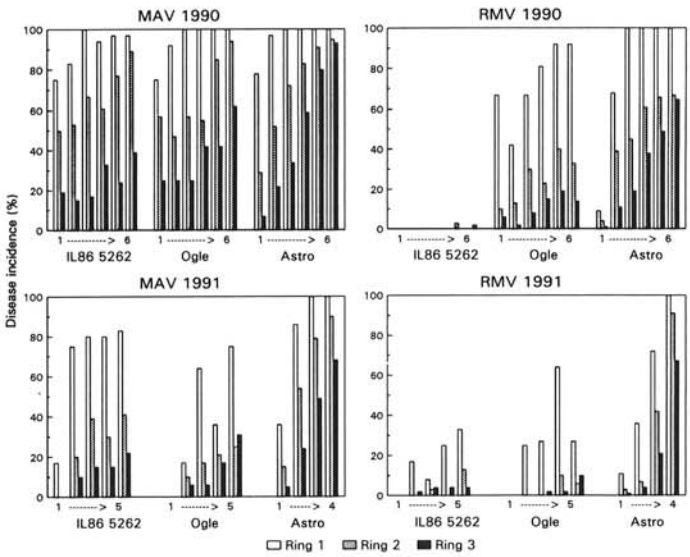

Fig 2. Disease incidence during 1990 and 1991 resulting from infection by the New York isolates MAV and RMV of barley yellow dwarf virus in three spring oat genotypes (IL86 5262, Ogle, and Astro) occurring at various distances from a virus and aphid vector source plant. Oat plants in 1-m^2 plots were divided into three groups on the basis of their distances from a hill of five virus-infected, vector-infested plants of the same genotype transplanted into the center of the plot: ring 1 (closest to the source plant), ring 2, and ring 3. The percentage of sampled plants determined by enzyme-linked immunosorbent assay to be infected with MAV or RMV is presented as a series of three-bar groupings in each graph. A three-bar group is presented for each sampling time. Each bar represents the mean incidence of three replicate plots.

into each plot in 1990. Each sample was a composite of tissue from several leaves from the hill. Virus titer was determined as previously described (9). Previous studies to characterize the resistance to various BYDV isolates in Ogle and IL86 5262 had used as a susceptible control a winter oat cultivar, Coast Black (10), but because of vernalization requirements, the susceptible spring oat cultivar Astro was substituted as the susceptible control in the field experiments. To ensure that Astro and Coast Black behaved similarly with respect to susceptibility and virus titer, studies to compare virus accumulation in both cultivars were done essentially as in the initial characterization studies described by Gray et al (10). Virus accumulation was compared with the NY-RMV, NY-MAV, and NY-PAV isolates in plants 2, 4, and 6 wk postinoculation.

Yield analysis. Individual plants whose infection history could accurately be determined were harvested from each genotype-virus isolate treatment and grouped into two categories: those plants infected within the first 3 wk of sampling and those infected within sampling weeks 4–7. The following yield measurements were determined for individual plants: overall plant height and mean total weight and straw weight and kernel weight on a per tiller basis. Plants of each genotype known to be healthy were harvested, and similar yield measurements were taken. Yield data were analyzed by ANOVA with the Minitab ANOVA and GLM (general linear model) procedures (12).

RESULTS

In neither of the 2 yr were plants infected with NY-RMV found in NY-MAV test plots, nor were plants infected with NY-MAV found in NY-RMV test plots, indicating an undetectable level of interarena movement. Visual inspection of plot maps marking the location of infected plants over time (data not shown) indicated minimal interplot movement within a nine-plot arena. Although we cannot rule out the possible infection of plants by MAV or RMV serotypes by aphids immigrating into plots from surrounding sources, this is unlikely. PAV was detected in less than 0.5% of the samples tested during 1990 and 1991.

Analysis of disease progress. Disease progress and final disease incidence data collected from each plot were analyzed independently, but the disease incidence data are presented in Figures 1 and 2 as an average of the three treatment replicates.

Final disease incidence of MAV was significantly different among genotypes in 1990 ($F = 7.62$; $P = 0.043$) and ranged from 0.70 to 0.79, 0.71 to 0.92, and 0.93 to 0.96 in the different plots of IL86 5262, Ogle, and Astro, respectively (Fig. 1). Pairwise comparisons of means of final disease incidence in each genotype indicate a significant difference ($P = 0.05$) only between IL86 5262 and Astro.

Final disease incidence of MAV was lower in all genotypes in 1991. The difference among genotypes was not significant ($F = 6.24$; $P = 0.059$) and ranged from 0.26 to 0.42, 0.21 to 0.43, and 0.52 to 0.97 in the different plots of IL86 5262, Ogle, and Astro, respectively (Fig. 1). Pairwise comparisons of mean final disease incidence indicated a significant difference ($P = 0.05$) between Astro and IL86 5262 and between Astro and Ogle but not between Ogle and IL86 5262.

Nonlinear models were judged to give the best fit to five of the nine disease progress curve data sets collected from the MAV-infected plots during 1990. The logistic model best described three data sets, and the monomolecular model best described two data sets. However, a linear model was judged to give the best overall fit to the disease progress data sets (Table 1). The slope (b_1) of the linear regression for each disease progress curve data set was used as an estimate of the rate of disease progress and was used to calculate the difference in disease progress rates among cultivars. The slightly better fit of some of the data by the nonlinear models did not warrant the use of the Richards rate parameters calculated from these models in comparing the rate of disease progress among epidemics, nor would the use of the Richards rate parameters have altered the results given below (data not shown). When the slope (b_1) calculated from the linear regressions was used, the mean rates of disease progress within the 1-m^2 plots were significantly different ($F = 21.37$; $P = 0.007$) among genotypes, being highest for Astro (0.153) and similar for Ogle (0.082) and IL86 5262 (0.080). Pairwise comparisons of mean rate parameters indicated no significant difference ($P = 0.05$) between Ogle and IL86 5262.

Analysis of disease progress data collected from the 1991 MAV plots gave results similar to those found in 1990. Nonlinear models in which the treatment asymptote was used were judged to give the best fit to three sets of disease progress data; however, the linear model was judged to give an acceptable fit to these data as well as to provide the best fit to disease progress curve data from the remaining plots (Table 1). The slope (b_1) of the linear regression line for each disease progress curve data set was used as an estimate of the rate of disease progress. When the rate parameters calculated from linear models were used, the difference among genotypes was significant ($F = 8.81$; $P = 0.034$), and pairwise comparisons indicated a significant difference ($P = 0.05$) between Astro and IL86 5262 and between Astro and Ogle but not between IL86 5262 and Ogle.

Final disease incidence of NY-RMV was significantly different among genotypes in 1990 ($F = 48.22$; $P = 0.002$) and ranged from 0 to 0.03, 0.37 to 0.44, and 0.56 to 0.84 in the different plots of IL86 5262, Ogle, and Astro, respectively (Table 2). Only two plants in one of the IL86 5262 plots contained detectable NY-RMV antigen. Pairwise comparisons of mean final disease incidence indicated a significant difference ($P = 0.05$) between all cultivar pairs.

The linear model was judged to give the best overall statistical fit to disease progress data from all the 1990 RMV plots. Rates were significantly different among genotypes ($F = 21.50$; $P = 0.007$) and between each possible pairwise comparison; the mean

TABLE 1. Final disease incidence and rate of disease progress of epidemics caused by the NY-MAV isolate of barley yellow dwarf virus in three replicates of three spring oat genotypes during 1990 and 1991

	1990			1991		
Genotype	Final disease incidence	Rate[y] (b_1)	R^2[z]	Final disease incidence	Rate (b_1)	R^2
IL86 5262-1	0.79	0.071 (0.015)	0.84	0.42	0.085 (0.023)	0.82
IL86 5263-2	0.70	0.064 (0.015)	0.82	0.26	0.056 (0.019)	0.74
IL86 5262-3	0.73	0.104 (0.008)	0.98	0.31	0.059 (0.038)	0.44
Ogle-1	0.74	0.108 (0.017)	0.91	0.43	0.089 (0.024)	0.82
Ogle-2	0.71	0.043 (0.025)	0.41	0.21	0.062 (0.012)	0.90
Ogle-3	0.92	0.096 (0.006)	0.98	0.30	0.074 (0.012)	0.93
Astro-1	0.95	0.164 (0.023)	0.93	0.52	0.136 (0.008)	0.99
Astro-2	0.93	0.130 (0.012)	0.96	0.91	0.262 (0.021)	0.99
Astro-3	0.96	0.166 (0.011)	0.98	0.93	0.264 (0.038)	0.96

[y] The rate parameter of the epidemic estimated by the slope (b_1) of the linear regression. The number in parentheses is the standard deviation of the slope coefficient.

[z] Coefficient of determination; measure of the goodness of fit of the data to the regression line.

TABLE 2. Final disease incidence and rate of disease progress of epidemics caused by the NY-RMV isolate of barley yellow dwarf virus in three replicates of three spring oat genotypes during 1990 and 1991

Genotype	1990			1991		
	Final disease incidence	Rate[y] (b_1)	R^{2z}	Final disease incidence	Rate (b_1)	R^2
IL86 5262-1	0.00	0.000	...	0.10	0.027 (0.010)	0.71
IL86 5262-2	0.00	0.000	...	0.13	0.029 (0.012)	0.66
IL86 5262-3	0.03	0.000	...	0.10	0.017 (0.013)	0.37
Ogle-1	0.41	0.062 (0.023)	0.65	0.20	0.050 (0.012)	0.86
Ogle-2	0.44	0.085 (0.015)	0.89	0.03	0.013 (0.024)	0.09
Ogle-3	0.37	0.064 (0.015)	0.82	0.10	0.030 (0.006)	0.88
Astro-1	0.56	0.098 (0.011)	0.95	0.85	0.290 (0.040)	0.96
Astro-2	0.70	0.116 (0.016)	0.93	0.73	0.230 (0.073)	0.83
Astro-3	0.84	0.172 (0.020)	0.95	0.78	0.239 (0.087)	0.79

[y] The rate parameter of the epidemic estimated by the slope (b_1) of the linear regression. The number in parentheses is the standard deviation of the slope coefficient.

[z] Coefficient of determination; measure of the goodness of fit of the data to the regression line.

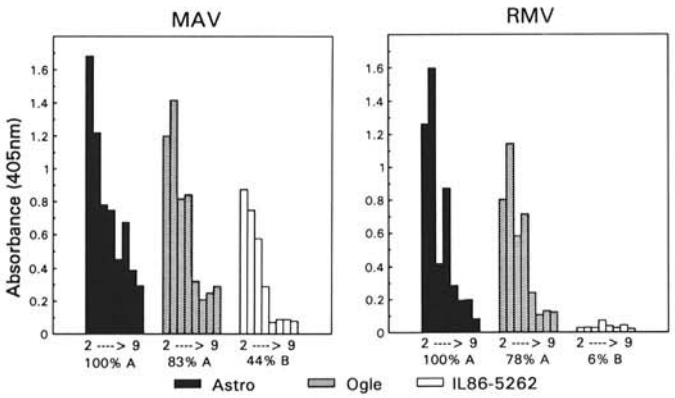

Fig 3. The temporal pattern of accumulation of two New York isolates (MAV and RMV) of barley yellow dwarf virus (BYDV) antigen, determined by enzyme-linked immunosorbent assay, in a BYDV-susceptible oat genotype, Astro, and two oat genotypes, Ogle and IL86-5262, possessing isolate-specific resistance to BYDV. Bars from left to right (in each eight-bar group) indicate mean levels of antigen ($n = 3$) that accumulated in the aerial portions of field-grown plants 2–9 wk postinoculation, respectively. Percentages given below each eight-bar group indicate the 8-wk mean level of virus antigen related to 8-wk mean antigen levels in the susceptible Astro genotype. Percentages followed by the same letter are not significantly different ($P = 0.05$) in pairwise comparisons of 8-wk mean levels of MAV or RMV antigen.

rate was highest in Astro (0.128), intermediate in Ogle (0.070), and lowest in IL86 5262 (0.00).

RMV epidemics were slow to initiate in all genotypes during 1991. Zero disease incidence was recorded in all plots on the first sampling date, and epidemics did not initiate until week 3 or 4 in five of nine plots. Final disease incidence was significantly different among genotypes during 1991 ($F = 168.89$; $P < 0.001$) and ranged from 0.10 to 0.13, 0.03 to 0.20, and 0.73 to 0.85 in the different plots of IL86 5262, Ogle, and Astro, respectively (Table 2). Pairwise comparisons of mean final disease incidence indicate no significant difference ($P = 0.05$) existed between IL86 5262 and Ogle.

The reduced number of nonzero data points for each RMV disease progress curve from 1991 made analysis with nonlinear models inappropriate for comparing mean rates of disease progress. Regression analysis was performed on nontransformed data. Rate of disease progress was significantly different among genotypes ($F = 183.24$; $P < 0.001$), but there was no significant difference ($P = 0.05$) between IL86 5262 and Ogle in pairwise comparisons.

Aphid and virus dispersal from the source plant. Aphid distribution data were not collected, but general observations were made as plant samples were collected for virus analysis. During 1990, both aphid species released were found on plants in the appropriate test plots throughout the growing season. This was also the case during 1991, although hot, dry weather reduced the aphid populations during 1991 relative to 1990.

TABLE 3. Analysis of variance of virus antigen titer of three barley yellow dwarf virus (BYDV) isolates between two BYDV-susceptible oat cultivars, Ogle and Coast Black, at 2, 4 and 6 wk postinoculation

BYDV isolate	Source	Degrees of freedom	F	P
RMV	Cultivar	1	1.13	0.305
	Week	2	5.61	0.015
	Cultivar × week	2	0.63	0.545
	Error	15	...	...
MAV	Cultivar	1	0.02	0.894
	Week	2	7.16	0.005
	Cultivar × week	2	0.62	0.547
	Error	15	...	...
PAV	Cultivar	1	0.76	0.396
	Week	2	5.59	0.013
	Cultivar × week	2	0.53	0.599
	Error	15	...	...

Incidence of NY-RMV and NY-MAV was highest in Astro plants closest to the source, but incidence within each ring increased consistently and rapidly throughout both growing seasons (Fig. 2). The number of infected plants in the Ogle or IL86 5262 plots also was higher nearer the source; however, in contrast to the Astro plots, disease incidence in the Ogle and IL86 5262 plots did not always increase at each subsequent sampling date. The percentage of infected plants tended to level off or to increase slowly following an initial flush of infections, especially in the sampling areas farther from the source plant (i.e., rings 2 and 3).

Virus accumulation in field-grown plants. The temporal pattern of accumulation of virus antigen (NY-RMV or NY-MAV) was consistent among genotypes, with the exception of NY-RMV in IL86 5262, which was not detected at significant levels at any time (Fig. 3). Generally, titers were highest in the first few weeks of sampling and then dropped off significantly. The ELISA for NY-RMV antigen in IL86 5262 plants inoculated with NY-RMV was often unable to confirm the infection. *R. maidis* nymphs were allowed a 48- to 72-h acquisition access period on leaf tissue from the "ELISA-negative" plants before transfer (in groups of 10–15) to healthy Coast Black oat indicator plants. NY-RMV was transmitted from all plants tested, indicating that the plants were infected but that the level of viral antigen was low. Mean virus titer was significantly different among genotypes for both NY-MAV ($F = 87.27$; $P = < 0.001$) and NY-RMV ($F = 129.34$; $P < 0.001$); however, pairwise comparisons indicated no significant difference ($P = 0.05$) in mean virus titer between Astro and Ogle for either virus.

The accumulation of NY-RMV, NY-MAV, and NY-PAV in Astro and Coast Black was not significantly different with regard

TABLE 4. Yield data of plants of three spring oat genotypes infected by two isolates of barley yellow dwarf virus during 1991 at various times after plant emergence

Genotype	Virus isolate	Infection group[x]	n[y]	Number of tillers	Plant height (cm)	Tiller weight (g)	Straw weight (g)	Kernel weight (g)
Astro	MAV	Healthy	15	5.5 ± 1.6 ab[z]	89.8 ± 7.2 a	1.59 ± 0.43 a	0.78 ± 0.21 ab	0.76 ± 0.24 a
		1–3 wk	83	4.2 ± 1.7 a	66.0 ± 6.8 c	1.60 ± 0.60 a	0.76 ± 0.26 a	0.80 ± 0.33 a
		4–7 wk	82	7.0 ± 3.2 b	72.2 ± 9.0 b	1.60 ± 0.50 a	0.89 ± 0.34 b	0.71 ± 0.26 a
Ogle	MAV	Healthy	10	4.2 ± 1.8 a	105.7 ± 3.9 a	2.70 ± 0.76 a	1.22 ± 0.29 a	1.47 ± 0.52 a
		1–3 wk	31	4.5 ± 1.8 a	79.9 ± 8.1 b	2.13 ± 0.63 a	0.92 ± 0.29 b	1.21 ± 0.37 a
		4–7 wk	15	4.9 ± 1.5 a	79.1 ± 10.2 b	2.29 ± 0.81 a	0.98 ± 0.36 ab	1.31 ± 0.49 a
IL86 5262	MAV	Healthy	14	5.4 ± 1.6 ab	108.4 ± 3.9 a	2.93 ± 0.61 a	1.39 ± 0.33 a	1.53 ± 0.36 a
		1–3 wk	84	4.5 ± 1.9 a	83.7 ± 9.5 b	1.97 ± 0.70 b	0.92 ± 0.36 b	1.05 ± 0.41 b
		4–7 wk	50	5.9 ± 2.5 b	84.8 ± 8.9 b	2.03 ± 0.59 b	1.02 ± 0.34 b	1.02 ± 0.35 b
Astro	RMV	Healthy	15	5.5 ± 1.6 ab	89.8 ± 7.2 a	1.54 ± 0.43 a	0.78 ± 0.21 a	0.76 ± 0.24 a
		1–3 wk	64	4.2 ± 1.8 a	75.4 ± 7.9 b	1.72 ± 0.58 a	0.84 ± 0.26 a	0.88 ± 0.35 a
		4–7 wk	84	5.5 ± 2.1 b	77.6 ± 7.5 b	1.89 ± 0.56 a	1.03 ± 0.35 b	0.85 ± 0.30 a
Ogle	RMV	Healthy	10	4.2 ± 1.8 a	105.7 ± 3.9 a	2.70 ± 0.76 a	1.22 ± 0.29 a	1.48 ± 0.52 a
		1–3 wk	6	4.3 ± 1.4 a	83.8 ± 9.7 b	2.42 ± 0.93 a	0.92 ± 0.35 a	1.50 ± 0.60 a
		4–7 wk	5	4.4 ± 1.1 a	85.8 ± 8.5 b	2.44 ± 0.74 a	1.05 ± 0.28 a	1.39 ± 0.47 a

[x] Plants not infected with virus or infected within 1–3 or 4–7 wk after emergence.
[y] Total number of plants from which yield data were collected and averaged.
[z] Mean ± standard deviation. Means followed by the same letter are not significantly different ($P = 0.05$) in pairwise comparisons.

to virus antigen titer at each time point after inoculation or with regard to the temporal pattern of accumulation, as indicated by the cultivar × week interaction term in the ANOVA (Table 3).

Analysis of yield data. The height of infected plants was significantly less ($P = 0.05$) than that of healthy plants, regardless of BYDV isolate, plant genotype, or time of infection (Table 4). The number of tillers and straw weight also tended to be reduced in plants infected early in the growing season (1–3 wk after emergence). Tiller and kernel weights were significantly affected only in IL86 5262 by NY-MAV infection. BYDV symptoms were mild or not expressed in Ogle or IL86 5262 during 1990 but were observed in all three genotypes during the hot, dry 1991 growing season.

DISCUSSION

The application of the linear model to estimate the rate of disease increase (dy/dt) cannot be used to predict or explain the biological characteristics of the epidemics, because the absolute rate would be independent of disease incidence throughout the epidemic. The linear model was used as an approximation of a more complicated model that cannot be determined from the data generated from these experiments. The models do, however, provide some information on the epidemiological benefits of the resistance or resistances expressed in Ogle and IL86 5262. The rate of disease progress was significantly less in experimental plots planted in resistant Ogle or IL86 5262 during both years of study and for both virus isolates.

Reduced virus antigen titer in Ogle and IL86 5262 was likely to contribute to the reduction in final disease incidence and rate of disease progress relative to levels in the susceptible Astro plots. Reduced viral antigen in IL86 5262 was previously correlated with reduced levels of transmission of RMV and MAV by *R. maidis* and *S. avenae,* respectively (10). Similar types of resistance have been shown to contribute to reduced virus incidence of other aphidborne viruses (4,8). The effectiveness of the resistance at reducing aphid transmission efficiency is dependent upon the duration of the acquisition period. The reduced titer increases the time necessary for aphids to acquire enough virus to ensure transmission; however, if allowed a long enough acquisition feeding period, a vector may acquire enough virus to become viruliferous despite the low virus titer in the source tissue (9,10).

Observations of *S. avenae* populations on the three oat genotypes indicated that aphid populations were smaller on Ogle and IL86 5262 relative to Astro. In addition, the aphids on IL86 5262 and Ogle tended to move off the source plants more quickly than those on Astro, supporting an earlier hypothesis that these two genotypes possess some aphid resistance in addition to the BYDV resistance (10). The disease distribution in the plots over time (Fig. 2) lends some support to the idea of aphid resistance. Disease distribution in the Astro plots over time is indicative of uniform movement of vectors and virus outward from the center source. It appeared there was continuous spread of the virus in these plots throughout the growing season. This was in contrast to the disease distribution in the resistant genotypes, in which there was a rapid initial increase in disease incidence throughout the plot followed by a lag phase before a slower rate of disease increase in the later part of the season. This is indicative of a rapid initial dispersion of aphids from the source plant, resulting in many primary infections throughout the plot. Reduced virus accumulation in the plants coupled with a reduced aphid population buildup would delay and suppress the occurrence of secondary infections within the Ogle and IL86 5262 plots. The result is a lower rate of disease progress and a lower final disease incidence.

The resistance to NY-MAV in IL86 5262 was expressed at levels similar to those reported from a greenhouse study, although the level of resistance in Ogle was less than previously found (10). This may be explained by differences in virus accumulation in greenhouse-grown vs. field-grown plants and also by the use of different sampling methods. The reduction in MAV titer may reduce transmission efficiency for short acquisition feeding periods but not for longer acquisition feeding periods (9,10). Ideal growing conditions during 1990 favored population development and plant growth, which may minimize the epidemiological benefits of both virus and aphid resistance. MAV disease progress curves were similar for all three oat genotypes during 1990. During 1991, hot, dry conditions minimized the aphid populations, increased their movement within and among plants, and contributed to an early maturing crop. The virus and aphid resistances in Ogle and IL86 5262 were more effective in reducing disease progress.

The resistance to NY-RMV in IL86 5262 and Ogle was expressed at similar levels in the greenhouse, resulting in virus antigen levels about five times lower than those in susceptible genotypes (10). The resistance in IL86 5262 was also expressed at high levels in field-grown plants tested during the course of this study (Fig. 3). The extreme reduction in virus available to aphids caused a reduced transmission efficiency and resulted in excellent control of NY-RMV during both years. Any aphid resistance that may be present in this genotype would add to the benefits of reduced titer by reducing feeding times of the aphids and further reducing acquisition and inoculation efficiencies (9,14). The resistance in field-grown Ogle plants was not expressed at levels indicated by the previous greenhouse tests. Mean virus antigen titer in field-

grown Ogle plants was not significantly lower than that in the susceptible Astro plants during the course of the study (Fig. 3). In the previous study (10), virus antigen titer was determined from whole plants, whereas in this study, individual leaves were sampled. This may account for the discrepancies between the mean titer in Ogle plants, since virus is not uniformly distributed. Alternatively, the significantly lower disease incidence in the Ogle plots relative to the Astro plots may be caused by reduced aphid populations or by the possibility that the aphids did not prefer to feed on the Ogle plants. Oat is not a preferred host of *R. maidis,* the primary vector of NY-RMV. This nonpreference reaction may be exacerbated by any aphid resistance and may be primarily responsible for the reduction in disease in the Ogle plots relative to the susceptible Astro plots.

The resistance in the oat genotype IL86 5262 needs further characterization, but it is the first resistance in oat to BYDV that has been characterized for a number of BYDV isolates both in greenhouse and field studies and found to be beneficial in terms of reducing overall disease incidence and rate of disease progress. The resistance is not effective against all BYDV isolates, and the level of resistance expressed varies depending on the infecting BYDV isolate (10). It is encouraging that high levels of resistance to BYDV exist in oat germ plasm and that the resistance may be identified by a relatively simple screening process that examines virus accumulation in plants. Reduced virus titer can result in a lower transmission efficiency and a subsequent reduction in disease incidence. However, in view of the findings presented here and in other studies, resistance to one BYDV isolate does not indicate resistance to all BYDV isolates (1,17). In addition, resistance identified in greenhouse experiments may not be expressed in the field (6). We must now learn to recognize the limitations of this type of resistance and improve its benefits by incorporating the resistance with other complementary types of control measures.

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