Effect of Oil and Insecticide on Epidemics of Potyviruses in Watermelon in Florida

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ABSTRACT

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The effect of JMS stylet oil and/or endosulfan on the incidence and spread of potyviruses in watermelon in the autumn of 1988 and the spring growing seasons of 1989 and 1990 was determined. The incidence of virus-infected plants reached 50% 5–7 days later in oil-treated plots each year, and the maximum rate of virus spread was reduced in 1990. In 1990, the average damage rating of melons from plots not receiving oil was significantly higher than the rating of melons from oil-treated plots. Weights of individual melons from oil-treated plants were significantly lower, however, and at the earliest stages of bloom, 6 wk after planting, there were fewer flowers on oil-treated plants. Endosulfan did not affect virus spread. Oil may be useful for delaying first infections in the spring crop when inoculum sources are limited.

In central and north Florida, the potyvirus watermelon mosaic virus 2 (WMV-2) causes consistent problems in watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai) production (1,14; S. E. Webb, *unpublished*). Early infection leads to reduced yields and blemished fruit (7). WMV-2 is transmitted in a nonpersistent manner by many species of aphids. The source of virus for the spring crop has not been identified, but many wild and cultivated cucurbits and leguminous summer annuals such as hairy indigo (*Indigofera hirsuta* L.), showy crotalaria (*Crotalaria spectabilis* Roth), and oneleaf clover (*Alysicarpus vaginalis* (L.) DC.) serve as hosts in the summer and autumn (1).

In the autumn, two other potyviruses also infect the crop. Symptoms of zucchini yellow mosaic virus (ZYMV) infection are more severe than those caused by WMV-2, and melons from infected plants are misshapen as well as discolored (9,13). ZYMV is found most often in late spring and autumn in central Florida (14). Overwintering hosts for ZYMV in Florida have also not been identified. Papaya ringspot virus type W (PRSV-W) causes severe problems in south Florida in the spring where it overwinters in wild cucurbits (2,3) but generally does not reach central and north Florida until early summer (14; S. E. Webb, *unpublished*).

In a study of spring epidemics of WMV-2, Adlerz (5) found that in only 4 of 11 yr (1967–1977) did the incidence of virus in watermelon breeding fields at the Central Florida Research Center exceed 5% at harvest. Since 1988, how-

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ever, disease incidence has reached 100% each year at the Research Center and growers in surrounding counties have also experienced serious losses (S. E. Webb, *unpublished*). Because resistant cultivars are not yet available, growers have expressed increased interest in other methods of reducing virus spread, including the use of mineral oil.

Since Bradley et al (6) first reported that mineral oil could interfere with aphid transmission of a nonpersistently vectored plant virus, many reports of its use to protect crops have followed (10,11,12,20,22,24,25). The most likely mechanism by which oil reduces transmission is that it interferes with the attachment or release of virus particles from aphid mouthparts (22).

Simons and Zitter (18) concluded that oils were not effective for controlling virus spread in watermelon. The potential of oil sprays to delay primary infections may not have been accurately assessed, however, because a source of virus was introduced into each plot (17). No further details of experimental design were given, nor were data from these experiments reported (17,18). Toba et al (21) showed that oil had potential as a control for aphid-borne viruses in muskmelon.

Primary infections of WMV-2 in watermelon fields are rare events in the spring in Florida (4; S. E. Webb, *unpublished*), with subsequent secondary spread the major component of epidemics. If the use of oil could reduce primary infection, then the onset of epidemics might be delayed. The longer the initiation of an epidemic can be postponed, the shorter the interval between first infection and harvest. This will reduce the yield loss the grower will sustain (5,7).

JMS stylet oil (JMS Flower Farm, Vero Beach, FL) is labeled for virus control in peppers, squash, and tomatoes. However, it has been recently registered for its insecticidal properties against whiteflies, leafminers, and mites on many vegetable crops in Florida, including watermelon, and may be extensively used in the future if whiteflies continue to cause problems. Endosulfan (Thiodan 3EC) is commonly used by watermelon growers as a foliar treatment to control colonizing aphids. However, Simons and Zitter (18) cite a report of increased spread of cucumber mosaic virus in cantaloupe due to the use of endosulfan. To determine if oil and/or insecticide would delay epidemics of virus in watermelon, we evaluated JMS stylet oil and endosulfan alone and in combination.

A preliminary report of this work has been published (23).

MATERIALS AND METHODS

A randomized complete block design, with four blocks, was used. The treatment structure was a factorial with two levels (present or absent) of two factors (oil and insecticide). The experiment was conducted in the autumn of 1988 and repeated in the spring growing seasons of 1989 and 1990 at the research farm of the Central Florida Research and Education Center in Leesburg.

Autumn 1988. On 18 August, the watermelon cultivar Charlee was directly seeded in raised beds. Each of the 16 plots consisted of 10 rows, 36 m long, with plants spaced 2.4 m apart within the rows instead of the 1.2–1.5 m spacing used commercially, to make it easier to keep track of individual plants. Rows were alternately spaced 1.5 and 4.6 m apart so that two rows could be sprayed at one time. Plots were separated by 4.6 m of bare soil on all sides. Beginning on 25 August, 3 days after emergence of seedlings, oil (0.75%) was applied alternately at 4- and 5-day intervals (total of 13 applications) at 2,667 kPa (400 psi), approximately 1,200 L/ha, using a boom sprayer with hollow cone nozzles (TX-5) spaced 20 cm apart. Endosulfan (0.84 kg a.i./ha) was applied seven times at approximately 10-day intervals, beginning 26 August. The fungicide mancozeb (Dithane DF), 1.68 kg a.i./ha, was applied weekly with an airblast sprayer. If the total rainfall for the week was less than 2.5 cm, water was provided by overhead irrigation once a week either the day before or the day after oil was applied. The same agronomic practices were used for all three experiments.

Aphids were collected daily from Monday through Friday from green tile water traps placed at canopy level, designed to sample aphids that would actually be landing in the crop (8). The surface area of each tile (117 cm^2) was approximately equivalent to the upper surface area of two young watermelon leaves. Five traps were operated in one block of each treatment. Aphids were not identified.

Every 3 days the locations of all plants showing symptoms of mosaic were recorded. Two young leaves were collected from every plant showing symptoms for the first time and tested by double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA), using polyclonal antisera (provided by D. E. Purcifull, University of Florida) specific for WMV-2, PRSV-W, and ZYMV. On 3 October, all newly symptomatic plants were tested except for those in the check plots. At this time we were no longer able to keep up with the increasing number of infected plants (samples were not frozen). At the end of the season, 40 additional plants were randomly chosen from each plot and tested by DAS-ELISA. Fruit was harvested on 8 and 17 November from five 15-m sections in each plot, rated, and weighed. Ratings were as follows: 1 = no virus symptoms; 2 = mild virus symptoms, i.e., faint spots and rings; and 3 = severe symptoms, i.e., many green spots and rings, pale background color instead of normal solid light green.

Spring 1989. Watermelon was planted on 6 March and seedlings emerged 2 wk later. Oil and endosulfan were first applied on 22 March when plants were still in the cotyledon stage. Oil was applied at 4- and 5-day intervals until 9 June, a total of 17 applications. Endosulfan was applied seven times, the last application on 31 May. Aphid traps were placed in three of four blocks (three traps per plot). Rather than test each plant developing symptoms, as we attempted to do in 1988, we used a random numbers table and plot maps to determine which 30 plants would be tested by DAS-ELISA. On 4 and 14 June, two young leaves were collected from each of the chosen plants (without regard to symptoms) in each plot and tested. Leaves were frozen for up to 2 mo before analysis. Fruit was harvested on 12 and 21 June.

Spring 1990. Watermelon was planted on 19 March, and oil was first applied on 2 April when plants were in the first-true-leaf stage. Oil was applied 13 times, the last application on 29 May. Endosulfan was applied on 18 April and 2 May. Aphid traps were placed in three of four blocks, using two traps per plot. Thirty randomly chosen plants were sampled on 12 and 28 May and 14 June and tested by DAS-ELISA, as in 1989. Fruit was harvested on 8 and 15 June and rated and weighed as described above. On 3 May, we measured the length of the longest runner and counted the number of blossoms on 20 plants per treatment to detect possible phytotoxic effects of oil.

Data analysis. For all three experiments, a location-shifted logistic model was fitted to the virus disease incidence data from each plot. The model, of the form $p_{ijk} = \gamma_{ij}[1 + a_{ij}\exp\{-r_{ij}(t_{ijk}-b_{ij})\}]^{-1}$ for $t_{ijk} > b_{ij}$ and $\gamma_{ij}(1 + a_{ij})^{-1}$ for $t_{ijk} \leq b_{ij}$, where p_{ijk} = proportion of plants showing virus symptoms for the kth measurement of the jth plot, ith treatment combination, $i = 1,...,4$, $j = 1,...4$, $k = 1,...,n_{ij}$, was fitted by the method of least squares to each of the block-treatment combinations using Proc NLIN (15). T_{ijk} was the day number for the kth measurement of the jth plot, ith treatment, and b_{ij} was the period in which no disease was present. The asymptote (γ) was assumed to be one for all treatments except the check in 1988, for which γ was estimated from the data, and for all treatments in 1989 and 1990. Damage caused by a colonizing aphid, *Aphis gossypii* Glover, to the check plots in 1988 made it difficult to visually identify diseased plants, and one replicate had to be omitted. Maximum rate ($\text{maxrate}_{ij} = (r_{ij}\gamma_{ij})/4$) and the time at which half the population is predicted

to be infected, $t_{1/2,\,ij} = \{[\ln a_{ij} - \ln(2\gamma_{ij} - 1)]/r_{ij}\} + b_{ij}$ were estimated by evaluating each function using the parameter estimates from the model for each block-treatment combination. An analysis of variance (ANOVA) was performed for the derived variables maxrate and $t_{1/2}$.

In 1988 and 1989, marketable yield (rating = 1) for each treatment was compared using ANOVA. In 1990, total yield was analyzed by rating. In addition, treatments were compared by average damage rating and average fruit weight in a weighted split-plot ANOVA, with weights being the number of melons upon which means were based. Harvest date was the subplot treatment.

Because no differences due to treatment could be detected in the total number of aphids caught in any of the three experiments, all captures for a given day were combined and expressed as aphids per trap per day, averaged over 3-day intervals.

RESULTS

Autumn 1988. In the autumn of 1988, symptoms of viral infection first appeared on 12 September, 3.5 wk after planting. Actual infection must then have occurred only 2.5 wk after planting, when most plants had only seven to nine leaves. The progress of disease is plotted in Figure 1A. The number of aphids caught did not exceed one per trap per day until after 9 October (Fig. 2A), but many virus-infected weeds and 20–30 infected watermelon plants held over from a late spring planting by a plant breeder (approximately 100 m from the border of our plots) were available as sources of virus. By 12 October, 55 days after planting, an average of 75% ± 0.11 (SE) of plants in endosulfan-treated plots and 55% ± 0.17 of plants in check plots showed symptoms of viral infection, compared with 24% ± 0.08 of those treated with oil, alone or in combination with endosulfan (Fig. 1A). Based on parameters estimated from disease progress data, the time required for 50% of plants to show symptoms of infection was 7.2 days longer (95% confidence interval = 2.1, 12.2) for oil-treated plots than for plots not treated with oil (61 days after planting vs. 53.8 days) (Table 1). The location-shifted logistic model fit the data well, with r^2 values for plots ranging from 0.94 to 0.99.

Of the 167 plants (36 from oil-treated plots) judged to be infected on the basis of symptoms from 15 September to 3 October, 77% were found, by ELISA, to be infected with WMV-2 only, 8% were infected with PRSV-W only, 5% were infected with both viruses, and 9% were negative. At the end of the season, very few plants were infected with WMV-2 only (Table 2). The percentage of plants infected with PRSV-W only ranged from an average of 3.5% of those in endosulfan-treated plots to 17% of those in

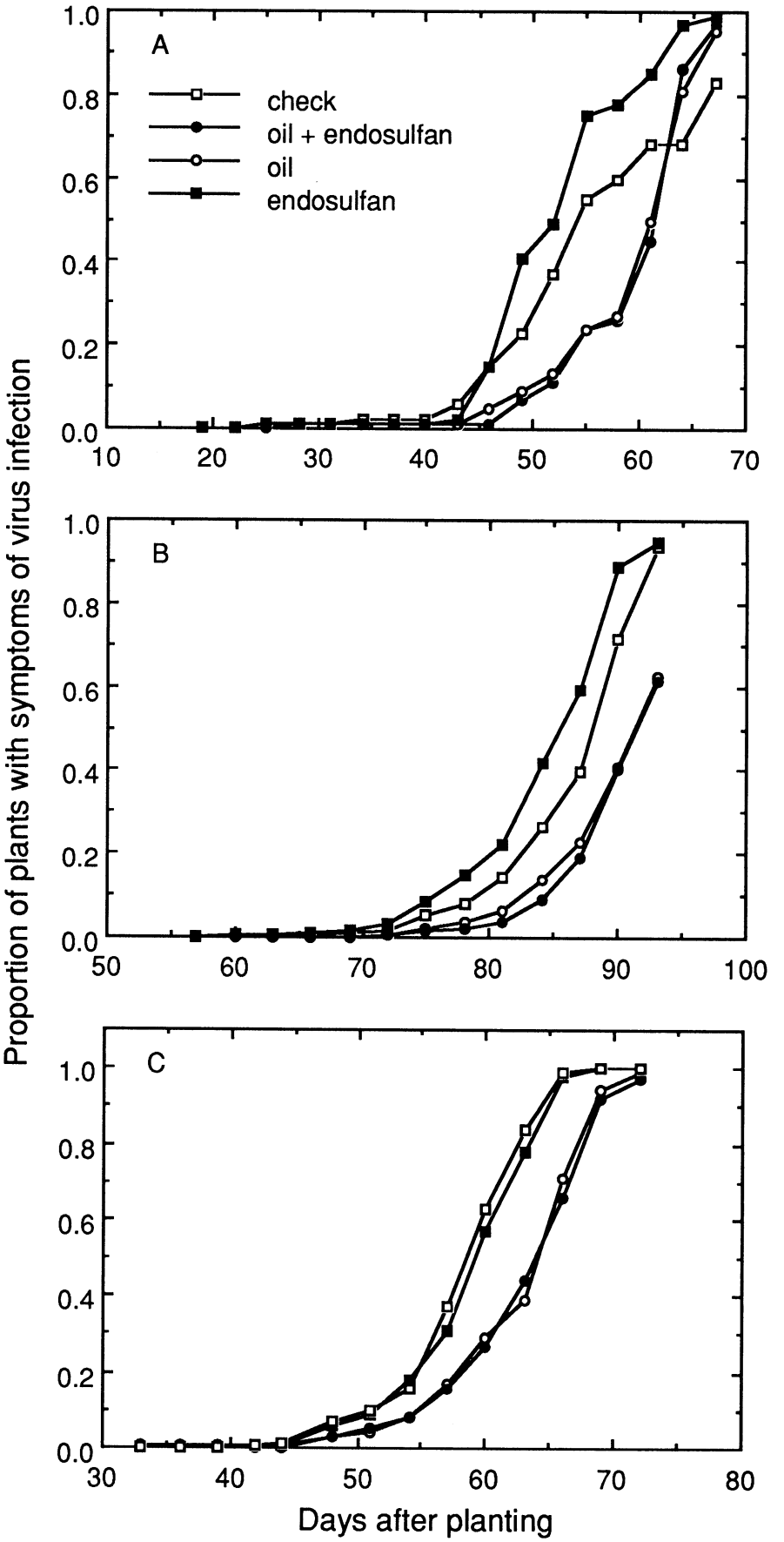

Fig. 1. Progress of potyvirus epidemics in watermelon in **(A)** fall 1988, **(B)** spring 1989, and **(C)** spring 1990. Both watermelon mosaic virus 2 (WMV-2) and papaya ringspot virus type W were common in 1988; WMV-2 was the only virus present until late in the season in 1989 and 1990. Data are means of four replications.

plots treated with oil alone. Only one plant was positive for ZYMV, in a mixed infection with PRSV-W.

Many plants became infected before fruit set in mid-October, and melons were small, pale, and misshapen. Other fruit symptoms consisted of rings and spots on the surface of the rind; no marketable fruit were harvested. The high incidence of PRSV-W, which causes more severe symptoms on fruit than does WMV-2 (13), contributed to the poor quality of melons.

Spring 1989. In the spring of 1989, the first symptoms of viral infection appeared almost 2 mo after planting (Fig. 1B) on 6 May, approximately 1 wk after aphids reached a peak of one per trap per day (Fig. 2B). Virus symptoms appeared first on plants in check plots and plots being treated with endosulfan. On 15 May, one plant with symptoms of infection was found in one plot of each of the two treatments receiving oil. As in 1988, time to 50% infection was significantly longer (Table 1) for oil-treated plots than plots not receiving oil (95.3 days after planting vs. 87.7 days, 95% confidence interval = 2.7, 12.5), and there was a lower incidence of disease at harvest in oil-treated plots (62% ± 0.10, mean ± SE) than in those not receiving oil (95% ± 0.02). The model fit the data very well, with r^2 values above 0.97 for each block-treatment combination.

On 4 June, only one of 477 plants tested by ELISA was found infected with PRSV-W; the remaining 419 positive samples contained WMV-2 only. Of the 480 samples collected on 21 June, nine were negative (seven from oil-treated plots), 471 were positive for WMV-2, and only two were positive for PRSV-W. No ZYMV was detected on either date.

No significant differences in marketable yield or quality were found. Very few melons showed symptoms of viral infection.

Spring 1990. In 1990, we planted on 19 March, 2 wk later than in 1989, and the first plant developing virus symptoms was found on 20 April, 16 days earlier than in 1989 (Fig. 1C). The first symptomatic plants appeared approximately 1 wk after a peak in the number of aphids caught in alighting traps (Fig. 2C). As in the previous experiments, the time at which 50% of the plants in oil-treated plots were infected was significantly later than for plants in plots not receiving oil (66.2 days after planting vs. 61.7) (Table 1). But this time, the maximum rate at which disease spread was also significantly reduced for oil-treated plots (0.082 vs. 0.094, $F = 7.02$, df = 1,9, $P = 0.0265$). As in previous years, the model used to generate parameter estimates fit the data very well (r^2 values > 0.95).

On 12 May, WMV-2 was the only virus detected by ELISA (Table 3). ZYMV was found in all treatments on 28 May, from 3% of the plants in endosulfan-treated plots to 8.5% of the plants in check plots. PRSV-W was not found in plant samples collected from oil-treated plots but was found in an average of 5% of plants from check and endosulfan-treated plots by 14 June.

When we averaged the total weights of melons harvested in each category for each treatment, we found that more of the total weight harvested from oil-treated plots was in category 1 (236 kg vs. 152 kg; $F = 3.86$, df = 1,9, $P = 0.0811$) and significantly more of the total weight harvested from plots not treated with oil was in categories 2 and 3 ($F = 14.54$, df = 1,9, $P = 0.0041$ and $F = 97.20$, df = 1,9, $P = 0.0001$ respectively) (Fig. 3). There was no significant difference

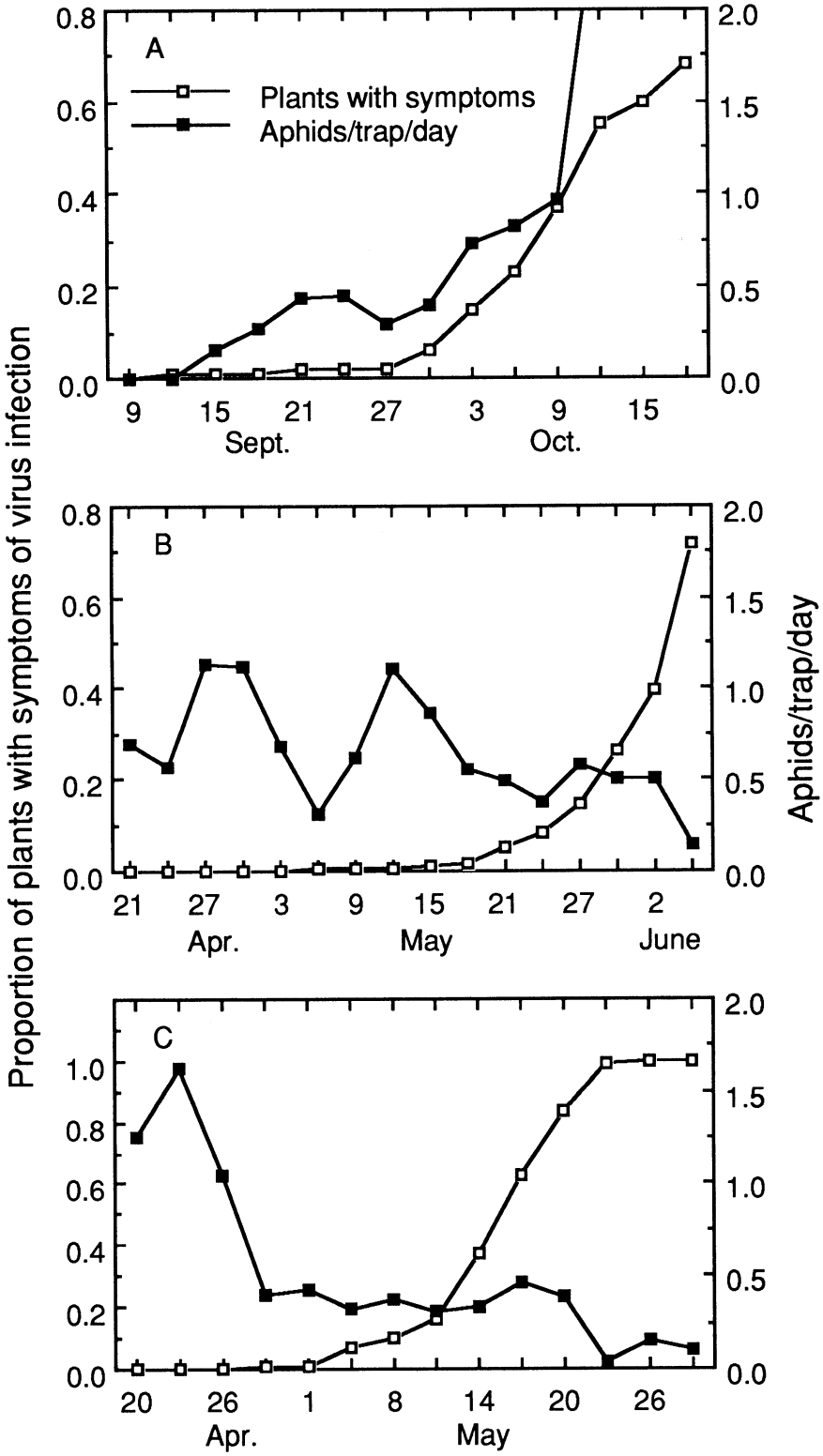

Fig. 2. Number of aphids caught per trap per day, averaged over 3-day intervals, compared with progress of potyvirus epidemics in check plots. **(A)** Fall 1988, **(B)** spring 1989, and **(C)** spring 1990.

in the total number of melons harvested per treatment. On the first harvest date, mean damage rating was decreased by the use of stylet oil with or without endosulfan ($P < 0.03$). The difference due to oil in the absence of endosulfan (2.35 − 1.54 = 0.81) was more than the difference in the presence of endosulfan (2.04 − 1.74 = 0.30) ($P = 0.0094$). On the second date, the use of oil decreased the mean damage rating from 2.27 to 2.06 ($P = 0.0529$). Mean weight of individual melons from oil-treated plots was reduced by 1.14 kg per melon ($P = 0.0003$). Runner length, measured when plants were just beginning to bloom, was not affected by treatments. On 3 May, there were almost twice as many blossoms on plants not treated with oil (1.0 vs. 0.525, $P = 0.0052$).

DISCUSSION

In our tests, oil sprays slowed the spread of virus mainly by delaying primary infection. Only in 1990 was the maximum rate of spread reduced in oil-treated plots. Oil may not be very effective once the incidence of virus in a field or plot reaches 10–20% (17). The rapid growth of watermelon plants and the large leaf surface area of individual plants make it difficult to adequately protect foliage. Because watermelon is planted at a density of only 1,800–2,500 plants per hectare, many fewer transmission events are needed to cause infection of all plants in a field than, for example, would be needed to infect all plants in a field of peppers grown at a density of 44,000 plants per hectare (17).

It is likely that inadequate spacing between plots may have also reduced the effectiveness of oil treatments (25). Most oil-treated plots were constantly exposed to aphids that could have acquired virus in adjacent untreated plots. However, in 1989, in an oil-treated plot that was located on the corner of the experimental planting and was bordered on only one side by an untreated plot, only one of 30 plants tested on 4 June, 1 wk before harvest, was found to be infected. In a 20- to 40-ha field, much longer delays should be possible than in our test plots. Szatmari-Goodman and Nault (20) tested the effects of oil sprays on the spread of maize dwarf mosaic and obtained good results when their field plots were widely separated. When plots were only 5 m apart, virus spread was reduced but not to the extent that it had been when plots were 1 km apart.

The effects of oil on yield depended on the timing of epidemics and season. In the autumn, when inoculum sources around the field were abundant, a 1-wk delay was not enough to keep plants from becoming infected at an early stage of plant growth. In the spring, sources of inoculum appeared to be rare and initial infections occurred later in the season. If, as in 1989, plants are setting fruit when the first infected plants are found, few differences in yield are likely between treated and untreated areas. When epidemics begin earlier in the spring, however, the use of oil may reduce the damage to melons, as it did in our plots in 1990.

Oil appeared to be phytotoxic to watermelon at the rate used in our study. There were fewer flowers on oil-treated plants early in the season, and, overall, melons harvested from oil-treated plants in 1990 were smaller. Further work needs to be done to determine how to minimize the detrimental effects of oil on watermelon. The volume of spray mixture applied could be reduced. Discontinuing the use of oil after fruit set may be advisable; infection by WMV-2 after fruit set is not as damaging as early infection.

Unlike carbofuran in corn (16), endosulfan did not increase virus spread when used at label rates, i.e., no more than three times per season. When used more frequently, endosulfan appeared to increase virus spread, but the difference was not statistically significant. Endosulfan clearly did not kill alate aphids quickly enough to prevent virus transmission.

In the spring, virus appeared after an increase in the number of alate aphids caught in alighting traps. In the autumn, fewer aphids were needed to initiate epidemics, presumably because of greater numbers of infected weeds and other cultivated cucurbits serving as sources of inoculum. Aphids were not identified to species in this study, but in later experiments with green tile traps we found that over 70% of the individuals collected were potential vectors of WMV-2, PRSV-W, and ZYMV (S. E. Webb, *unpublished*).

Stylet oil, if used properly, can significantly delay the onset of viral epidemics in watermelon. The extra cost may be justified only in areas where virus has consistently been a problem early in the spring growing season. Based on the current cost of JMS stylet oil ($2.00/L), the application rate used in this study (8.8 L/ha), and the estimated seasonal costs for labor and machinery to apply it (19), we estimate that it would cost $235–$300/ha for the season to use oil

Table 1. Analysis of variance for days to 50% infection for three epidemics of cucurbit potyviruses in watermelon[a]

		1988		1989		1990	
Source	df	*F*	*P* value	*F*	*P* value	*F*	*P* value
Block	3	0.19	0.8977	1.93	0.1956	5.85	0.0169
Endosulfan	1	0.20	0.6641	0.12	0.7367	0.69	0.4292
Oil	1	10.49	0.0119	12.44	0.0064	98.13	0.0001
Endosulfan + oil	1	0.22	0.6520	0.94	0.3579	0.15	0.7085

[a]To obtain the estimate of days to 50% infection ($t_{1/2}$), a location-shifted logistic model was fitted to the disease incidence data from each plot. Parameter estimates from the model were used to derive the variable $t_{1/2}$.

Table 2. Mean ($n = 4$) percentage of virus-infected watermelon plants and standard error of the mean, by virus, 1988

Treatment[a]	WMV-2[b]	PRSV-W[c]	WMV-2 + PRSV-W	ZYMV[d]	Negative[e]
Check	0.0	14.7 (11.0)	85.3 (11.0)	0.0	0.0
Endosulfan	3.8 (1.9)	3.5 (1.9)	92.7 (3.6)	0.0	0.0
Oil	0.5 (0.5)	17.0 (6.9)	80.0 (9.1)	0.5 (0.5)	2.0 (2.0)
Endosulfan + oil	0.0	13.3 (2.7)	86.7 (2.7)	0	0

[a]Forty randomly chosen plants were sampled from each block in each treatment. Samples from check plots were collected from 27 to 31 October; all others were collected from 8 to 15 November.
[b]Plants infected with WMV-2 only; virus was identified by DAS-ELISA.
[c]Plants infected with PRSV-W only.
[d]In mixed infection with PRSV-W.
[e]Absorbance values were less than the mean of five negative controls plus three standard deviations.

Table 3. Mean ($n = 4$) percentage of virus-infected watermelon plants and standard error of the mean, by virus, 1990[a]

	12 May	28 May			14 June		
Treatment	WMV-2	WMV-2	PRSV-W	ZYMV	WMV-2	PRSV-W	ZYMV
Check	55.3 (13.5)	100.0	0.8 (1.5)	8.5 (7.0)	100.0	5.0 (2.3)	22.5 (11.7)
Endosulfan	52.0 (11.1)	100.0	4.3 (5.1)	3.0 (0.0)	100.0	5.0 (4.4)	19.3 (7.3)
Oil	35.8 (12.6)	93.3 (5.2)	0.0	3.3 (2.9)	94.0 (6.1)	0.0	9.0 (14.1)
Endosulfan + oil	27.0 (4.2)	96.8 (4.7)	0.0	7.5 (5.4)	100.0	0.0	12.5 (9.1)

[a]Thirty randomly chosen plants were sampled from each of four blocks in each treatment. The same plants were sampled again on 28 May and 14 June, including plants infected with more than one virus. Viruses were identified by DAS-ELISA.

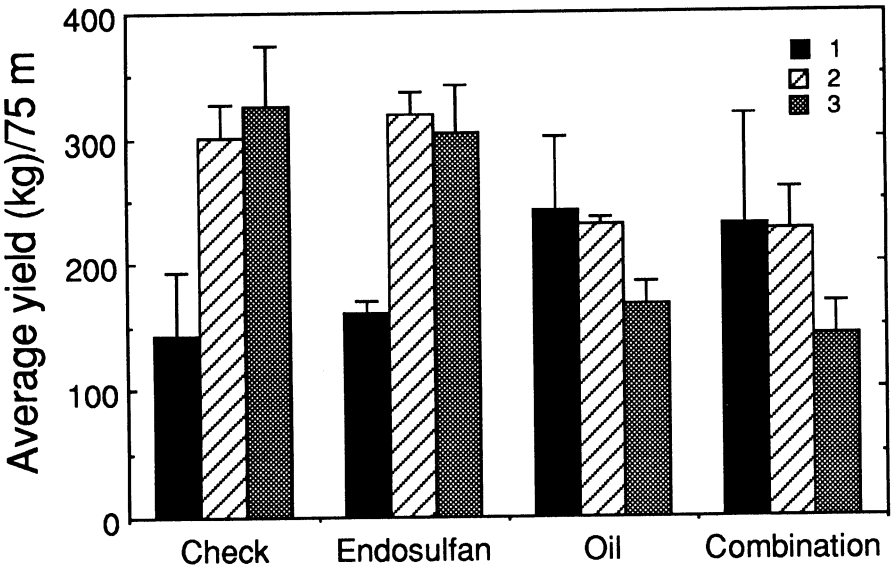

Fig. 3. Average yield of watermelon in 1990, in which 75 m of row was harvested in each of four replications per treatment. Yield was divided into three categories: 1 = marketable, no symptoms of infection; 2 = mild symptoms, i.e., faint spots and rings; and 3 = severe symptoms, i.e., pale rind color, dark spots and rings. Bars indicate standard error of the mean ($n = 4$).

(12–15 applications, ending after fruit set). We have assumed that the cost of half the applications would be the cost of material alone, because oil is commonly mixed with fungicides. During 1991–1992, growing and harvesting watermelon in north Florida cost $3,320/ha (19). Given an average selling price of $0.12/kg, a 5–7% increase in yield would offset increase in cost due to oil.

We are attempting to identify, through surveys, the characteristics of virus-prone areas and are also attempting to correlate the first incidence of disease and peak aphid flights with winter and early spring weather conditions. This would allow a grower to better assess the risks of a disease problem and provide a better basis for making a decision to increase the cost of production by using oil. Oils, used in commercial fields in conjunction with other cultural controls, may be more effective than we have demonstrated if, for example, the watermelon crop is at an early stage of growth at the time viruliferous vectors are present in the spring.

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