Suppression of Aphid Colonization by Insecticides: Effect on the Incidence of Potyviruses in Tobacco

T. P. PIRONE, Department of Plant Pathology, University of Kentucky, Lexington 40546; B. RACCAH, Department of Virology, ARO, The Volcani Center, Bet Dagan, Israel; and L. V. MADDEN, Department of Plant Pathology, Ohio Agricultural Research and Development Center, The Ohio State University, Wooster 44691

ABSTRACT

Pirone, T. P., Raccah, B., and Madden, L. V. 1988. Suppression of aphid colonization by insecticides: Effect on the incidence of potyviruses in tobacco. Plant Disease 72:350-353.

The buildup of aphids (primarily *Myzus persicae*) in tobacco was prevented by the use of systemic insecticides in order to determine the relative importance of colonizing aphids in the spread of the nonpersistent tobacco etch and tobacco vein mottling viruses. Three pairs of plots, each containing about 3,300 tobacco plants, were compared; one member of each pair was treated with insecticide and the other served as a control. The experiment was done in three successive years. Insecticide treatment was extremely effective in suppressing aphid colonization and usually effective in reducing virus disease incidence and the absolute rate of disease increase (r'). In one pair of plots, however, r' was significantly increased in 1983, unaltered in 1984, and significantly reduced in 1985. Ecological and seasonal factors that affect the numbers and movement of aphids appear to play a major role in determining the relative importance of colonizing aphids as agents of spread of nonpersistent viruses.

The rapidity with which nonpersistent viruses may be inoculated by their aphid vectors has led to the generally accepted conclusion that insecticidal control of the vectors is not an effective method of preventing virus spread (9). While most studies, including studies on tobacco (11), support this conclusion, there are a few instances in which some reduction in the incidence of virus infection was obtained by insecticide treatment (14,15).

In a 1982 study of the relationship between aphid incidence and the incidence of tobacco etch virus (TEV) and tobacco vein mottling virus (TVMV) in tobacco, we found that none of 1,249 alate aphids collected in a suction trap transmitted virus when placed on test plants (10). Furthermore, the pattern of distribution of virus-infected plants suggested that initial introduction of virus was random but that subsequent spread occurred from initial foci. The time of rapid virus spread was strongly correlated with increased colonization by *Myzus persicae* (Sulzer) (10).

These observations suggested that virus introduction from outside weed sources was sporadic and that the primary infections that occurred in tobacco served as sources for further within-field spread by *M. persicae*. We thus attempted to assess the relative importance of colonizing aphids on virus spread by using insecticides to suppress colonization. To do this, we reasoned that control and insecticide-treated plots should not be adjacent to avoid spread from virus-infected, aphid-infested control plants into the insecticide plots. The plots also needed to be large enough to minimize border effects. The data reported and discussed here deal specifically with the effect of insecticide treatment on virus incidence. Other reports related to this study include temporal analysis of virus increase (6), analysis of spatial patterns of virus increase (7), and correlation between aphid incidence and virus incidence (10).

Salaries and research support provided by state and federal funds appropriated to the University of Kentucky, the Ohio Agricultural Research and Development Center, and The Ohio State University. Additional funding supplied by a grant from the R. J. Reynolds Company to the University of Kentucky.

Kentucky Agricultural Experiment Station Journal Series Paper 87-11-169.

Accepted for publication 25 November 1987 (submitted for electronic processing).

MATERIALS AND METHODS

Six sites were selected on the University of Kentucky's South Farm near Lexington. Three pairs of plots were established; these were paired for their similarity with regard to exposure, slope, and, to the greatest extent possible, surrounding vegetation. The greatest distance between any two plots was about 825 m and the least, about 230 m. Standard procedures used in the cultivation of burley tobacco were followed unless otherwise noted.

Tobacco (*Nicotiana tabacum* L.) cv. Burley 21 was used in 1983 and cv. Kentucky 14 was used in 1984 and 1985; these cultivars reacted identically with regard to virus susceptibility and aphid colonization and as sources of virus for aphid transmission in greenhouse experiments (Pirone, *unpublished*). Each plot of pairs A and B had 22 rows of approximately 150 plants each, while each plot of pair C had 50 rows of approximately 60 plants each. Further details of plot design and data recording are given elsewhere (6,7).

Insecticide treatment. One field of each pair was treated with insecticide to suppress colonization by aphids. The insecticides used were disulfoton (Di-Syston 15G), a systemic insecticide that effectively controls aphids on tobacco (13), and acephate (Orthene 75% EC), which is effective as a foliar spray for control of aphids on tobacco (4). Disulfoton was applied broadcast at the rate of 4.48 kg a.i./ha immediately before transplanting. Acephate was applied as a foliar spray at the rate of 0.84 kg a.i./ha at approximately 2-wk intervals, or more often if there was evidence of initiation of aphid colonies. Dates of application were: 13 and 24 June, 12 and 27 July, and 10 August 1983; 8 and 22 June, 2, 9, 18, 24, and 31 July, and 13 August 1984; and 21 and 28 June, 5, 12, 19, and 30 July, and 9 and 16 August 1985. Control plots were not sprayed. In an attempt to compensate for possible effects of plot location on aphid incidence, the insecticide treatments were applied to the plots on an alternate year basis, i.e., the plots treated in 1983 and 1985 were untreated in 1984, and vice versa.

Aphid monitoring. Aphids were collected from only the main pair of fields (pair A). Horizontal ermine-lime tile traps (2) containing a mixture of ethylene glycol and water were placed in the center and near the corners of each field, for a total of five traps per field. A designated upper leaf of a specific plant, usually one immediately adjacent to each trap, was also used for collecting aphids to compare with the trap catch. Aphids were removed from the tile or plant (usually each day), placed in vials containing 95% ethyl alcohol, and identified later by one of us (BR). Details of the aphid species collected are reported elsewhere (10). Because *M. persicae* was the only aphid found to colonize the control plants in this study, we report here only the comparative data for *M. persicae*, collected from insecticide-treated and control plots.

Virus monitoring. The main pair (A) of fields was monitored for virus-infected plants three times a week and the other fields (B and C), once a week. Each plant in a plot was visually inspected for systemic symptoms, and newly infected plants were marked and the infecting viruses recorded. Systemic symptoms appear approximately 7 days after aphid inoculation (10), and the symptoms caused by TEV and TVMV are distinctive enough to allow visual discrimination between these viruses and also to distinguish them from the other viruses that sometimes occurred in these plots (tobacco streak virus, tobacco ringspot virus, peanut stunt virus) (6). For the purposes of this report, the data for these two nonpersistently transmitted poty-viruses (TEV and TVMV) are considered together.

Data analysis. The logistic model was fitted to the virus disease incidence data of each plot. Details of the nonlinear model fitting procedure and the reasons for using the logistic model have been published (6). The logistic model has three parameters representing initial disease (y_0), maximum disease incidence (K), and the relative rate at which disease incidence approaches K (r). Because disease incidence approached different maxima over the 3 yr, and among plots within a year, epidemics were compared using a derived parameter representing the mean absolute rate of disease increase (r'). This parameter was determined by multiplying the estimates of r and K; it is a scaled version of the mean weighted absolute growth rate of Richards (12). The r' values for the control and treated plot of each pair were compared with a Student's t test, using the standard deviation of the estimated r' parameters.

RESULTS

Insecticide treatment was very effective in suppressing aphid colonization. Plants in the untreated plots were heavily infested with both alate and apterous *M. persicae* in all 3 yr. No, or at most very limited, colonization occurred in the sprayed plots; groups of apterous aphids (evidence for colonization) could be found on only a few scattered plants. The fact that the plots were checked on a daily basis, with insecticide being applied as soon as there was any evidence of colonization, doubtless led to this successful control. However, alate *M. persicae*, as well as alates of other species, were found on plants and in traps in the sprayed plots. The comparative data for alate *M. persicae* collected from the specifically designated tobacco leaves and from the green tile traps in the treated and untreated plots of the main pair of plots (A) are presented in Figure 1. As can be seen from these data, there were generally fewer aphids trapped in the sprayed plots, presumably the result of suppressed colonization. However, in some years, or on certain collection dates, the numbers were similar in the treated and untreated plots, reflecting the incidence of aphids moving into the plot.

The data for virus incidence in the sprayed and unsprayed plots are presented in Figure 2. The incidence of virus-infected plants varied considerably from year to year. In any particular year, however, the overall incidence of virus was similar in the three pairs of plots, with incidence being highest in 1984,

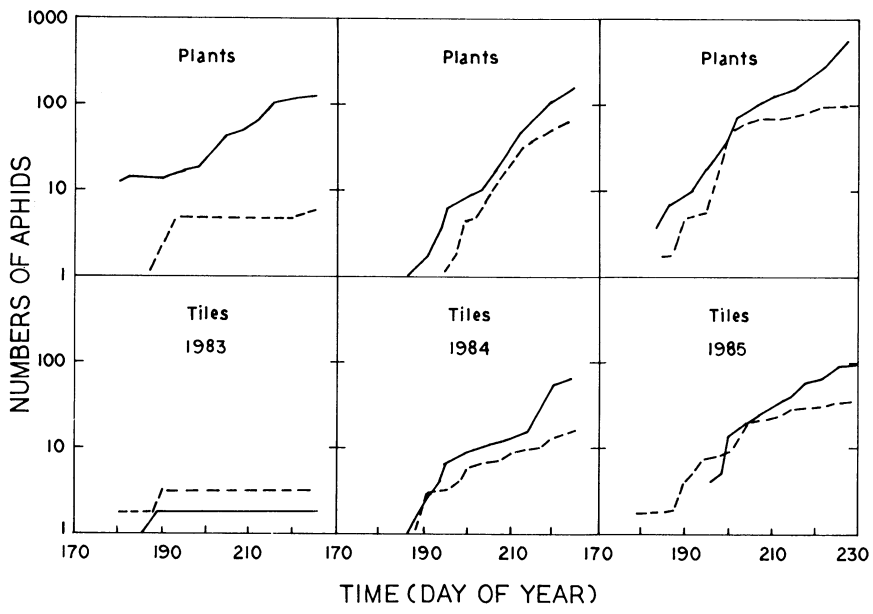

Fig. 1. Total numbers of *Myzus persicae* collected from the five designated plants or from the five green tile traps in field tobacco plots A of Figure 2 that were treated with insecticide (broken line) to suppress aphid colonization or were not treated (solid line).

lowest in 1983, and intermediate in 1985.

In the plot pairs A and C, insecticide treatment was usually effective in reducing virus disease incidence and the absolute rate of disease increase (r'). For plot pair A, r' was significantly reduced in 1983 and 1984 but not in 1985 (Table 1). This rate was significantly reduced in the treated plot of the C pair in all 3 yr (Table 1). In pair B, however, r' was significantly reduced in 1985, unaltered in 1984, and significantly increased in 1983 (Table 1).

DISCUSSION

Previous studies (e.g., 1,3–5,9) have usually shown that insecticides are ineffective in reducing the incidence of nonpersistent viruses in crops. In some instances, however, statistically significant reduction has been reported (14,15). Reduction in virus incidence early in the season, but with no significant reduction at later times, has also been reported (3).

The overall results presented here suggest, not surprisingly, that the spread of nonpersistent viruses within a crop may be reduced or at least delayed by the application of insecticides to control the buildup of potential colonizing species of aphids. Delaying the time of inoculation of tobacco with viruses such as TEV and TVMV by as little as 1–2 wk can result in significant yield increases (8; W. C. Nesmith and Pirone, *unpublished*). Hence, the application of insecticides for virus control could be economically feasible. The data also indicate, however, that the effectiveness of insecticides in reducing the spread of these viruses by controlling colonizing aphids is not predictable. Ecological and seasonal factors that affect the numbers and movement of transient aphids play a major role in determining the relative importance of colonizing aphids as agents of virus spread.

In this study, we chose a rather large plot size to minimize potential border effects and to better represent the typical size of growers' fields. This plot size, however, made it more difficult to compare the treatments statistically, since there were only three blocks (i.e., field pairs), resulting in only 2 df for the error variance in an analysis of variance. Additionally, an interaction between blocks and treatments was common, i.e., disease or its rate of increase was reduced in some blocks but not altered or even increased in others. Such an interaction makes it impossible to test for overall treatment effects because the interaction variance is confounded with the error variance. It is not possible to use subsamples of the plots (e.g., rows or quadrats) to obtain a separate error variance term because the disease values within a plot have high autocorrelation, as described elsewhere (6).

Use of smaller but more numerous plots, although improving the statistical comparisons, would have produced artificial results. All plants in a small plot could become infected in a very short period of time because of the influx of viruliferous aphids from nearby virus sources. We believe, therefore, that reasonably large plots are necessary to understand the dynamics of virus epidemics and the effect of chemical treatments on virus dynamics. Statistical tests can then be made by modeling disease progress and comparing the estimated parameters of the epidemic models.

Table 1. Weighted mean absolute rate (r') of virus disease increase (%/day) in tobacco plots over 3 yr in Kentucky and the Student's t value for comparing the insecticide-treated plots with their paired controls

Year	Plot	r' Control	r' Treated	Student's t value	df[a]
1983	A	1.6	1.0	3.0**[b]	25
	B	1.3	8.6	−3.6*	6
	C	5.3	3.0	4.6**	7
1984	A	16.7	13.6	4.4**	31
	B	16.9	13.5	1.1	12
	C	17.8	15.5	2.9*	11
1985	A	18.4	15.7	1.6	32
	B	13.6	5.9	7.7**	11
	C	23.1	8.0	6.3**	11

[a] Degrees of freedom for Student's t test.
[b] The r' for the control plot is significantly different from r' for the insecticide-treated plot at * = P = 0.05 and ** = P = 0.01.

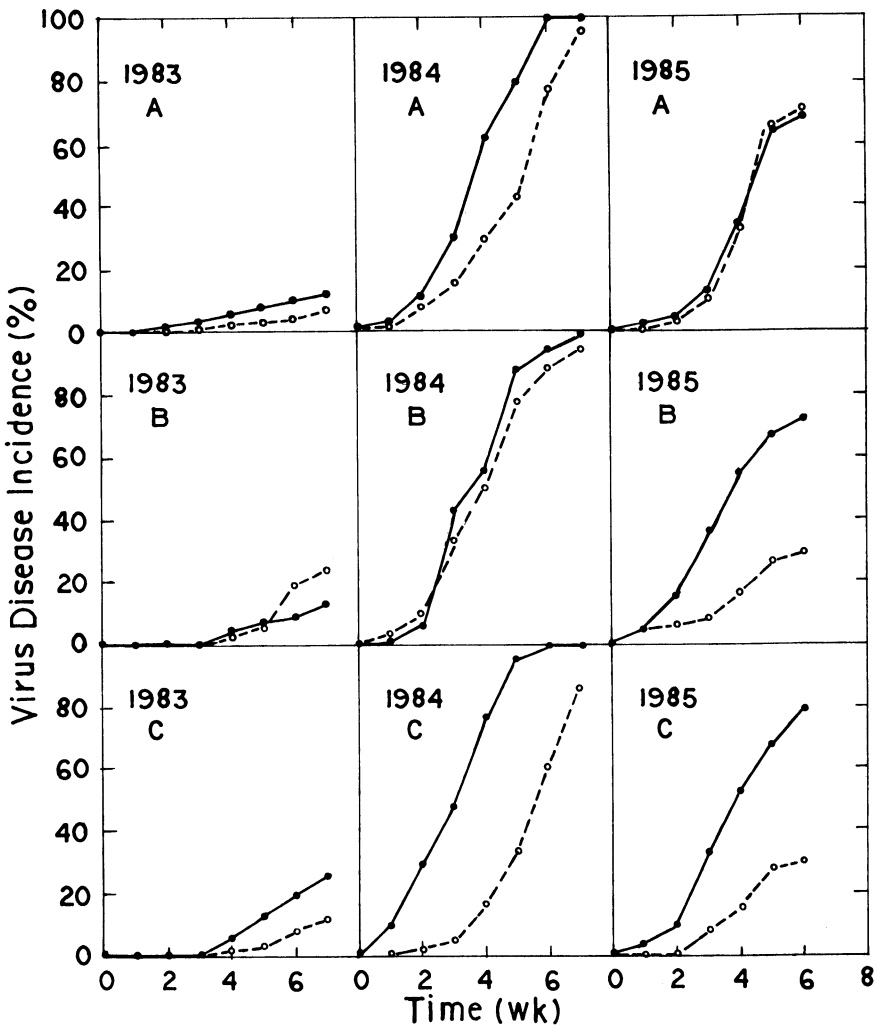

Fig. 2. Incidence of tobacco vein mottling and tobacco etch viruses in field plots of burley tobacco that were treated with insecticide (broken line) to suppress aphid colonization or were not treated (solid line). Week 1 is the first week in July.

ACKNOWLEDGMENTS

We acknowledge the invaluable assistance of Doug McMurray in collecting and entering virus

incidence data. The assistance of George Jones (insecticide application), James Hourigan (data collection), Grayson Brown and John Byars (programs for data entry), John Hoitink (programs for data plotting), and W. C. Nesmith (advice on tobacco production practices) is also gratefully acknowledged.

LITERATURE CITED

1. Ferro, D. N., Mackenzie, J. D., and Margolies, D. C. 1980. Effect of mineral oil and a systemic insecticide on field spread of aphid borne maize dwarf mosaic virus in sweet corn. J. Econ. Entomol. 73:730-735.
2. Irwin, M. E. 1980. Sampling aphids in soybean fields. Pages 239-259 in: Sampling Methods in Soybean Entomology. M. Kogan and D. C. Herzog, eds. Springer-Verlag, New York. 587 pp.
3. Jayasena, K. W., and Randles, J. W. 1985. The effect of insecticides and a plant barrier row on aphid populations and the spread of bean yellow mosaic potyvirus and subterranean clover red leaf luteovirus in *Vicia faba* in South Australia. Ann. Appl. Biol. 107:355-364.
4. Koziol, F. S., and Semtner, P. J. 1984. Extent of resistance to organophosphate insecticides in field populations of the green peach aphid (Homoptera: Aphididae) infesting flue-cured tobacco in Virginia. J. Econ. Entomol. 77:1-3.
5. Kuhn, C. W., Jellum, M. D., and All, J. N. 1975. Effect of carbofuran treatment on corn yield, maize chlorotic dwarf and maize dwarf mosaic virus diseases, and leafhopper populations. Phytopathology 65:1017-1020.
6. Madden, L. V., Pirone, T. P., and Raccah, B. 1987. Temporal analysis of two viruses increasing in the same tobacco fields. Phytopathology 77:974-980.
7. Madden, L. V., Pirone, T. P., and Raccah, B. 1987. Analysis of spatial patterns of virus-diseased tobacco plants. Phytopathology 77:1409-1417.
8. Pirone, T. P. 1974. Effect of tobacco vein mottling virus on yield of burley tobacco cultivars. Tob. Sci. 18:113-114.
9. Raccah, B. 1986. Nonpersistent viruses: Epidemiology and control. Adv. Virus Res. 31:387-429.
10. Raccah, B., Pirone, T. P., and Madden, L. V. 1987. Correlation between the incidence of aphid species and the incidence of two nonpersistent viruses in tobacco. Agric. Ecosyst. Environ. In press.
11. Regan, T. E., Gooding, G. V., Jr., and Kennedy, G. G. 1979. Evaluation of insecticides and oil for suppression of aphid-borne viruses in tobacco. J. Econ. Entomol. 72:538-540.
12. Richards, R. J. 1959. A flexible growth function for empirical use. J. Exp. Bot. 10:290-300.
13. Semtner, P. J. 1979. Insect predators and pests on tobacco following applications of systemic insecticides. Environ. Entomol. 8:1095-1098.
14. Smirnov, Y. V. 1975. Application of insecticides for controlling aphids transmitting soybean mosaic virus. Akademia Nauk SSR. Dal nevost. Naychnyi 28:151-154. (In Russian)
15. Zschiegner, H. J., Kramer, W., Sass, O., Fritsche, R., and Dobnik, H. 1971. Successful restriction of the spread of nonpersistent viruses by methods of virus vector control. Pages 319-323 in: Proc. Br. Insectic. Fungic. Conf. 6th.