PI 96983 was reported to be conditioned by a dominant gene *Rsv* (5). Resistance to all seven SMV strains transferred from PI 360.844 to the soybean line OX670 was shown to be conditioned by a dominant gene Rsv_2 (1). From this study, it was not possible to determine if the genes conditioning SMV resistance in PI 483.084 and PI 486.355 were different from Rsv_2; however, when PI 360.844 plants were inoculated with isolate C14, all 20 inoculated plants developed severe necrotic symptoms similar to the reaction of PI 483.084. Therefore, resistance in PI 483.084 and PI 360.844 was probably conditioned by the same gene, Rsv_2, since reactions of PI 483.084 and PI 360.844 to all the seven SMV strains and isolate C14 were similar. Since the dominant gene in PI 486.355 differs from the gene in PI 483.084, it probably was at a third locus. Further study is needed to compare these genes by evaluating reactions of the F_2 and testcross progeny plants to SMV strains.

LITERATURE CITED

1. Buzzell, R. I., and Tu, J. C. 1984. Inheritance of soybean resistance to soybean mosaic virus. J. Hered. 75:82.
2. Cho, E. E., and Goodman, R. M. 1979. Strains of soybean mosaic virus: Classification based on virulence in resistant soybean cultivars. Phytopathology 69:467-470.
3. Cho, E. E., and Goodman, R. M. 1982. Evaluation of resistance in soybeans to soybean mosaic virus strains. Crop Sci. 22:1133-1136.
4. Hunst, P. L., and Tolin, S. A. 1982. Isolation and comparison of two strains of soybean mosaic virus. Phytopathology 72:710-713.
5. Kiihl, R. A. S., and Hartwig, E. E. 1979. Inheritance of reaction to soybean mosaic virus in soybeans. Crop Sci. 19:372-375.
6. Koshimizu, Y., and Iizuka, N. 1963. Studies on soybean virus diseases in Japan. Tohoku Nat. Agric. Exp. Stn. Bull. 27:1-103.
7. Kwon, S. H., and Oh, J. H. 1980. Resistance to necrotic strain of SMV in soybeans. Crop Sci. 20:403-404.
8. Lim, S. M. 1982. A new source of resistance to SMV in soybeans. (Abstr.) Phytopathology 72:943.
9. Milbrath, G. M., and Soong, M. M. 1976. A local lesion assay for soybean mosaic virus using *Phaseolus vulgaris* L. cv. Top Crop. Phytopathol. Z. 87:255-259.
10. Ross, J. P. 1969. Pathogenic variation among isolates of soybean mosaic virus. Phytopathology 59:829-832.
11. Ross, J. P. 1977. Effect of aphid-transmitted soybean mosaic virus on yields of closely related resistant and susceptible soybean lines. Crop Sci. 17:869-872.

Resistance

Resistance to Watermelon Mosaic Virus II Multiplication in *Cucumis melo*

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ABSTRACT

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The objective of this study was to compare watermelon mosaic virus II (WMV II) multiplication in susceptible genotypes of *Cucumis melo* and in plants of breeding line 91213. WMV II multiplication was measured weekly at three leaf positions by using the ELISA and a local lesion assay to monitor the accumulation of viral antigen and infectious virus, respectively. WMV II multiplication in comparable tissues and following comparable incubation was significantly lower in 91213 than in the other genotypes. There was no evidence for the presence of inhibitors in 91213 that would interfere with either assay. The difference in WMV II multiplication in 91213 and two other genotypes was also observed under field conditions. This form of resistance may have significantly reduced spread in these field trials.

WMV II is responsible for serious losses each year in *Cucumis melo* L. and *Cucurbita pepo* L. Control of this disease has relied heavily on oil sprays, reflective mulches, and avoidance. A source of WMV II resistance similar to the high levels of resistance available for WMV I (15,16) has not been identified for these cucurbit species (10). Other forms of resistance (for example, Lecoq et al [8] describe a form of resistance that suppresses the transmission efficiency of cucumber mosaic virus by an aphid) may be available.

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We detected abnormally low levels of WMV II antigen in infected plants of breeding line 91213 of *C. melo* which were associated with low levels of WMV II acquisition. These studies were initiated to analyze the forms of quantitative resistance to WMV II.

The specific objectives of this research were to compare WMV II multiplication in the susceptible or normal genotypes to that in 91213 and then to determine if this form of resistance would influence the interplant movement of WMV II in the field.

MATERIALS AND METHODS

Host and virus. A single WMV II isolate was used throughout this study. This isolate was obtained from a commercial field of yellow crook-neck summer squash (*C. pepo*) grown in North Carolina. The homogeneity of the isolate was ensured by repeated

(×3) single-local-lesion transfers in *Chenopodium amaranticolor* Coste & Reyn. The isolate was identified as WMV II based on host range and serological tests (11). The isolate was maintained in *Pisum sativum* L. "Alaska" and increased in either *C. pepo* or *C. melo* to supply inoculum for these experiments.

Four lines of *C. melo* were used to develop a map of WMV II multiplication. These are the same genotypes we are currently using to test the effects of host resistance to a virus and its aphid vector on virus spread. Cultivars Top Mark and Hale's Best Jumbo were selected as representative of *C. melo*. Breeding line 91213 has been observed in our studies to generally support lower levels of WMV II multiplication than other lines and cultivars. It has also been the source of antixenosis/antibiosis resistance to *Aphis gossypii* Glover in previous investigations of aphid resistance (6) as well as investigations in this laboratory on the effects of aphid resistance on virus spread (L. Romanow, *unpublished*). Cultivar 91213 is an inbred line originating from LJ 90234. An aphid-resistant Top Mark cultivar (AR-Top Mark), which was developed by A. N. Kishaba and G. W. Bohn in a backcross breeding program using Top Mark as the recurrent parent and LJ 90234 as the source of aphid resistance, was also included in this investigation to determine if there was any relationship between the aphid and virus resistance.

Additional genotypes of *C. melo* were assayed to determine the range of WMV II titers in this species. The lines were: 92119, 92149, 90921, 91462, 91463, 92147, 92131, 92140, 92138, 92141, 92152, 92137, 92153, 92139, 92117, 92151, W-6, AR-5, B63-3, 34340, PMR-45, ANK-1, and aphid-resistant cultivar Hale's Best Jumbo (also developed by Kishaba and Bohn). In addition, WMR-29, W-6, B63-3, and cultivar Songwhan Charmi were also included as genotypes with varying degrees of resistance to WMV I. These lines were provided by A. N. Kishaba, University of California, Riverside; J. D. McCreight, USDA, Brawley, California; M. Pitrat, Station d'Amelioration des Plantes, Montfavet. France; C. E. Thomas, USDA, Charleston, S. C.; and R. E. Webb, USDA, Beltsville, MD. The titers were determined by using the ELISA method as described below. The third and sixth leaves were assayed from two plants of each genotype 3–4 wk following inoculation.

Virus assay. Antiserum for ELISA was produced in New Zealand White rabbits using purified (11) WMV II combined with Freund's complete adjuvant as the immunogen. The homologous titer in microprecipitin tests using a 1:10 extract of WMV II-infected squash was greater than 1:4,000. The titer against noninfected squash was less than 1:8. A slightly modified ELISA protocol (3) for plant viruses was followed. The plates were incubated with 0.4–0.6 μg of serum protein per milliliter of coating buffer for 1 hr at 36 C and washed in 0.15 M phosphate-buffered saline, pH 7.4), (PBS) containing 0.05% Tween-20 PBS-Tween) and 2% ovalbumin. Plant extracts (1:100 dilution, w/v) were prepared in the conjugate buffer (PBS-Tween + 2% polyvinylpyrrolidone 40,000) and incubated in coated plates overnight at 4 C. Alkaline phosphatase-conjugated antibody (0.4–0.6 μg of serum protein per milliliter) was incubated 2 hr at 36 C and the *p*-nitrophenyl phosphate substrate was incubated for 90 min at room temperature. All other procedural details were performed as described (3). The absorbance at 405 nm was measured with a Titertek Multiskan ELISA plate reader (Flow Labs, McLean, VA). Each sample was replicated in four wells and the mean absorbance value was used for statistical analysis. The coefficient of variation within sample replicates was less than 10% for over 80% of the samples.

Local lesion assays were conducted on half-leaves of *C. amaranticolor*. Line 91213 was assayed for WMV II at a 1:50 (w/v) dilution of plant extracts in 50 mM potassium phosphate buffer, pH 7.2; all other lines were assayed at a 1:500 dilution and resulting lesion numbers were adjusted (10×) to reflect this dilution. Each sample was assayed on two randomly selected half-leaves of each of two plants of *C. amaranticolor*. Lesion numbers of the other lines were adjusted to reflect the relative dilution.

Virus invasion. Virus invasion of Top Mark, AR-Top Mark, Hale's Best, and 91213 was monitored with ELISA and local lesion assays for 8 wk following inoculation with WMV II. Plants grown under greenhouse conditions were inoculated at the first or second true leaf stage. The second, fourth, and sixth leaves from the stem apices of two plants of each genotype were assayed 1, 2, 3, 4, and 6 wk after inoculation. The eighth, tenth, and twelfth leaves were assayed during week 8. Half of each leaf was assayed by ELISA and the opposite half by inoculation to *C. amaranticolor*. The experiment was conducted four times between early May and late September. The initial trial was used to establish the appropriate tissue dilutions, for each assay. Data from the final three trials were analyzed by using the general linear model of the Statistical Analysis System (SAS) (12). Local lesion numbers were transformed by the method of Kleckowski (7) for analysis of variance and mean separations. The untransformed data are presented.

The influence of host constituents on the ELISA and local lesion assays was determined by assaying purified virus suspended in extracts from healthy plants of the respective genotypes. Purified WMV II was diluted (10^{-1} to 10^{-4}) with healthy 91213 or Top Mark sap diluted (10^{-2}) in sample buffer and assayed by ELISA. A dilution series ($10^{-0.7}$ to $10^{-4.7}$) of purified WMV II in phosphate-buffered extracts of healthy 91213 and Top Mark were also assayed on *C. amaranticolor*.

The influence of genotype on the infection efficiency of a standard WMV II inoculum was also determined. Four inoculum concentrations (10^{-1} to 10^{-4}) were prepared from WMV II-infected *C. pepo* about 14 days postinoculation. Each concentration was assayed on 10 plants of each genotype. Comparisons were made by calculating the inoculum dilution at which 5 and 50% of the plants of each genotype would become infected. The inoculum dilution and confidence intervals (95%) were calculated by using SAS probit analysis (12).

Field studies. The incidence of WMV II within the genotypes and the virus titer were measured in 91213 when grown in the field. Two plantings were established separated by eight rows of corn. Each planting consisted of an equal number of plots planted to Top Mark, AR-Top Mark, or 91213. Each plot contained four rows on 1.5-m centers with eight plants on 0.6-m centers in each row. Planting A had six replications arranged in a completely randomized design. Planting B was arranged in a randomized complete block with four replications. Two plots of each genotype were in each block. One plot of each genotype in each block was mechanically inoculated with WMV II. Spread of WMV II into the uninoculated plots of both plantings was visually determined when fruit reached maturity. Fifty plants were randomly sampled and serologically assayed for WMV I and WMV II. Only WMV II was detected. The fifth leaf from 10 plants of each genotype was collected from the mechanically inoculated plots and assayed by ELISA 48 and 62 days after inoculation.

RESULTS

There was a significant ($P < 0.01$) difference in virus multiplication between 91213 and other genotypes of *C. melo*. Virus multiplication will be used here when referring to both viral antigen and infectivity (lesion number/weight of infected tissue). The mean level of WMV II antigen and infectious virus over the 6-wk incubation period, was lower ($P<0.01$) in comparable tissues of 91213 than in those of the other three genotypes (Fig. 1). Virus multiplication was not significantly different between Hale's Best, Top Mark, and AR-Top Mark . Hereafter, these three genotypes will be referred to collectively as the susceptible genotypes. Mean ELISA values ranged from 0.32 in the youngest leaves to 0.55 in the oldest leaves of the susceptible genotypes. The mean numbers of local lesions obtained from assay of the susceptible genotypes ranged from 126 in the youngest leaves to 466 in the oldest leaves. The mean levels of virus multiplication in the different-aged leaves of 91213 were not statistically separable (Fig 1). The mean ELISA values from the three 91213 leaves ranged from 0.20 to 0.25 and mean local lesion numbers ranged from 23 to 26.

The temporal patterns of virus multiplication that occurred over the 6-wk incubation period in the susceptible genotypes and in 91213 were analyzed separately due to the difference in the

development of infectious virus between 91213 and the susceptible genotypes (Fig 2). The level of WMV II antigen and infectious virus increased in the susceptible genotypes during the first 3 wk following inoculation. The mean ELISA values increased from 0.23 at 1 wk after inoculation to 0.52 at 3 wk after inoculation. Similarly, the mean local lesion number increased from 136 at 1 wk after inoculation to 434 at 3 wk after inoculation. Both assays indicated a consistent trend of increasing virus content with increasing leaf age during the first 3 wk (Fig 2) with no leaf × week interaction ($P > 0.60$).

The level of virus multiplication at each week during the 6-wk incubation period was less in 91213 than in the susceptible genotypes. The weekly mean ELISA values for 91213 were 50 to 60% less than for the susceptible genotypes except for the third week which was 30% less. Although lower, the temporal change in accumulated viral antigen, on a per-leaf basis, in 91213 was similar to the pattern for the susceptible genotypes (Fig 2). The weekly mean ELISA values increased ($P < 0.01$) from 0.09 at 1 wk after inoculation to 0.37 on the third week. The values decreased to 0.25 and 0.27 by the fourth and sixth weeks, respectively, after inoculation. These ELISA values corresponded to a mean viral antigen concentration in 91213 of about 25% of that found in the susceptible genotypes except for the third week after inoculation which was 50% of the susceptible genotypes.

The local lesion assays indicated that the amount of infectious virus in 91213 was 10–20% of the amount recovered from the susceptible genotypes. The mean local lesion numbers obtained from 91213 at 1, 2, 3, 4, and 6 wk after inoculation were 19, 12, 39, 23, and 49, respectively ($P = 0.06$).

The level of virus multiplication at leaf positions 4 through 12 in 91213 8 wk postinoculation was less than in the susceptible genotypes (Fig. 3). The virus antigen in the leaves of the susceptible genotypes was significantly greater in leaves at positions 4–12 than in leaves at position 2 (Fig. 3a). The amount of infectious virus recovered from the leaves of susceptible genotypes increased with leaf age up to the sixth to eighth position and then remained constant through the 12th position (Fig. 3b).

Virus antigen level in the 91213 leaves was not significantly different (Fig. 3a). The mean levels of infectious virus appeared to vary; however, the differences were not statistically significant nor were consistent trends apparent (Fig. 3b).

The results of additional experiments demonstrated that the host constituents of 91213 did not significantly influence the efficiency of either the ELISA or the local lesion assay. The ELISA values for purified WMV II diluted in either healthy Top Mark or 91213 were numerically lower when diluted in 91213 sap than Top Mark, but the differences were not statistically significant ($P > 0.10$). A highly significant ($P < 0.01$) correlation ($r = 0.90$) did exist between the virus dilution and the log of the ELISA values. Furthermore, there was not a significant ($P > 0.10$) differential effect by either host on infectivity of purified WMV II. Eight twofold dilutions (1:10 to 1:1,280) of sap from WMV II-infected Top Mark resulted in 223 to 29 local lesions per half-leaf. The log of the local lesion number was significantly ($P < 0.005$) correlated ($r = 0.89$) with the host dilution.

The type and intensity of symptom expression were similar for the susceptible genotypes. Symptoms in young leaves of these genotypes consisted of veinal chlorosis and diffuse chlorotic spots. These symptoms evolved into a chlorotic mottle or general chlorosis as the leaves matured. Occasionally, leaves on older plants developed a mosaic symptom. The resistant line, 91213, initially exhibited a distinct mosaic and/or veinal chlorosis with symptoms in succeeding leaves limited to veinal chlorosis and diffuse chlorotic spotting. These symptoms ultimately resulted in a very mild chlorotic mottle in mature leaves.

The titer of WMV II in 91213 was compared to an additional 21 breeding lines and cultivars. Only three lines at leaf position 3 and two lines at leaf position 6 did not have significantly higher levels of WMV II antigen ($P = 0.05$) than found in 91213. When the ELISA values were analyzed by using the log transformation to correct for the greater variance at the higher antigen concentrations, only leaf 6 on 92137 did not contain significantly higher levels of WMV II antigen.

Our experience indicates that 91213 was more difficult to inoculate mechanically and also was less receptive to inoculation by *A. gossypii* than the other genotypes. In an attempt to quantify this relationship, the four genotypes were each inoculated with a dilution series made from a WMV II-infected squash plant. The concentration of WMV II needed to achieve an infection efficiency (percent of inoculated plants which became infected) of 5 or 50% on 91213 was three- to fourfold greater than the concentration needed

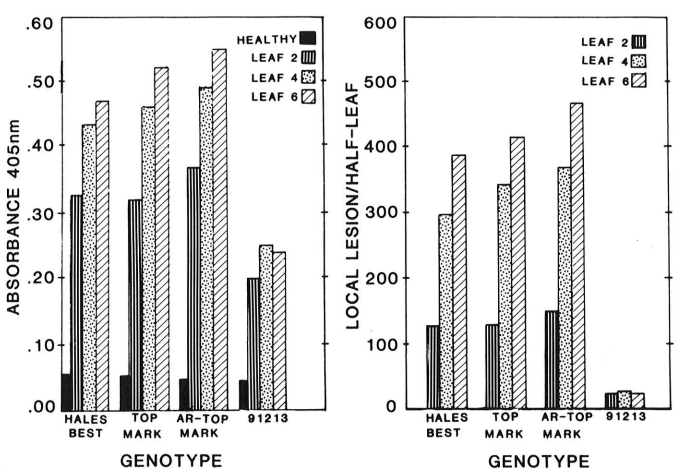

Fig. 1. The relative levels of watermelon mosaic virus II (WMV II) antigen and infectious virus in Hale's Best, Top Mark, aphid resistant-Top Mark, and 91213 genotypes of *Cucumis melo*. Mean separations were not conducted on individual leaf means as there was not a significant leaf × genotype interaction for susceptible genotypes or for 91213.

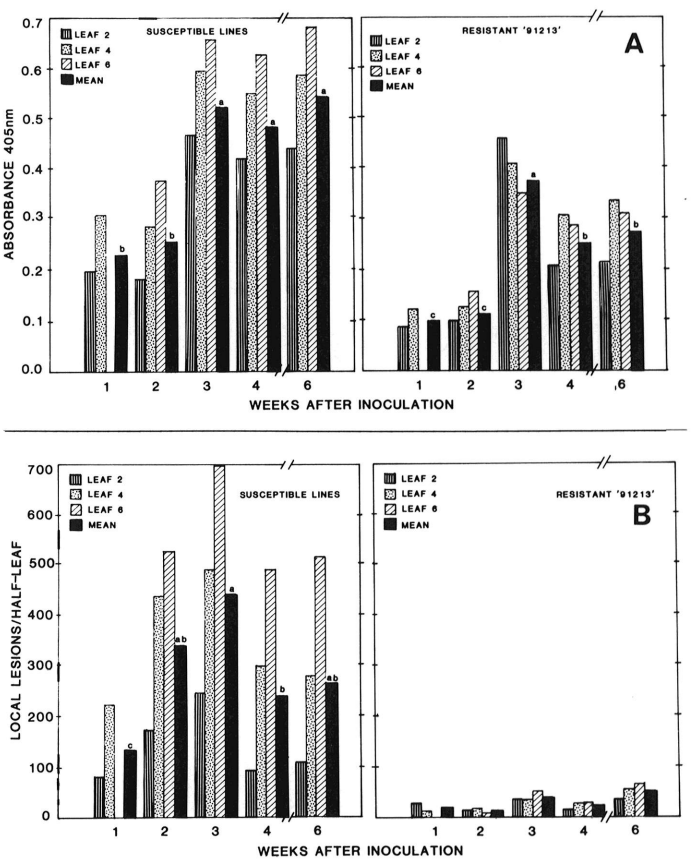

Fig. 2. The temporal pattern of watermelon mosaic virus II (WMV II) accumulation in susceptible genotypes and the resistant 91213 genotype during the first 6 wk of incubation. **A,** The temporal pattern of WMV II antigen accumulation as determined by ELISA. **B,** The temporal pattern of infectious WMV II accumulation as determined by local lesion assay.

TABLE 1. The infection efficiency[a] of a standard watermelon mosaic virus (WMV) II inoculum on WMV II-resistant and -susceptible genotypes of *Cucumis melo*

Genotype	Infection efficiency	Inoculum dilution	Confidence limits 95% Lower	Confidence limits 95% Upper
Top Mark	50	24	7	37
	5	2,092	885	9,164
AR-Top Mark	50	17	6	32
	5	1,871	775	8,971
Hale's Best	50	20	9	37
	5	1,740	751	7,502
91213	50	6	1	14
	5	591	263	2,754

[a]The infection efficiency is expressed as the reciprocal of the WMV II inoculum dilution at which 5 or 50% of the plants become infected based on four replications of 10 plants at each dilution for each genotype.

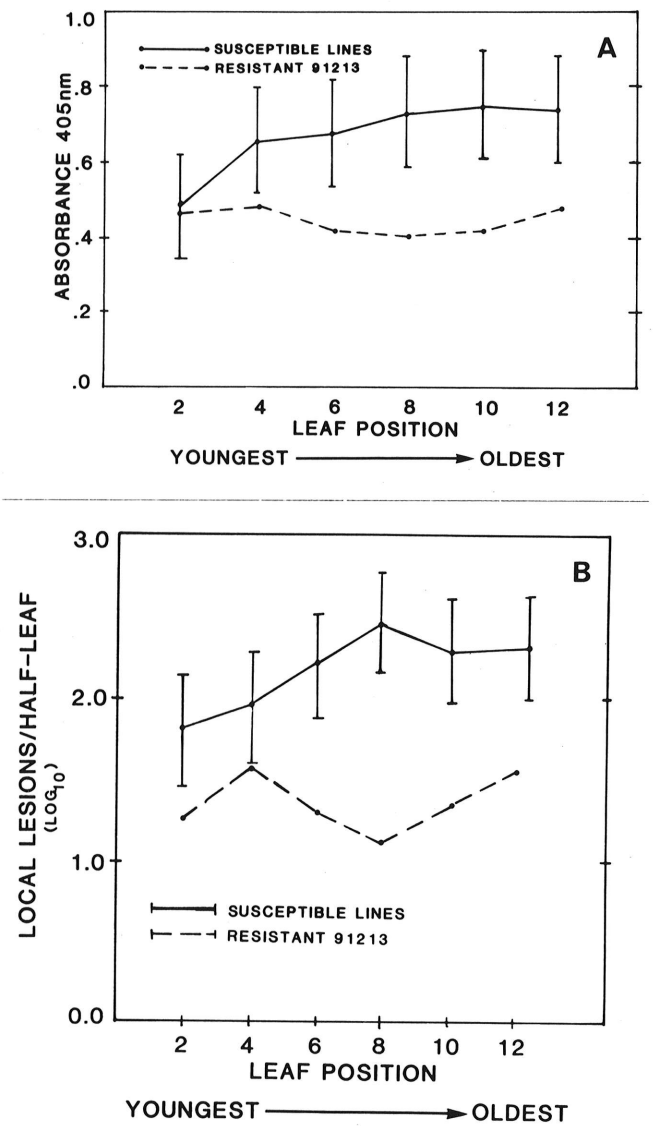

Fig. 3. The relative levels of watermelon mosaic virus II (WMV II) accumulation in the second through twelfth leaves 8 wk after inoculation. **A,** The relative levels of WMV II antigen in the various leaves of the susceptible genotypes and the resistant genotype 91213. **B,** The relative levels of infectious WMV II in the various leaves of the susceptible genotypes and the resistant genotype 91213.

to achieve the same infection efficiency with the susceptible genotypes (Table 1).

The relative accumulation of WMV II antigen in 91213 was also compared under field conditions to the accumulation of antigen in the susceptible genotypes (Table 2). The level of WMV II in 91213 was significantly lower 62 days (at harvest) following inoculation. Although the amount of antigen was numerically lower 48 days after inoculation, the differences were not significant ($P > 0.05$). The low levels in cultivar Top Mark were probably due to the severe heat and water stress that occurred at that time.

Natural spread of WMV II in field plots containing resistant and susceptible genotypes indicated that this type of virus resistance may significantly reduce spread. The incidence of naturally infected (WMV II) 91213 plants in the block containing adjacent plots of mechanically inoculated plants was less than the incidence of natural infection in plots containing susceptible genotypes (Table 3). The incidence of WMV II-infected 91213 plants in a planting separated from the former planting by eight rows of corn was also significantly less ($P < 0.01$) than in the susceptible genotypes.

DISCUSSION

Previous investigations have not detected resistance to WMV II in *C. melo* (eg, 15). The predominant form of virus resistance in 91213 results in suppression of the accumulation of WMV II antigen and infectious virus in leaf tissue. The amount of antigen and infectious virus measured over the 6-wk incubation period was not significantly different among the six youngest leaves of 91213; however, there was a distinct gradient of increasing viral antigen and infectious virus from young to old leaves in the susceptible genotypes (Fig. 1). Further, the temporal pattern of accumulation of viral antigen was similar in the susceptible genotypes and in 91213 (Fig. 2). However, the temporal accumulation of infectious virus in 91213 was distinct from the pattern of viral antigen accumulation in the susceptible genotypes and in 91213. We have designated this *suppressive virus resistance*. Suppressive is defined in Webster's Seventh New Collegiate Dictionary as "to restrain from a usual course of action" or "to inhibit growth or development of."

TABLE 2. Mean ELISA absorbance value (405 nm) of WMV II-infected lines of *Cucumis melo* grown under field conditions

Genotypes	Postinoculation: 48 days Source plants Infected	48 days Source plants Healthy	62 days Source plants Infected	62 days Source plants Healthy
Top Mark	0.25	0.010	0.51	0.006
AR Top Mark	0.29	0.010	0.45	0.008
91213	0.17	0.008	0.09	0.009
$LSD_{0.05}$	NS[a]	NS	0.12	NS
$LSD_{0.01}$	NS	NS	0.16	NS

[a]NS = not significant.

TABLE 3. Watermelon mosiac virus (WMV) II infection incidence in WMV II-susceptible and -resistant lines of *Cucumis melo*

Genotype	Infection at harvest (%): Mixed planting[a]	Healthy planting[b]
Top Mark	83.4	83.0
AR Top Mark	76.5	82.1
91213	55.0	40.1
$LSD_{0.05}$	25.3	12.5
$LSD_{0.01}$	···	16.7

[a]The mixed planting contained five blocks with two plots (32 plants each) of each genotype; one plot in each block of each genotype was mechanically inoculated at planting. The percent is that of naturally infected plants in the remaining plots.

[b]The healthy planting was a completely random design with six plots (28 plants) of each genotype.

A passive barrier to infection (Table 1) distinct from that provided by the aphid resistance (6) (Table 3) was also detected. The aphid resistance is specific to *A. gossypii* and only influences the transmission (inoculation) efficiency of the resisted aphid species (L. Romanow, *unpublished*). The nature of this barrier is unknown, although, available evidence suggests a factor other than an inhibitor in plant sap such as described for potato virus Y in pepper (14).

The type of resistance to WMV II described in 91213 also results in a distinct alteration of disease development from that observed in the susceptible genotypes. The severe mosaic initially expressed by 91213 becomes less severe as the leaves mature, whereas, the mottle on the susceptible genotypes tends to intensify or remain unchanged as the leaf matures. It is not known how this resistance will influence fruit yield.

The suppressive resistance in 91213 also interferes with the spread of WMV II in the field. The incidence of WMV II infection among plants of 91213 at harvest was significantly less than that in either virus-susceptible genotype (Table 3). We have shown that WMV II acquisition by aphids from the suppressive virus-resistant genotype is less efficient than from the virus-susceptible genotypes (L. R. Romanow, *unpublished*). Thus, the abundance of virus-susceptible plants (ie, plants with high virus titer) in neighboring plots supports our hypothesis that the passive resistance to infection may be equally as important as resistance to multiplication in limiting virus spread due to the presence of the high titer sources. This resistance may become a greater factor in natural field situations where within-plot secondary spread may be suppressed due to the low titer sources. This hypothesis is based on the assumption that the plots in these experiments were too small to prevent significant interplot virus movement.

The temporal accumulation of WMV II measured here in the susceptible genotypes is consistent with the limited information available for other potyviruses (1,2,5,9,13,17). The effect of this type of resistance on disease development varied with the host-virus system. Lower virus titer, as measured by various methods, was correlated with delayed symptom expression (1), reduced symptom severity (5,9), reduced spread (2), and a decrease in transmission efficiency (13,17). However, little information is available on the temporal pattern of virus accumulation in the different host systems. Cooper and Jones (4) recently pointed out the gap in our understanding of quantitative host resistance to plant viruses. The type of resistance found in 91213 differs from that of these other systems in that the suppressive effect of the resistance on the accumulation of infectious virus is greater than on the accumulation of WMV II antigen. Further, the disease syndrome expressed by 91213 was distinct from other resistant hosts of potyviruses in that initially a severe mosaic was expressed which was then followed by the milder symptoms. These two factors may have resulted in this apparently useful form of resistance being overlooked.

LITERATURE CITED

1. Anzola, D., Romaine, C. P., Gregory, L. V. and Ayers, J. E. 1982. Disease response of sweet corn hybrids derived from dent corn resistant to maize dwarf mosaic virus. Phytopathology 72:601-604.
2. Bawden, F. C. and Kassanis, B. 1946. Varietal differences in susceptibility to potato virus Y. Ann. Appl. Biol. 33:46-50.
3. Clark, M. F., and Adams, A. N. 1977. Characteristics of the microplate method of enzyme-linked immunosorbent assay for the detection of plant virus. J. Gen. Virol. 34:475-483.
4. Cooper, J. I. and Jones, A. T. 1983. Responses of plants to viruses: Proposals for the use of terms. Phytopathology 73:127-128.
5. Johnson, M. C., and Pirone, T. P. 1982. Evaluation of tobacco introduction 1406 as a source of virus resistance. Phytopathology 72:68-71.
6. Kennedy, G. G., and Kishaba, A. N. 1977. Response of alate melon aphids to resistant and susceptible muskmelon lines. J. Econ. Entomol. 70:407-410.
7. Kleczkowski, A. 1968. Experimental design and statistical methods of assay. pp. 615-730. in Maramonosch, K. and Koprowski, H. eds. Methods in Virology Vol IV. Academic Press, New York 763 pp.
8. Lecoq, H., Cohen, S., Pitrat, M., and Labonne, G. 1979. Resistance to cucumber mosaic virus transmission by aphids in *Cucumis melo*. Phytopathology 69:1223-1225.
9. Marco, S., and Cohen, S. 1979. Rapid detection and titer evaluation of viruses in pepper by enzyme-linked immunosorbent assay. Phytopathology 69:1259-1262.
10. Provvidenti, R., Robinson, R. W. and Munger, H. M. 1978. Resistance in feral species to six viruses infecting Cucurbita. Plant Dis. Rep. 62:326.
11. Purcifull, D. E., and Hiebert, E. 1979. Serological distinction of watermelon mosaic virus isolates. Phytopathology 69:112-116.
12. SAS Institute Inc. 1982. SAS User's Guide: Statistics. 1982 edition. Statistical Analysis Institute Inc., Cary, NC. 584 pp.
13. Simons, J. N. 1958. Titers of three nonpersistent aphid-borne viruses affecting pepper in south Florida. Phytopathology 48:265-268.
14. Simons, J. N., and Moss, L. M. 1963. The mechanism of resistance to potato virus Y infection in *Capsicum annuum* var. Italian El. Phytopathology 53:684-691.
15. Sowell, G., Jr., and Demski, J. W. 1981. Resistance to watermelon mosaic virus in muskmelon. FAO Plant Prot. Bull. 29:71-73.
16. Webb, R. E. 1967. Cantaloupe breeding line B66-5: Highly resistant to watermelon mosaic virus I. HortScience 2:58-59.
17. Zitter, T. A. 1975. Transmission of pepper mottle virus from susceptible and resistant pepper cultivars. Phytopathology 65:110-114.