Ecology and Epidemiology

Virus-Suppression and Aphid Resistance Effects on Spatial and Temporal Spread of Watermelon Mosaic Virus 2

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ABSTRACT

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The spatial and temporal characteristics of epidemics induced by watermelon mosaic virus 2 (WMV 2) were monitored in replicated plantings of three genotypes of *Cucumis melo*. One genotype was resistant to *Aphis gossypii* and suppressively resistant to WMV 2, another was resistant to *A. gossypii*, and a third was a commercial variety susceptible to both WMV 2 and *A. gossypii*. Fourteen epidemics in two nonoverlapping plantings were analyzed separately. Final virus incidence in the aphid-resistant genotype and the aphid/virus resistant genotype averaged 11 and 33% lower, respectively, than that in the susceptible genotypes during the spring planting. The nine epidemics in the different genotypes of the spring planting were statistically best described by various nonlinear models. The rate of disease progress also varied among genotypes. Infected plants of all three muskmelon genotypes were consistently observed in a clustered pattern, but the degree of clustering differed among genotypes.

The five epidemics occurring during the summer planting were also described by different nonlinear models, but the best single-model, statistical fit of the disease progress data from these epidemics was provided by the linear model. The rate of disease progress and final disease incidence were not significantly different among genotypes and the infected plants were observed in a random pattern. The increased incidence of WMV 2 during the summer planting was attributed to an increase in the number of alighting aphids and sources of WMV 2 in the surrounding area. The effects of the seasonal abundance and species composition of alate aphid populations, the amount and proximity of virus sources, and the effectiveness of the different resistance components are discussed in relation to the field epidemics of WMV 2.

Additional key words: epidemiology, potyvirus.

Recently, a quantitative form of resistance to WMV 2, which suppressed the level of virus multiplication, was reported in the *Cucumis melo* L. genotype 91213 (19). This genotype also possesses antibiosis/antixenosis mediated resistance to *Aphis gossypii* Glover (11,12), which is the only aphid species regularly colonizing *C. melo* in North Carolina. Romanow et al (23) have quantified the effects of both resistance components on acquisition and inoculation of WMV 2 by *A. gossypii* and *Myzus persicae* Sulzer. The suppressive virus resistance reduced the acquisition efficiency of WMV 2 by aphids from 91213 relative to that from virus-susceptible genotypes. The aphid resistance was specific for *A. gossypii* and reduced the efficiency with which *A. gossypii*, but not *M. persicae* inoculated the plants with WMV 2. Preliminary field studies indicated that one or both forms of resistance may significantly reduce the final incidence of WMV 2 (19).

The transmission of aphid-borne, nonpersistent viruses has been shown to be reduced in cultivars or breeding lines of several hosts

that possess some form of resistance to virus infection (1,2,23,26,27) or to aphid vectors (9,14). The epidemiological significance of these types of resistance has been described qualitatively for several host/virus/vector systems (2,16,19,27), but there is little quantitative information available (18).

The aphid/virus/plant system we are investigating resembles that of Lecoq et al (13–16). They have reported that resistance to *A. gossypii* in the *C. melo* genotype Songwhan Charmi (SC) was closely associated with resistance to inoculation by cucumber mosaic virus (CMV), WMV 1 and WMV 2 by *A. gossypii*, but not to inoculation of those viruses by other aphids (14). The resistance components delayed the development of epidemics induced by CMV in the resistant SC genotype relative to a susceptible genotype (16). It is unclear from the data, however, whether the delay in the epidemic was a result of reduced acquisition and subsequent spread of CMV by aphids from infected SC plants or to the resistance of SC to *A. gossypii* and the associated resistance to inoculation by *A. gossypii*, or to an interaction of both.

The objective of this study was to quantify the spatial and temporal development of epidemics induced by WMV 2 in a susceptible cultivar of *C. melo* and in genotypes possessing either aphid resistance or aphid resistance coupled with suppressive virus

resistance. We also sought to elucidate the type of virus spread (primary or secondary) that occurred and to explain any differences observed among the epidemics.

MATERIALS AND METHODS

Data collection. Field experiments were conducted during 1984 at the Horticultural Crops Research Station, Clinton, NC. Experiments were established on 27 April and on 6 August. Seeds were sown in seedling trays and transplanted into field plots at the one- to two-leaf stage for the first (spring) experiment. Direct seeding into plots was used for the second (summer) experiment. Three *C. melo* genotypes were planted; Top Mark, a commercial cultivar, susceptible to both WMV 2 and *A. gossypii*; 91213 (described above) and Aphid-Resistant Top Mark (AR-Top Mark) (23), which possesses only the antibiosis/antixenosis resistance to *A. gossypii* found in 91213 but lacks the resistance of 91213 to virus multiplication. A 3×3 Latin square with genotypes as treatments was used as the experimental design to minimize any directional effects of incoming vectors. Each of the nine plots contained 10 rows of 20 plants, with a row spacing of 1.5 m and a plant spacing within rows of 0.6 m. Plots were separated by about 6 m of fallow. Field limitations required a reduction in the number of plots to six in the summer experiment; therefore, one of the three columns of the Latin square was eliminated.

To ensure adequate levels of WMV 2 in the spring experiment, 20 plants in each plot, arranged as ten, randomly selected, within-row pairs of plants, were inoculated mechanically with an isolate of WMV 2 that had been maintained by aphid transmission. The actual proportion of plants in each plot which became infected by mechanical inoculation is given in Table 1. No plants were artificially inoculated in the summer experiment.

Plants from the spring experiment were sampled weekly for 8 wk (28 May–17 July). Plants from the second experiment were sampled five times from 28 August through 2 October. For each sample, six leaf disks (2 cm diameter) were removed from a minimum of three leaves per plant. When possible, samples were taken from the second or third leaf from the growing tips. A modified ELISA protocol (19) was used to assay each six-disk sample for WMV 2. All assays were conducted within 48 hr of collection. Plant extracts (about 1:40 dilution, w/v) were prepared in conjugate buffer (PBS-T + 2% PVP 40,000) using a Virtis 45 homogenizer with an Ultrashear blade (Virtis Corp. Garden City, NY). Each sample was replicated in two wells. Tests were considered positive if the optical density values at 405 nm of both wells were more than double the mean value of the healthy controls. Plants with positive test results for three consecutive sampling dates were eliminated from further sampling.

Alighting alate aphids were monitored using horizontal ermine lime green traps (8). One trap was placed in the center of each plot. Aphids were collected and preserved in 95% ethanol each day leaf samples were collected.

Analysis of disease progression. Disease progress was analyzed for each plot separately. Data taken on 18 June (Julian date 169) were used as the starting point for epidemic analysis in the spring experiment. Thus, five observation dates were included in the analysis. Data from all the observation dates were used for epidemic analysis in the summer experiment. Disease incidence data were transformed using the linearizing transformation appropriate for the monomolecular, Gompertz, and logistic models (3,17) before using ordinary least squares regression techniques to estimate parameters of the linear model. Transformed data were tested for goodness-of-fit to the models using the General Linear Model procedure of the Statistical

TABLE 1. Nonlinear models that provide the best statistical fit[a] to disease incidence of WMV-2 on three muskmelon genotypes grown during the spring of 1984

Genotype[b] plot no.	Proportion of plants mechanically inoculated	Final disease incidence	Model[c]	Y-max[d]	R^2	CV	Rho[e]
91213-5	0.065	0.29	M	1.0	0.98	10.2	0.0055
			L	0.29	0.78	180.0	0.0152
91213-3	0.040	0.11	G	1.0	0.96	5.8	0.0073
			G	0.29	0.96	16.4	0.0033
91213-7	0.005	0.11	G	1.0	0.96	7.1	0.0070
			G	0.29	0.97	15.7	0.0034
	Mean Rho (Y-max = 1.0) 0.0066 A[f] Mean Rho (Y-max = 0.29) = 0.0073 A						
ARTM-4	0.045	0.35	G	1.0	0.99	7.4	0.0135
			L	0.35	0.82	386.5	0.0217
ARTM-2	0.055	0.31	L	1.0	0.96	13.8	0.0220
			L	0.35	0.94	62.0	0.0111
ARTM-9	0.045	0.28	G	1.0	0.95	19.5	0.0170
			G	0.35	0.98	78.0	0.0100
	Mean Rho (Y-max = 1.0) = 0.0175 B Mean Rho (Y-max = 0.35) = 0.0145B						
TM-1	0.045	0.65	L	1.0	0.99	13.6	0.0172
			L	0.65	0.73	177.8	0.0358
TM-8	0.040	0.57	L	1.0	0.98	14.7	0.0252
			L	0.65	0.97	38.9	0.0204
TM-6	0.040	0.38	L	1.0	0.99	4.3	0.0198
			L	0.65	0.99	6.6	0.0143
	Mean Rho (Y-max = 1.0) = 0.0207C Mean Rho (Y-max = 0.65) = 0.0235B						

[a] Coefficients of determination (R^2), coefficients of variation (cv) and subjective evaluation of plots of standardized residuals vs. predicted values were the criteria used to chose the most appropriate model.

[b] 91213 a *Cucumis melo* genotype possessing suppressive virus resistance to WMV 2 and antibiosis/antixenosis mediated resistance to *Aphis gossypii* ARTM (Aphid-resistant Top Mark) possesses the same antibiosis/antixenosis mediated resistance to *A. gossypii*, TM (Top Mark) is susceptible to WMV 2 and colonization of *A. gossypii*.

[c] The models used were the integrated forms of the following: logistic (L) $dy/dt = ry(1 - y)$, Gompertz (G) $dy/dr = ry(-\ln y)$, monomolecular (M) $dy/dt = r(1 - y)$.

[d] Y-max is the asymptote parameter used in the models to indicate the maximum disease level. The treatment (genotype) maximum + 0.01 and 1.0 were used for each epidemic.

[e] Rho is the Richards rate parameter or weighted mean absolute growth rate and is calculated as Rho $= Ar/(2m + 2)$ where $A =$ Y-max, r is the slope (b_1) of the regression line and represents an estimate of the rate parameter for the specific model, and $m = 0$, 1, or 2 for the monomolecular, Gompertz, or logistic model, respectively.

[f] Mean Rho value of the Y-max, followed by the same letter are not significantly different ($P = 0.05$) according to the Waller-Duncan K-ratio t-test.

Analysis System (24). 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.

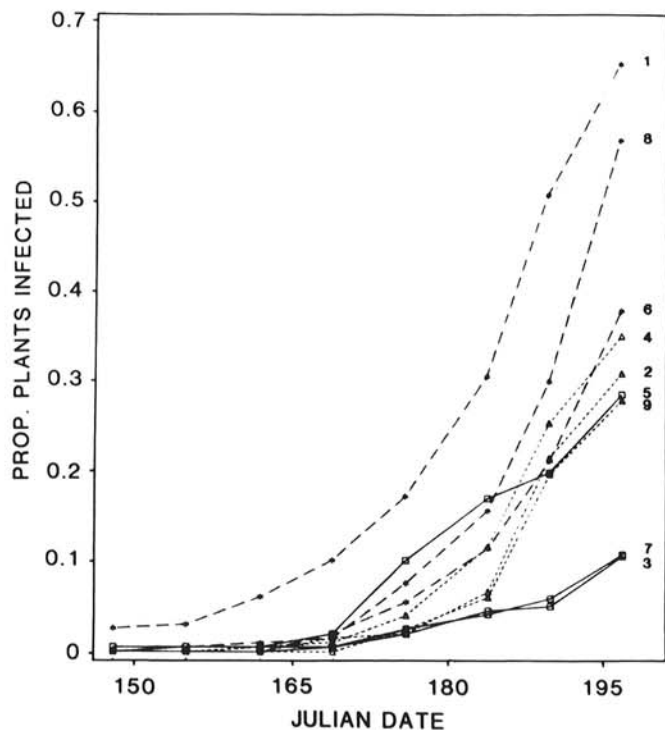All epidemics in the spring experiment did not reach an

Fig. 1. Untransformed disease progress curves for WMV 2 epidemics occurring in individual plots of the Top Mark (stars and dashed lines), AR-Top Mark (Δ - - - Δ) and 91213 (□······□) muskmelon genotypes during the spring planting. The numbers to the right of each curve refer to the plot number.
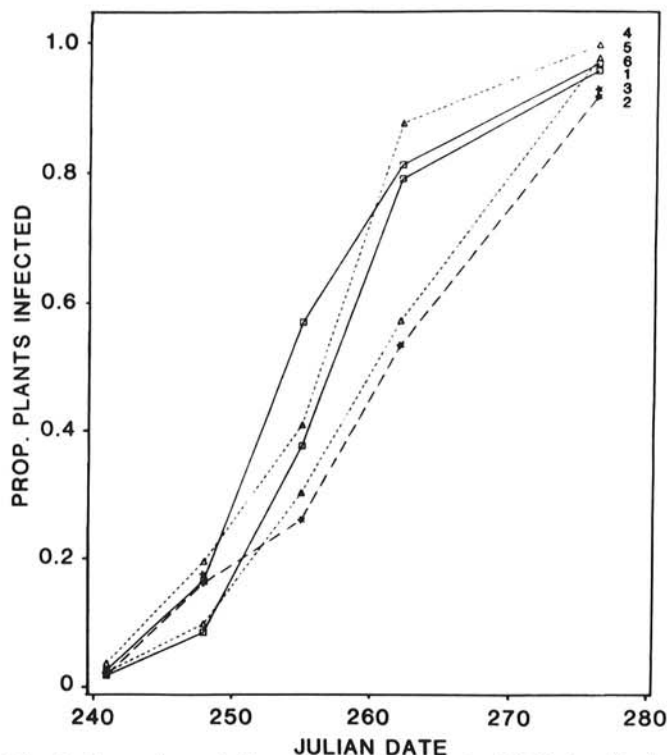

Fig. 2. Untransformed disease progress curves for WMV 2 epidemics occurring in individual plots of Top Mark (star and dashed line), AR-Top Mark (Δ- - - Δ) and 91213 (□······□) muskmelon genotypes during the summer planting. The number to the right of each curve refers to the plot number.

asymptote as they were still in the increasing phase of development at the time the crop was harvested. To obtain reliable estimates of the rate parameter (7,20), three empirical estimates of the asymptote were used: 1.0, the actual plot maximum + 0.01, and the treatment maximum + 0.01. Final disease incidence levels for all the epidemics occurring in the summer experiment were greater than 90%, therefore functions with an asymptote parameter, $A = 1.0$ were used in the analysis. Because different models were appropriate for statistically describing different epidemics, rates of increase (r) among epidemics were compared by an analysis of variance using the Richards rate parameter (rho weighted mean absolute growth rate) (22) defined as:

rho $= Ar/(2m + 2)$

where, A = asymptote parameter for disease,
r is the slope (b_1) of the regression line and represents an estimate of the rate parameter for the specific model, and
$m = 2$ for the logistic model, $= 1$ for the Gompertz model, $= 0$ for the monomolecular model.

Spatial pattern analysis. The positions of all infected plants in each plot were recorded in plot maps on each sampling date. The quantitative spatial pattern of infected plants was analyzed for representative test plots of each of the three muskmelon genotypes using a two-dimensional distance class analysis (6). Spatial analyses were performed on data from all sampling dates when disease incidence in the plot was between 10 and 60%.

RESULTS

Analysis of disease progression. Final disease incidence was significantly different among genotypes ($F = 9.80$, $P = 0.013$) for the spring crop (Fig. 1) and ranged from 0.38 to 0.65, 0.28 to 0.35, and 0.11 to 0.29 in the different plots of Top Mark, AR-Top Mark, and 91213, respectively. The susceptible genotype (Top Mark) had a significantly greater mean incidence of infected plants than either of the resistant genotypes (AR-Top Mark and 91213). Although the final incidence of infected plants was numerically greater in two of the AR-Top Mark plots than in the 91213 plots the differences were not significant ($P = 0.05$) according to the Duncan–Waller K-ratio t test. Epidemic onset occurred at the same time in eight of the nine plots (Fig. 1), and in plot 1 1–2 wk earlier.

The model that was judged to give the best fit to data from the disease progress curves of the spring crop depended on the asymptote value (A) used. When $A = 1.0$, one epidemic was described by the monomolecular model, four by the Gompertz model, and four by the logistic model (Table 1). When $A =$ treatment maximum (i.e., the maximum final disease incidence recorded for each muskmelon genotype), three epidemics were described by the Gompertz model, and six by the logistic model (Table 1). The worst statistical fit to data from the disease progress curves of the spring crop resulted when the actual final disease incidence of the plot being analyzed was used as the asymptote value (i.e., A = plot maximum); therefore, these models were eliminated from subsequent analyses.

Rates of disease progress were compared among muskmelon genotypes using rho values calculated from the model that provided best statistical fit to the disease incidence data for each epidemic. The mean rate of disease progress was highest for Top Mark, intermediate for AR-Top Mark, and lowest for 91213 when either an asymptote value of 1.00 or the treatment maximum was used. The differences among genotypes were not significant when the latter asymptote values were used (Table 1).

Final disease incidence was greater than 90% in all plots during the summer planting (Fig. 2), but there was a significant difference in the mean final disease incidence among genotypes ($F = 13.2$ $P = 0.033$). The incidence in the Top Mark plots was significantly lower than in both AR-Top Mark and 91213 as determined by the Duncan-Waller K-ratio t-test. Because of errors made during the ELISA analysis of the Top Mark samples from plot 3 on 11 and 18 September (Julian dates 255 and 262), the disease incidence and spatial pattern data were unreliable. Therefore, with the exception of final disease incidence, no spatial or temporal analyses of the disease incidence data from plot 3 (Top Mark) were possible.

Logistic errors when analyzing samples from Top Mark plot 2 on 11 and 18 September caused the loss of spatial pattern information, although disease incidence was determined.

The Gompertz model provided the best statistical fit to the transformed disease progress data for the epidemics in 91213, whereas the logistic model was best for AR-Top Mark and Top Mark (Table 2). Comparison of the rho parameters (Table 2) for the transformed data suggested that the epidemic in Top Mark progressed at a slower rate than those in AR-Top Mark or 91213. The best single model statistical fit to the untransformed data from each of the epidemics was provided by the linear model. A test of the homogeneity of slopes of the untransformed linear regression models indicated no significant difference among treatments (genotypes) ($F = 1.45$, $P = 0.258$).

Spatial pattern analysis. In the spring experiment, the WMV 2-infected plants occurred in clusters in all plots; however, the degree of clustering was different among muskmelon genotypes when the disease incidence exceeded 15%. The data presented in Figure 3 typify the spatial pattern of infected plants observed in all the test plots of a genotype. The grouping of significant distance classes in the upper left corner of Figure 3a indicates the infected Top Mark plants occurred in clusters of up to 20 plants orientated across rows, along rows, and diagonally, and cluster size increased over time (data not shown). The clusters were also randomly located in the plots, e.g., multiple clusters, such as the two distinct groupings of significant distance classes in Figure 3a, were evident in other Top Mark plots. Infected AR-Top Mark plants were arranged as doublets, either across rows [e.g., the significant (1,0) X, Y distance class in Fig. 3b] or along rows, and the doublets were located randomly in the lattice (e.g., Fig. 3b). When disease incidence exceeded 30%, runs of three to four plants occurred and a majority of the infected plants were at the plot periphery. Infected

91213 plants occurred in clusters of up to 10 plants orientated along columns and rows and diagonally (e.g., the grouping of significant distance classes in the upper left of Fig. 3c). The clusters of infected 91213 plants were located along the column edge of the field (as indicated by the grouping of significant distance classes in

TABLE 2. Characterization by least squares regression analysis of disease progression data ($Ymax = 1.0$) on three muskmelon genotypes grown in the summer of 1984

Genotype-plot #	Final disease incidence	Acceptable[a] model	b_1[b]	R^2	CV	Rho[c]
91213-5	0.97	Gompertz	0.140	0.99	55.2	0.035
91213-5	0.97	Linear	3.02	0.93	29.4	...
91213-2	0.96	Gompertz	0.143	0.99	13.2	0.036
91213-2	0.96	Linear	2.92	0.92	26.4	...
ARTM-3	1.0	Logistic	0.293	0.98	81.9	0.049
ARTM-3	1.0	Linear	0.149	0.92	27.6	...
ARTM-3	1.0	Gompertz	3.02	0.92	511.8	0.037
ARTM-6	0.98	Logistic	0.224	0.99	42.2	0.037
ARTM-6	0.98	Linear	2.89	0.98	15.6	...
TM-1	0.92	Linear	2.64	0.98	14.8	...
TM-1	0.92	Logistic	0.184	0.95	69.4	0.020
TM-1	0.92	Gompertz	0.110	0.97	271.0	0.027
TM-4	0.94	...	...	...	...	...

[a] Model acceptance was judged on the basis of R^2 values, cv, and the presence or absence of a discernible pattern in the plot of standardized residuals against predicted values.

[b] b_1 is an estimate of the slope, b_1 represents the rate parameter for each model.

[c] Rho is the Richards rate parameter, rho $= b_1/2m + 2$ where $m = 2$ for the logistic model and $m = 1$ for the Gompertz model.
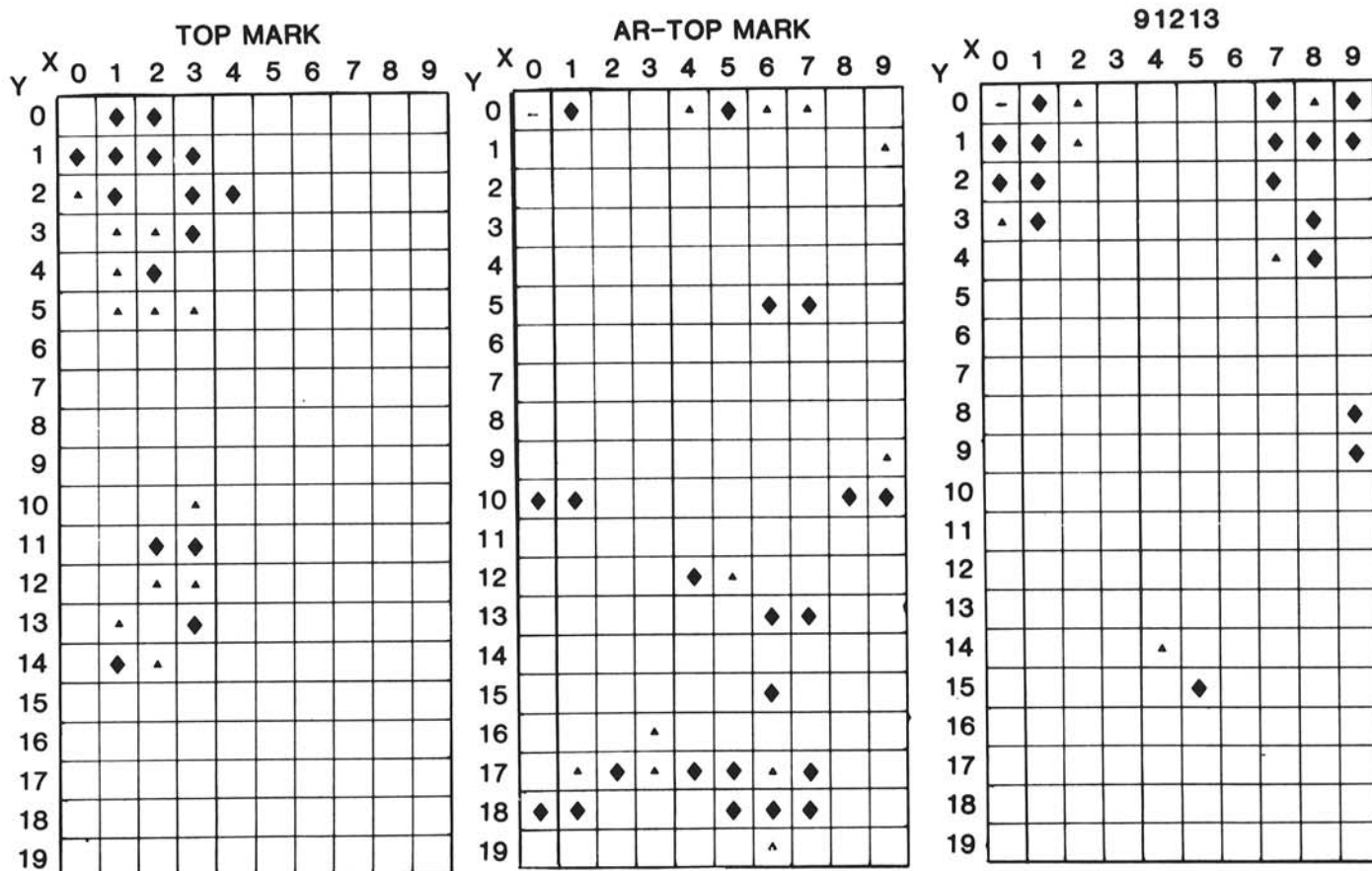

Fig. 3. Typical results of the two-dimensional distance class analysis describing the spatial arrangement of WMV 2 infected plants in representative plots of the three muskmelon genotypes during the spring. All possible pairs of infected plants within a test plot were categorized into 199 two dimensional, [X,Y], distance classes. The number of pairs of infected plants in each distance class were statistically analyzed to determine if there was a significant difference in the number of pairs of infected plants observed from that expected if the infected plants within the plot were randomly located. The number of pairs of infected plants in each marked distance class are significantly greater (◆ = $P = 0.05$, ▲ = $P = 0.1$) than expected. The disease incidence was 30, 22, and 17% in the Top Mark, AR-Top Mark, and 91213 plots, respectively.

columns 7, 8, and 9 of Fig. 3c) and spread within columns over time (data not shown).

In the summer experiment, AR-Top Mark and 91213 plants infected with WMV 2 were randomly distributed in their respective plots when disease incidence was below 46% (Fig. 4). Runs of up to five infected plants were evident in one AR-Top Mark plot and one 91213 plot when disease incidence was 46 and 56%, respectively (data not shown). The infected plants in the other AR-Top Mark and 91213 plots were randomly distributed throughout the experiment.

Seasonal abundance of alighting aphids. The numbers of aphids alighting in the test plots during the spring experiment increased two- to threefold in the final 2 wk of sampling. This increase coincided with an increase in disease incidence in the Top Mark plots (Fig. 5). Alighting aphid populations during the summer experiment were two- to 12-fold higher than the average number alighting in the spring (Fig. 5). *Aphis* sp. accounted for over half of all aphids collected in the first experiment, whereas during the second experiment they composed less than 10% of the total.

DISCUSSION

The mathematical description of virus disease epidemics by the logistic and Gompertz models has been associated with a polycyclic disease cycle and secondary virus spread (25). A clustered spatial pattern of infected plants has also been associated with secondary spread of aphid-borne nonpersistent viruses (25). The WMV 2 epidemics occurring in the spring experiment in all three muskmelon genotypes were statistically best described by the logistic or Gompertz model, with the exception of one epidemic in 91213, moreover, the WMV 2-infected plants were spatially clustered in all three muskmelon genotypes.

These characteristics suggest that the main sources of inoculum were infected plants within the experimental plots. *A. gossypii* colonies were not observed on the plants and few virus sources occurred near the test area. The spatial and temporal modeling

results and the observational data indicate that a majority of the disease incidence was due to secondary spread of the virus, by alate aphids (colonizing and/or noncolonizing), from a few initially infected plants within each test plot. The aphid resistance of AR-Top Mark and 91213 causes aphids to become more restless, which results in increased interplant movement (10,11). This behavioral effect would explain the smaller, more dispersed clusters of WMV 2-infected plants in aphid-resistant genotypes (i.e., AR-Top Mark and 91213) and a rate of disease progress in AR-Top Mark not significantly less than observed in the susceptible Top Mark.

Linear increases in virus incidence over time have been associated with spread from sources of inoculum that do not increase within the crop; i.e., primary spread from sources outside the test plots (25). Initial spread of a nonpersistent virus from infected sources outside the field leads to a random pattern of primary infections, which may or may not be followed by secondary spread within the crop to give rise to clusters of infected plants. Secondary spread will depend on the numbers and activity of the incoming viruliferous aphids as well as on interactions among host, virus, and aphids (10). The best single model statistical fit of the disease progress data from all the epidemics occurring in all three muskmelon genotypes during the summer experiment was provided by the linear models (Table 2), whose slopes (an estimate of overall rate of disease increase) were not significantly different among genotypes. The spatial pattern of WMV 2-infected plants during the summer experiment also supports the hypothesis that an increase in disease resulted from primary spread (random distribution). Clusters of infected plants were evident depending on the level of disease incidence and the plot examined; however, the clusters were small and did not appear to increase in size over time (data not shown). Clustered patterns of infected plants may occur from primary spread if alighting viruliferous aphids were responsible for infecting multiple plants in close proximity to each other (4). Thus, the spatial and temporal trends of disease progress during the summer experiment suggests that a majority of the disease was caused by primary spread from outside sources and infected plants within the plot contributed little to further spread.

Moyer et al (19) reported a reduction in final disease incidence in 91213 and AR-Top Mark compared with Top Mark in preliminary field trials. In the present study, the final disease incidence was significantly reduced in 91213 and lower in AR-Top Mark relative to the susceptible Top Mark in the spring experiment (Fig. 1), whereas the effective aphid resistance in AR-Top Mark and the aphid and suppressive virus resistance in 91213 were apparently overwhelmed during the summer experiment (Fig. 2). The disease progress curves from either planting provide no indication of a delay in the temporal development of the epidemics in the resistant genotypes (91213 and AR-Top Mark) relative to the susceptible genotype (Top Mark), although delays in epidemic onset are
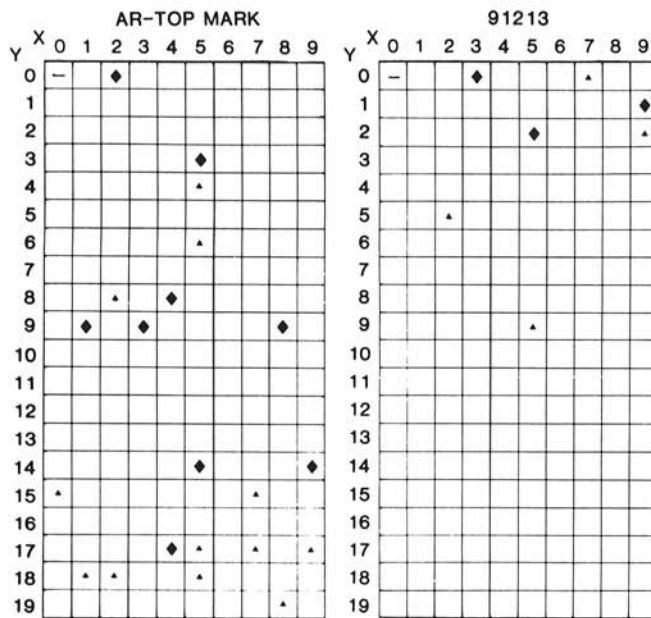
Fig. 4. Typical results of the two dimensional distance class analysis describing the spatial arrangement of WMV 2-infected plants in representative plots of 91213 and AR-Top Mark during the summer. All possible pairs of infected plants within a test plot were categorized into 199 two-dimensional, [X,Y], distance classes. The number of pairs of infected plants in each distance class were statistically analyzed to determine if there was a significant difference in the number of pairs of infected plants observed from that expected if the infected plants within the plot were randomly located. The number of pairs of infected plants in each marked distance class are significantly greater (◆ = P = 0.05, ▲ = P = 0.1) than expected. The disease incidence was 30 and 36% in the AR-Top Mark and 91213 plate, respectively.
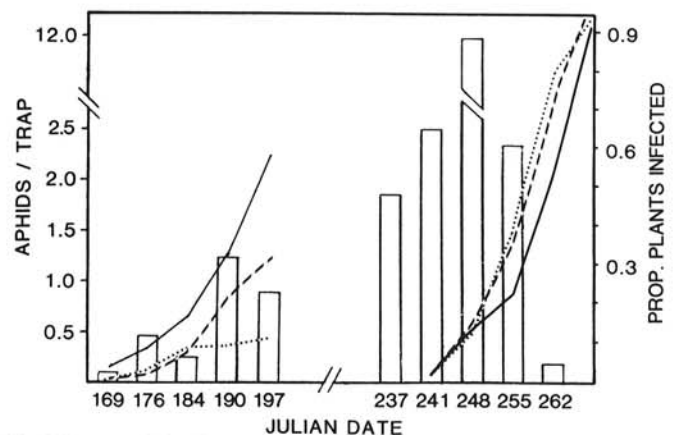
Fig. 5. Seasonal abundance of alate aphids caught in ermine lime green tile traps during the spring (Julian dates 169–197) and summer (Julian dates 227–262) plantings. Mean disease incidence level in the three muskmelon genotypes (Top Mark —) (AR-Top Mark ---) (91213 ......).

characteristic of aphid and virus resistance in other host-virus-vector systems (16).

Variation in epidemics within and among treatments of suppressive virus resistance and partial aphid resistance results from several interrelated factors, including the numbers and relative importance of each aphid species, and the number and proximity of virus sources outside the test area (10,13). *A. gossypii* is abundant during the spring, but a relatively unimportant species of the total aphid population during late summer in North Carolina (5,21). This study supports previous work showing aphid resistance to be effective in reducing the incidence and spread of disease when *A. gossypii* is a major component of the total aphid population (14). The aphid resistance is unlikely to be of practical significance when *A. gossypii* is of limited importance as a virus vector (e.g., 4). There was a significant increase in the number of aphids alighting in the test plots during the second experiment (Fig. 5). A majority (>90%) were not *A. gossypii*, and, therefore, were unaffected by the aphid resistance, which affects only the inoculation efficiency by *A. gossypii*.

Suppressive virus resistance and its effect on acquisition is not aphid species specific (23), therefore its efficiency will depend on the availability and proximity of virus sources outside the resistant fields and the size and shape of the resistant fields. WMV 2 is a limiting factor for squash production during the late summer and fall in the central region of North Carolina; therefore, there would have been a significant increase in the amount of WMV 2 inoculum outside the test area during the summer experiment. Suppressive virus resistance (which affects acquisition efficiency) would be of little epidemiological consequence if a majority of the aphids alighting on the crop become viruliferous on a source located outside the field of resistant plants. The potentially higher number of viruliferous aphids alighting in the summer test plots, relative to the spring, and the small size of the test plots may have masked the potential effectiveness of this resistance that would be observed in large fields.

From the data presented here, partial aphid resistance and suppressive virus resistance decrease rates of disease progress and final disease incidence when secondary spread from infected plants within the field is of major importance. Continued study coupled with breeding programs aimed at increasing the effectiveness of these types of resistance may produce crop cultivars, which will significantly reduce the incidence and spread of nonpersistent aphid-borne virus diseases throughout the growing season and in all circumstances.

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