

INHERITANCE OF RESISTANCE TO WATERMELON MOSAIC VIRUS 2  
IN CUCUMBER (CUCUMIS SATIVUS L.)

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## INTRODUCTION

Cucumber production currently ranks fourth among the vegetable crops grown in Hawaii with sales of \$624,000 (5). Cucumbers are often plagued with disease problems, particularly that of watermelon mosaic virus (WMV). WMV is found throughout the year and serious losses are common. Surveys of yield losses have not been recorded for cucumbers but heavy losses have been reported in summer squash (12), watermelon (13), and cantaloupes (40). Progress has been made in developing WMV 2 resistant cucumber cultivars and breeding lines. The present study will attempt to determine the nature of inheritance of WMV 2 resistance in the cucumber cultivars developed by the University of Hawaii plant breeders. Several foreign cucumber cultivars have shown resistance to WMV 2 and these will also be included in the study. The term resistance in this study will be defined as a high level of tolerance to WMV 2.

Sitterly (52) states that one of the specific goals to be achieved in cucumber plant breeding is the investigation of the relationship between watermelon mosaic virus and the cucumber plant. It is hoped that this study will provide useful information relative to watermelon mosaic virus resistance in cucumbers.

## LITERATURE REVIEW

### I. Host--The Cucumber

Leppik (30) concluded through phytogeographic findings, that Northeast Africa, Arabia, and the Eastern Mediterranean area may be the primary gene center for the genus Cucumis L. Numerous pathogens, insects, and nematodes host specific to Cucumis are found in this area. It is the primary center of distribution of wild cucumbers and many species are found to possess multiple disease resistance. Cucumis species in the primary center possess 12 pairs of chromosomes (30,73). India is considered the secondary gene center of the genus Cucumis (30). The chief evidence that cucumber is indigenous to India is the finding of Cucumis hardwickii Royle, a cucumber-like plant, at the foot of the Himalayas in India (7,73). C. hardwickii is similar to C. sativus L. in many respects, except that the exterior of the fruit is smooth and the flesh is extremely bitter. A number of good breeding stocks was found in this area. C. sativus has 7 pairs of chromosomes and morphological features such as angular stems which distinguish it from other Cucumis species (30,52). Cucumbers have been cultivated in India for at least 3,000 years and were introduced into China and Europe later (7,73).

Sitterly (52) states that genotypic balance is of utmost importance in breeding for cucurbit disease resistance and that breeding for disease resistance should not be the only objective. This balance should result in a plant that is able to survive and produce a desirable product for man. Good genotypic balance would include desired

conformation of the fruit, small seed cavity, crisp flesh, slow developing seed, a waxy surface, and a nonbitter flavor.

## II. Occurrence, Distribution, and Importance of WMV

Walker (60) reported the occurrence of watermelon mosaic virus in Polk County, Florida in 1933, prior to which reports of its natural occurrence are not found in literature. Walker noted the potential seriousness of the disease and warned that a close watch should be made for its recurrence. Since its first discovery watermelon mosaic has been observed to be a serious disease problem in many cucurbit growing regions in the United States and in the world. WMV has been reported in the United States in Arizona (33,41,63), California (20, 23,36,63,71), Florida (1,2,3,4,51), Georgia (12,38), Hawaii (21,49, 56), New Jersey and New York (46,67), Texas (32), and Washington (53). WMV has also been reported in Argentina (74), Australia (22), Bulgaria (37), Czechoslovakia (48), El Salvador (15), Hungary (37), Israel (8), Japan (24,25,26,27), Mexico (42,71), Morocco (17), New Zealand (55), Puerto Rico (43), South Africa (58), and Venezuela (29).

Yield losses have been reported for various cucurbits. Demski and Chalkley (12) reported that WM caused yield losses in summer squash averaging 43, 28, and 9 percent, respectively, for early, midterm, and late inoculations with the virus. Early and mid-term inoculations caused nearly 100 percent losses in marketability and 70 percent losses were obtained for late inoculations. In another study, Demski and Chalkley (13) reported the influence of WMV on watermelon production. WMV infected plants were found to have shorter main and side runners,

and smaller leaves which reduced fresh weight over 55 percent. Fruit number and size were also reduced. Yield losses were found to be greatest when plants were infected at an early stage of growth. Losses varied from 73 percent (early infection) to 19 percent (late infection). Nelson (40) found that early infections of cantaloupes with Cucumber Mosaic or WM reduced plant size and marketable yields. Reductions in fresh weight of 75 and 50 percent for CM and WM, respectively, were obtained when plants were inoculated when the runners were 2 to 4 feet long. Fruit of mosaic infected plants were smaller with a slight to moderate reduction in soluble solid content. Thomas (55) reported yield losses of 63 and 53 percent in "Buttercup" and "Golden Hubbard" squash (Cucurbita maxima L.), respectively, when plants were inoculated at an early stage of growth with WMV. Yield losses were not obtained with late inoculations in winter squash and Thomas concluded that yield reduction was related to the duration of WMV infection in the plant.

Reports of yield losses in cucumber due to WMV infection were not found in literature but losses have been severe on Oahu at the Poamoho and Waimanalo Experimental Farms of the University of Hawaii (J. C. Gilbert, Personal Communication).

### III. Characteristics of WMV

A. Physical. Van Regenmortel (58, 59) investigated the properties of WMV and found the virus particle to be filamentous and rod-shaped. Particle size was determined to be in the range of 725-745 m $\mu$  long and 15 to 20 m $\mu$  wide. Milner and Grogan (35) obtained lengths of

746 m $\mu$  for WMV 1 and 751 m $\mu$  for WMV 2. Purcifull and Edwardson (47) found the normal length of WMV to be 760 m $\mu$ . Other physical properties were obtained for WMV (31,35,58,59). Dilution end-point was found to be  $10^{-3}$  to  $10^{-4}$ . Thermal inactivation point for WMV was between 55° and 65°C. Serological relationships and cross-protection tests were also developed to aid in identifying WMV or WMV strains (35,36,70).

Schmelzer and Milicic (48) found cytoplasmic inclusion bodies in hair cells of cucurbit or cucumber plants infected with European WMV strains. These bodies were amorphous and sometimes consisted of accumulated needle-like structures. Purcifull and Edwardson (47) found particles 2 and 3 times the normal length which were presumed to be virus aggregates representing dimers and trimers, respectively.

B. Strains. Anderson (4) described 2 WMV strains from Florida. The type strain produced milder symptoms than the yellow strain. Webb and Scott (69, 70) concluded from their studies that WMV consists of 2 distinct viruses, WMV 1 and WMV 2, based on differences in cross-protection inoculations, serological relationships and host range. WMV 1 was found infectious only to the members of the Cucurbitaceae and WMV 2 infectious to an extensive host range other than the cucurbit family. Webb (65) found that a cucurbit, Luffa acutangula Roxb., was valuable for separating isolates of WMV 1 from isolates of WMV 2. L. acutangula was susceptible to WMV 1 but immune to WMV 2. A muskmelon breeding line, B 633-3, was found to be a local lesion host for WMV 1 (65,71). WMV 1 induces local lesions in B 633-3, while WMV 2 induces a severe mottle and leaf malformation. Chenopodium amaranticolor Roxb.

was used to identify WMV strains in which isolates of the WMV 2 type would induce local lesions (11,34,35). Milne and Grogan (34,35) concluded on the basis of serological tests and variable host range of WMV isolates, that WMV 1 and WMV 2 are related strains of WMV and not distinct viruses. The physical properties, particle morphology, vector relationships, and symptom production in cucurbits were also found to be similar.

WMV 1 and WMV 2 are widely distributed in the Southern United States and in the Northwest (71). WMV 2 because of its wide host range is often found in greater proportion to WMV 1 in most cucurbit producing areas, although Diaz (15) reported that WMV 1 is widely distributed in El Salvador. Cultivated and wild cucurbits are the virus source for WMV 1 (1,2,15,56). WMV 2's host range includes certain species of the Leguminosae, Malvaceae, Chenopodiaceae, and Euphorbiaceae (22,23,35,36,46,70).

C. Symptoms. Symptoms produced by WMV have been described by various researchers (4,38,59,60,70,73). Typical symptoms include interveinal chlorosis, mottle consisting of green bands along veins or of raised green blisters, stunting, distorted, curled leaves with margins sharply indented, and long narrow shoestring leaf apices. Flowers of infected plants are often deformed and may fail to set fruit. Symptoms on the fruit range from dark water-soaked spots to severe distortion.

Foster and Webb (19) found that symptoms on muskmelon inoculated with WMV and other cucurbit viruses individually decreased in severity

with an increase of temperature. Van Regenmortel et al. (69) also found symptom expression of WMV to be dependent on temperature and light intensity.

Webb and Scott (71) reported that symptom differences between WMV 1 and WMV 2 were not sufficiently distinctive to be a reliable means of virus group identification. Provvidenti and Shroeder (46) reported that WMV 1 produced symptoms more severe than WMV 2, when both strains infected the cucumber cultivar Marketer.

D. Transmission. Freitag (20) found that WMV was a typical aphid-borne virus and was transmitted by the melon and green peach aphids during short feeding periods following a period of fasting. Courdriet (10) reported that the green peach aphid (Myzus persicae (Sulzer)) was the most efficient vector for CMV and WMV transfer to cantaloupes. Five other aphid species were found to be less efficient as vectors. Other aphid species have been found to transmit WMV (22,37,46,74). Toba (57) reported that transmission varied with the age of infection in watermelon plants. Transmission was found to be highest at the first week after inoculation of the virus-host plant and lowest at the fourth week, using the youngest fully extended leaf showing mosaic symptoms selected each week for acquisition feedings. Maximum transmission was obtained after about 15 to 20 seconds of acquisition feeding. Acquisition threshold period, the minimum period of time required for the aphid vector to acquire an infective charge of virus from a virus-source plant, was found to be between 10 and 12 seconds. Inoculation threshold period, the minimum feeding time required by an

aphid to successfully infect the healthy test plant, was found to be at least 9 seconds. Namba and Higa (39) reported that in laboratory studies, the transmission incidence of WMV by Myzus persicae in the afternoon or after 12 hours of light in simulated day-night conditions was in general lower than those in the mornings or after 12 hours of darkness.

#### IV. WMV Resistance Breeding Studies

Gilbert (21) reported what proved to be WMV in CMV resistant cucumber lines. A selection was found to be more resistant than its parental line (a Cornell accession) and showed better tolerance to mosaic than other cucumber varieties. The level of resistance was thought to be conditioned by a number of recessive genes. Illima Hybrid, released in 1959, was found to perform adequately in the presence of WMV. Shanmugasundaram et al. (49), studying cucurbit viruses in Hawaii, observed resistance to single infections of WMV 1, WMV 2, CMV, and to a mixture of CMV and WMV 1 in the cucumber breeding line Hawaii 64A15. When cotyledons of 64A15 were inoculated, very mild symptoms were observed after the true leaves developed, but the plants soon recovered without apparent symptoms. The virus could be recovered from the plants which indicated a high level of tolerance. On the 64A15 seedlings, WMV 2 in combination with either WMV 1 or CMV produced more severe mosaic symptoms than each alone or the CMV and WMV 1 combinations. The selection 64A15 was one of the parents in the commercial WMV resistant hybrid cucumber, Lehua.

Whitaker and Bohn (72) reported that several accessions of Cucumis melo var. conomon (Thunb.) Makino (oriental pickling melon) were

tolerant or resistant to mosaic when exposed to natural infection. Grogan et al. (23) found that Whitaker's material showed some tolerance to WMV in greenhouse inoculation tests but was not tolerant to CMV. Webb and Bohn (68) found several Plant Introduction lines partially tolerant or resistant to some isolates of WMV. Some plants from PI 180280 developed local symptoms but were resistant to systemic invasion. Webb (64) also found that some plants in PI 124112 developed small necrotic lesions without spread to secondary leaves. Foster and Dennis (18) tested cantaloupe breeding lines and found most to be susceptible. Four lines were found to contain symptomless plants and tests of progenies of these stocks showed that the high degree of resistance was conditioned by a heritable character or characters. Webb (66) released a cantaloupe breeding line B66-5, which was highly resistant to WMV 1. B66-5 was a 4th generation inbred from the cross, PI180280 (India) x Seminole, backcrossed twice to Seminole, and then outcrossed to Edisto 47. Bohn (6) incorporated WMV 1 resistance to PMR breeding lines by 4, 5, and 6 successive backcrosses selected for resistance in each generation. Resistance gene frequency was reduced but plant and fruit quality improved when BC4, BC5, and BC6 heterozygous resistant lines were mass selected for 6 generations. High resistance gene frequency was restored by recycling the lines in resistance tests and sibbing.

Sowell and Demski (54) tested 59 watermelon cultivars and found all to be susceptible to WMV 2. Demski and Sowell (14) screened the United States Department of Agriculture's entire collection of plant introductions of Cucurbita pepo L. and Citrullus lanatus L. to find

immunity or hypersensitivity to WMV 2. Thirty to 100 percent of the plants in each introduction in both genera were found susceptible to WMV 2. Although all PI's were susceptible, it was concluded that other types of resistance were possible, such as resistance to virus multiplication expressed as a delay of symptom expression.

## V. Resistance Studies of Other Cucurbit Viruses

A. CMV. Elmer (30) and Porter (44) were the first workers to study the reaction of cucumbers to mosaic and used the resistant variety Chinese Long, which Porter had discovered in China. Elmer found the variety to be highly resistant to CMV. Porter (45) found the Chinese Long variety to be highly resistant to CMV 1 and susceptible to CMV 2. Resistance to CMV was suggested to be due to one or a few recessive genes. Shifriss et al. (50) concluded from their studies that CMV resistance was due to 3 basic dominant genes in the presence of a maximum number of dominant modifiers. Wasuwat and Walker (61) using resistant Wisconsin SMR-14, determined that CMV resistance was due to a single dominant gene. It was demonstrated in greenhouse tests that differences between resistant and susceptible plants were best determined 20 days after inoculation. All resistant plants showed symptoms but the symptoms were mild and tended to disappear. In susceptible plants, mosaic symptoms were systemic and fruits were mottled. It was concluded in another study that the resistance mechanism was one which restricts virus multiplication sufficiently to minimize the detrimental effect on the growth and productivity of the host (62). Kooistra (28) concluded from resistance

studies using Hokus (which derived its resistance from Tokyo Long Green) and Natsufushinari as the resistant parents, that a high degree of resistance to CMV 1 in cucumbers is characterized by intermediate inheritance and seemed to be based on 3 genes, each carrying partial resistance.

B. Melon Mosaic Virus. Cohen et al. (9) used Kyoto 3 Feet as a source of resistance to melon mosaic virus (MMV) in cucumbers. Resistance to MMV was classified according to external symptoms and was found to be governed by a single dominant gene.

## MATERIALS AND METHODS

All new cucumber breeding lines developed in Hawaii are screened for mosaic virus resistance. The predominant virus affecting cucurbits in Hawaii is watermelon mosaic virus, particularly the WMV 2 strain. WMV 2 is the more prevalent strain in many parts of the world. The original source of WMV 2 used in this study was obtained from an infected cucumber plant on Oahu. Identification of the strain was determined through a host range test which included the following plants: squash (Summer Straight Neck), watermelon (Charleston Gray), cantaloupe (B66-3), tobacco (Nicotiana glutinosa L.), Luffa acutangula Roxb., and Chenopodium amaranticolor L. Cantaloupe B66-3 shows local lesions for WMV 1 and systemic infection for WMV 2. L. acutangula is a specific systemic host for WMV 1 but shows no reaction with WMV 2. C. amaranticolor shows local lesion symptoms for WMV 2 and no symptom reaction with WMV 1. Electron microscope studies have also confirmed the virus used in the cucumber breeding program in Hawaii to be WMV. After determining the virus strain, the isolate was maintained in the greenhouse on Hawaii breeding line 69B 12. The cucumber line 69B 12 was able to grow adequately in the greenhouse in presence of the virus.

A series of inoculations was made to determine possible susceptible and resistant parents for foundation crosses. Susceptible parents were selected from older Mainland cucumber cultivars which have little or no disease resistance. Two cultivars, Marketmore and Tablegreen, are resistant to cucumber mosaic virus (CMV) but susceptible to WMV. These were included in the group of susceptible parents to determine what

effect CMV resistance has on WMV resistance. Resistant parents included cucumber breeding lines developed at the University of Hawaii, which are true breeding for resistance to WMV. Several foreign cultivars were also examined for resistance to WMV and two of these introductions were included in the foundation crosses. The foreign cultivars were Sooyow (Mikado Strain) and Sooyow (Takii Strain), which will be referred to as SM and ST, respectively. These cultivars were reported to be Chinese types but improved in Japan. The fruits of both Sooyow types are long (12-18"), 1 1/2 - 2" in diameter, ribbed and highly spined. The two types differ from each other in that SM has more spines, larger seed, larger seed cavity, and a darker green foliage. Fruits of the Sooyow types have the highly desired trait of having very crisp flesh. The Sooyow varieties were involved in the cucumber breeding program to incorporate the crisp character in Hawaii's cucumber lines. Biji Tunin, a cucumber introduction from Indonesia, was found to possess some resistance to WMV. It was involved in a few basic crosses as the cultivar had many characters which were undesirable such as black spines and orange fruit, soft flesh, large seeds, and large seed cavity.

The foundation crosses involved the following cultivars:

<u>Susceptible</u>	<u>Resistant</u>
A & C or Colorado	Hawaii 67A9
Ashley	Hawaii 67A13
Marketer	SM (Sooyow, Mikado Strain)
Marketmore	ST (Sooyow, Takii Strain)
Straight Eight	
Tablegreen	

F<sub>1</sub>'s, F<sub>2</sub>'s, and backcrosses were made between the 6 susceptible and 4 resistant cultivars. Also, F<sub>1</sub>'s, F<sub>2</sub>'s, and backcrosses were made between 67A9, 67A13, and the Sooyow strains.

Field plantings of the segregating progeny were used as the plants required at least 1 month of growth for best symptom expression. Losses due to disease, i.e. damping-off, and insects were also less in the field than in the greenhouse plantings. Field plantings were necessary because of the large number of plants involved.

The seedlings were inoculated by rubbing the virus inoculum on the cotyledons. The inoculum consisted of 0.1M sodium phosphate buffer, carborundum for an abrasive, and infected plant tissue ground up with a mortar and pestle. A second inoculation was made 1 week later to eliminate escapes or to inoculate late emerging seedlings. Another inoculation was made on those plants which failed to show virus symptoms.

A system of classification based on external symptoms was used in classifying individuals in the segregating population.

Class 1. Symptomless. No WMV resistant lines observed so far fall in this category.

Class 2. Symptoms are present only near the point of inoculation. Fruits show no symptoms. The resistant cultivars fall in this category.

Class 3. Mosaic symptoms are present only at the leaf margin and leaf tip. Plant growth and fruit shape are normal but fruit may show some mottling or water-soaked spots.

Class 4. Leaves show moderate chlorosis and mottling, plants show moderate stunting. Fruits are produced but are mottled and deformed.

Class 5. Severe chlorosis, mottling, and distortion of leaves and severe stunting of the plants occur in this group. If fruits do develop, they are severely deformed. The susceptible parents fall in this classification.

## DISCUSSION OF RESULTS

After the initial screening of cucumber cultivars for susceptibility or resistance to WMV 2, a series of 24 foundation crosses between the 2 groups was initiated in 1971. Resistant parents included HAES 67A9 and HAES 67A13, which are sister lines that were found to be true breeding for WMV 2 resistance. Two cucumber introductions from Japan, Sooyow Mikado Strain (SM) and Sooyow Takii Strain (ST), were also included as resistant parents. These were not reported to possess any WMV resistance but were found to possess high resistance to the virus in the initial screening program. Susceptible parents included 6 Mainland cultivars with little or no resistance to any specific disease. Marketmore and Tablegreen, however, do possess resistance to cucumber mosaic virus. A & C or Colorado, Ashley, Marketer, and Straight Eight were also included in the susceptible classification. Foundation crosses were also made between the 2 HAES breeding lines and the 2 Sooyow cultivars to determine if the resistance between the 2 types were similar. From the foundation crosses, a series of  $F_2$ 's, backcrosses to the susceptible parents, and backcrosses to the resistant parents were made. Adequate seed stock from all the crosses was obtained by the summer of 1973 to permit field plantings. The plantings of the segregating populations were made at the Poamoho Experimental Farm from June, 1973 to August, 1974. It was decided that field plantings were necessary as large populations were involved. Field plantings also allowed easier control of diseases and insects and permitted at least one month's growth for best indexing of the

virus symptoms on the plants. Several problems did arise when some plantings were lost to heavy rains and flooding. An unexpected problem with birds eating the newly emerged seedlings was eliminated by placing wire screens over the hills soon after planting. Losses due to cutworms and rodents did occur various times throughout the year.

Since a large number of crosses were involved, the plantings were divided into different groups:  $F_1$ 's,  $F_2$ 's, backcrosses to the susceptible parents, and backcrosses to the resistant parents. Each group consisted of 4 replications in a randomized complete block design. In order to facilitate handling, such as inoculations and symptom indexing, each group was divided into plantings of 2 replications with seeding dates 2 weeks apart. The susceptible and resistant parents were included in each planting as checks.

Inoculations were made at the cotyledon stage of growth or about 1 week after planting. A second inoculation was done 1 week later to prevent any escapes and to inoculate late emerging seedlings. Cucumber plants were found to be susceptible to WMV 2 at any stage of growth, so plants which appeared to be escapes were re-inoculated before symptom readings were made. The resistant parents showed WMV symptoms on the first few true leaves following inoculation of the cotyledons, but appeared to grow out of the initial symptoms. Plants with high resistance were able to resume normal growth and produce fruits that were not deformed by the virus. Resistant plants appeared to prevent the movement of the virus outward as new side shoots arising from axillary buds near the base of the plant showed some mosaic symptoms

on the first leaf or two. Susceptible plants as well as plants with moderate resistance continued to show virus symptoms on all new growth of the plant. All susceptible parents showed very severe mosaic symptoms when inoculated with WMV 2 with the exception of Tablegreen, which produced symptoms that were intermediate. The other 5 parents produced symptoms of severe chlorosis, veinclearing, leaf distortion, stunting, and often no fruit production. Fruits that were produced were severely deformed. Tablegreen was classified in the Class 4 system of symptom indexing and A & C, Ashley, Marketer, Marketmore, and Straight Eight were placed in Class 5 or extreme susceptibility (Table 1).

No differences were observed between the resistant parents, as all outgrew the initial symptoms and produced fruits free of virus defects. The Sooyow lines appeared to be as resistant as the HAES lines and all plants were classified in Class 2. No plants were found to be free enough of or immune to WMV 2 to be placed in Class 1.

F<sub>1</sub>'s between the susceptible and resistant parents exhibited symptoms very similar to that of the susceptible parents, indicating that the resistance to WMV 2 was largely recessive in nature. The F<sub>1</sub>'s with the exception of those crosses with Tablegreen were classified in Class 4, while F<sub>1</sub> crosses with Tablegreen were placed in Class 3 (Table 2). Although chlorosis, veinclearing, and distortion were severe, there was no severe stunting of the plants as would be found in Class 5. This difference may be due to the hybrid vigor of the plant as is sometimes found in F<sub>1</sub> combinations. F<sub>1</sub> crosses with Tablegreen produced plants which showed more resistance than the

Table 1. Classification of the parental lines based on symptoms produced by WMV 2 infection

Parent	C l a s s				
	1	2	3	4	5
A & C					223
Ashley					250
Marketer					281
Marketmore					251
Straight Eight					232
Tablegreen				273	
67A9		256			
67A13		231			
SM		215			
ST		247			

Table 2. Classification of plants from the F<sub>1</sub> crosses based on symptoms produced by WMV 2 infection

F <sub>1</sub> Cross	C l a s s				
	1	2	3	4	5
A & C x 67A9				72	
x 67A13				84	
x SM				73	
x ST				54	
Ashley x 67A9				77	
x 67A13				81	
x SM				76	
x ST				80	
Marketer x 67A9				77	
x 67A13				82	
x SM				66	
x ST				69	
Marketmore x 67A9				68	
x 67A13				76	
x SM				61	
x ST				73	
Straight Eight x 67A9				74	
x 67A13				79	
x SM				70	
x ST				61	
Tablegreen x 67A9			70		
x 67A13			79		
x SM			69		
x ST			77		
67A9 x SM		74			
67A9 x ST		63			
67A13 x SM		69			
67A13 x ST		60			

crosses involving the more susceptible parents. Growth was nearly normal with symptoms only occurring near the leaf margins and leaf tips, indicating that possibly Tablegreen does possess some resistance to WMV 2. No differences in resistance were noted between the resistant parents in any of the 24  $F_1$  combinations with the susceptible parents. No segregation occurred in the crosses between the 2 HAES lines and the 2 Sooyow lines. All progeny were classified in the Class 2 type of resistance.

A series of plantings were made of the  $F_2$  population of the 28 foundation crosses to study the segregation pattern of the progeny. It was indicated from the  $F_1$  populations that resistance was recessive in character. Observations of the  $F_2$  population confirmed this view and it appeared that more than 1 recessive gene was involved. Chi-square values were determined for each  $F_2$  population by grouping Classes 3, 4, and 5 as susceptible and Classes 1 and 2 as resistant (Tables 3 and 4). A 2 gene concept of 15 susceptible to 1 resistant was found to give the closest fit of any genetic ratio. It was found that chi-square values of most populations did coincide with the 2 recessive gene concept with probability values being greater than 0.05. Several populations did come close to exceeding 0.05, particularly those of Marketer x ST and Marketmore x 67A13. Because of high chi-square values of some of the populations it may be assumed that possibly errors in classification were made as more resistant plants appeared than was calculated. Environmental conditions or poor growth due to nematodes, insects, and diseases, or depletion of fertilizers, may have resulted in classifying several Class 3 plants as Class 2 or resistant.

Table 3. Classification of the F2 progeny based on symptoms produced by WMV 2 infection \

Cross	Symptom Range				
	1	2	3	4	5
<u>67A9</u>					
A & C x 67A9		11	22	16	118
Ashley x 67A9		17	28	15	112
Marketer x 67A9		15	39	34	126
Marketmore x 67A9		13	19	15	104
Straight Eight x 67A9		15	26	30	117
<u>67A13</u>					
A & C x 67A13		8	20	29	120
Ashley x 67A13		11	23	15	99
Marketer x 67A13		17	31	40	119
Marketmore x 67A13		16	24	16	108
Straight Eight x 67A13		15	16	21	105
<u>SM</u>					
A & C x SM		12	27	26	114
Ashley x SM		18	27	38	120
Marketer x SM		15	30	27	115
Marketmore x SM		14	28	24	119
Straight Eight x SM		16	21	29	114
<u>ST</u>					
A & C x ST		13	33	24	104
Ashley x ST		15	24	17	113
Marketer x ST		18	21	27	123
Marketmore x ST		17	22	28	115
Straight Eight x ST		11	25	30	104
<u>Tablegreen</u>					
Tablegreen x 67A9		43	70	77	
Tablegreen x 67A13		49	60	78	
Tablegreen x SM		42	71	64	
Tablegreen x ST		49	68	75	

Table 4. Summary of the segregation distribution and chi-square values for the F<sub>2</sub> progeny exposed to WMV 2 infection

Cross	Susceptible	Resistant	Chi-square	P
<u>67A9</u>				
A & C x 67A9	156	11	0.0345	.90
Ashley x 67A9	155	17	2.327	.20
Marketer x 67A9	198	15	0.2373	.70
Marketmore x 67A9	141	13	1.208	.30
Straight Eight x 67A9	173	15	0.8228	.50
Composite	823	71	4.2940	<.05
<u>67A13</u>				
A & C x 67A13	169	8	0.980	.50
Ashley x 67A13	137	11	0.2746	.70
Marketer x 67A13	190	17	1.3227	.30
Marketmore x 67A13	148	16	3.2337	.05
Straight Eight x 67A13	159	15	1.6453	.20
Composite	803	67	3.109	.05
<u>SM</u>				
A & C x SM	167	12	0.8393	.50
Ashley x SM	177	18	2.6543	.10
Marketer x SM	172	15	0.7551	.50
Marketmore x SM	166	14	0.7361	.50
Straight Eight x SM	164	16	1.9707	.20
Composite	846	75	5.3168	<.05
<u>ST</u>				
A & C x ST	161	13	0.4046	.70
Ashley x ST	154	15	1.8502	.20
Marketer x ST	171	18	3.4745	.05
Marketmore x ST	165	17	2.9446	.05
Straight Eight x ST	159	11	0.0370	.80
Composite	810	74	5.8828	<.05
<u>Tablegreen</u>				
Tablegreen x 67A9	147	43	0.724	.50
Tablegreen x 67A13	138	49	0.1411	.70
Tablegreen x SM	135	42	0.1558	.70
Tablegreen x ST	143	49	0.0277	.80
<u>Between Hawaii and Sooyow lines</u>				
67A9 x SM		116		
67A9 x ST		128		
67A13 x SM		150		
67A13 x ST		159		

Best symptom readings were obtained when plants were growing unimpeded by other factors. The  $F_2$  progeny, with the exception of the  $F_2$ 's with Tablegreen, were grouped under their respective resistant parents to determine the overall contribution of the resistant parent. With the exception of the 67A13 composite, chi-square values were lower than 0.05. This may be due to the accumulated effect of more observed resistant plants than calculated than would be noted when each population was studied separately.

Tablegreen was found to behave quite differently than the other susceptible parents. Observations of the  $F_2$  populations showed that possibly Tablegreen may have one gene for resistance and its crosses appeared to be segregating for the other gene. A ratio of 3 susceptible to 1 resistant was utilized in the chi-square test and a good fit was obtained for the 4  $F_2$  populations with Tablegreen as the susceptible parent. In a personal communication with Dr. J. C. Gilbert, it was learned that 67A9 and 67A13 were advanced selections of crosses with a cucumber accession from Cornell University. This accession had good CMV resistance and was found to be segregating for WMV resistance. Tablegreen was developed at Cornell by Dr. H. Munger and possibly received its gene for resistance to WMV from the same accession. Marketmore also was developed at Cornell as an improved Tablegreen. It appears to have lost the gene for partial resistance to WMV 2.

No segregation occurred in  $F_2$  populations of the crosses of 67A9 and 67A13 with SM and ST. The progeny were similar to both parents in resistance indicating that all 4 resistant lines have similar

resistance genes. Although backcrosses were made, they were not tested as the  $F_2$ 's showed no segregation.

Observations of the backcross populations to both the susceptible and the resistant parents confirmed that resistance is probably controlled by 2 recessive genes. In backcrosses to the susceptible parents, all progeny should be susceptible if resistance is conveyed by recessive genes. A series of plantings consisting of backcrosses to the susceptible parents was made and all progeny was found to be susceptible except for a few symptomless plants in several populations (Table 5). These may have been escapes, an admixture of seed or possibly volunteer plants emerging from previous plantings.

One of the most important series of crosses involved the backcrosses to the resistant parents where the segregation pattern would or would not confirm the genetic ratios postulated for the  $F_2$  populations. In this phase of study the segregation of plants in the backcrosses to the resistant parents substantiated the findings observed in the  $F_2$ . A 2 gene recessive model theoretically would have a ratio of 3 susceptible to 1 resistant in backcrosses to the resistant parent. Chi-square values were computed and a good fit was obtained for the 3:1 backcross ratio (Table 6). Grouping the 5 susceptible parents under their respective resistant parent also provided a good fit to the 3:1 ratio. Tablegreen which was postulated to have 1 recessive gene was computed separately. This was confirmed in backcrosses to the resistant parents. A good fit was obtained in the 4 backcrosses to the theoretical 1 susceptible to 1 resistant ratio when Tablegreen was used as the susceptible parent.

Table 5. Summary of the segregation distribution for the backcrosses to the susceptible parents exposed to WMV 2 infection

Backcross	Susceptible	Resistant
(A & C x 67A9) x A & C	159	
(A & C x 67A13) x A & C	150	
(A & C x SM) x A & C	180	
(A & C x ST) x A & C	182	1
(Ashley x 67A9) x Ashley	163	
(Ashley x 67A13) x Ashley	157	
(Ashley x SM) x Ashley	174	
(Ashley x ST) x Ashley	157	
(Marketer x 67A9) x Marketer	172	
(Marketer x 67A13) x Marketer	144	
(Marketer x SM) x Marketer	179	
(Marketer x ST) x Marketer	192	1
(Marketmore x 67A9) x Marketmore	169	
(Marketmore x 67A13) x Marketmore	174	
(Marketmore x SM) x Marketmore	180	
(Marketmore x ST) x Marketmore	165	2
(Str. 8 x 67A9) x Str. 8	165	
(Str. 8 x 67A13) x Str. 8	164	
(Str. 8 x SM) x Str. 8	174	
(Str. 8 x ST) x Str. 8	162	2
(Tablegreen x 67A9) x Tablegreen	164	3
(Tablegreen x 67A13) x Tablegreen	143	6
(Tablegreen x SM) x Tablegreen	172	1
(Tablegreen x ST) x Tablegreen	168	

Table 6. Summary of the segregation distribution and chi-square values for the backcrosses to the resistant parents exposed to WMV 2 infection

Backcross	Susceptible	Resistant	Chi-square	P
<u>67A9</u>				
(A & C x 67A9) x 67A9	125	42	0.0015	.90
(Ashley x 67A9) x 67A9	136	46	0.2790	.70
(Marketer x 67A9) x 67A9	132	46	0.0523	.80
(Marketmore x 67A9) x 67A9	138	53	0.7341	.50
(Straight Eight x 67A9) x 67A9	133	51	0.7246	.50
Composite	664	238	0.9249	.50
<u>67A13</u>				
(A & C x 67A13) x 67A13	139	49	0.980	.50
(Ashley x 67A13) x 67A13	137	45	0.0056	.95
(Marketer x 67A13) x 67A13	130	41	0.0823	.80
(Marketmore x 67A13) x 67A13	144	52	0.2449	.70
(Straight Eight x 67A13) x 67A13	142	50	0.0861	.80
Composite	692	237	0.1434	.70
<u>SM</u>				
(A & C x SM) x SM	131	41	0.3179	.70
(Ashley x SM) x SM	121	43	0.1301	.80
(Marketer x SM) x SM	131	46	0.1918	.70
(Marketmore x SM) x SM	137	45	0.0227	.90
(Straight Eight x SM) x SM	130	50	0.4159	.70
Composite	650	225	0.2154	.70
<u>ST</u>				
(A & C x ST) x ST	135	47	0.0511	.90
(Ashley x ST) x ST	122	42	0.0153	.90
(Marketer x ST) x ST	133	49	0.5933	.50
(Marketmore x ST) x ST	138	40	0.6067	.50
(Straight Eight x ST) x ST	134	48	0.1832	.70
Composite	662	223	0.0961	.80
<u>Tablegreen</u>				
(Tablegreen x 67A9) x 67A9	112	108	0.0273	.90
(Tablegreen x 67A13) x 67A13	81	83	0.0244	.90
(Tablegreen x SM) x SM	89	85	0.0920	.80
(Tablegreen x ST) x ST	82	88	0.2118	.70

In the initial screening of cucumber cultivars for WMV 2 resistance, Biji Tunin, a cucumber accession from Indonesia, was found to show considerable resistance to WMV 2. A few basic crosses were made to determine the nature of the resistance of Biji Tunin. Although no chi-square values were computed its resistance appears to be similar to the HAES and the Sooyow lines. Seeds were inadequate to permit testing of the backcross populations (Table 7).

Table 7. Observations of cultivar Biji Tunin and its progeny exposed to WMV 2 infection

Cross	Susceptible	Resistant
BT		70
BT x AC F <sub>1</sub>	45	
BT x 67A9 F <sub>1</sub>		59
BT x AC F <sub>2</sub>	90	7
BT x 67A9 F <sub>2</sub>		105

## CONCLUSION

Thirty foundation crosses involving susceptible and resistant cucumber cultivars were tested for resistance to WMV 2. Resistance to WMV 2 was defined as a high level of tolerance. Resistant plants were able to grow out of the initial symptoms and resume normal growth and fruit production. Through a series of F<sub>1</sub>'s, F<sub>2</sub>'s, and backcrosses it was determined that WMV 2 resistance in cucumbers is controlled by 2 recessive genes. Resistance in breeding lines developed at the University of Hawaii and 2 introductions from Japan was found to be similar. Tablegreen, a cucumber cultivar with intermediate resistance, was found to have 1 recessive gene for resistance. Biji Tunin, a cucumber introduction from Indonesia, was found in a limited study to have possibly the same resistance genes as the Hawaii and Japan lines.

#### LITERATURE CITED

1. Alderz, W. C. 1969. Distribution of watermelon mosaic viruses 1 and 2 in Florida. Proc. Florida Hort. Soc. 82:161-165.
2. \_\_\_\_\_. 1972. Momordica charantia as a source of watermelon mosaic 1 for cucurbit crops in Palm Beach County, Florida. Plant Disease Reprtr. 56:563-564.
3. Anderson, C. W. 1951. Viruses of cucurbits in Central Florida. Proc. Florida Hort. Soc. 61:1109-1112.
4. \_\_\_\_\_. 1954. Two watermelon mosaic virus strains from Central Florida. Phytopath. 44:198-202.
5. Anonymous. 1973. Statistics of Hawaiian Agriculture. Hawaii Crop and Livestock Reporting Ser. 77 pp.
6. Bohn, G. W. 1973. Muskmelon breeding for virus and crown blight resistance. Hort. Science 8:253.
7. Candolle, A. De. 1882. Origine des plantes cultivies. Germes Bailliere, Paris. 377 pp.
8. Cohen, S. and F. E. Nitzany. 1963. Identity of viruses affecting cucurbits in Israel. Phytopath. 53:193-196.
9. \_\_\_\_\_, E. Gertman, and N. Kedar. 1971. Inheritance of resistance to melon mosaic virus in cucumber. Phytopath 61:253-255.
10. Courdriet, D. L. 1962. Efficiency of various insects as vectors of cucumber mosaic and watermelon mosaic viruses in cantaloupes. J. Econ. Entomol. 55:519-520.
11. Demski, D. W. 1968. Local lesion reactions of Chenopodium species to watermelon mosaic virus 2. Phytopath 58:1196-1197.
12. \_\_\_\_\_ and J. H. Chalkley. 1972. Effect of watermelon mosaic virus on yield and marketability of summer squash. Plant Disease Reprtr. 56:147-150.
13. \_\_\_\_\_ and \_\_\_\_\_. 1974. Influence of watermelon mosaic virus on watermelon. Plant Disease Reprtr. 58:195-198.
14. \_\_\_\_\_ and G. Sowell, Jr. 1970. Susceptibility of Cucurbita pepo and Citrullus lanatus introduction to watermelon mosaic virus 2. Plant Disease Reprtr. 54:880-881.

15. Diaz, F. F. 1972. Identification and destruction of watermelon mosaic virus in El Salvador. *Plant Disease Repr.* 56:437-440.
16. Elmer, O. H. 1927. A mosaic resistant variety of cucumber. *Phytopath.* 17:18.
17. Fischer, H. M. and B. E. L. Lockhart. 1974. Serious losses in cucurbits caused by watermelon mosaic virus in Morocco. *Plant Disease Repr.* 58:143-146.
18. Foster, R. E. and H. E. Dennis. 1963. Resistance in muskmelon to a strain of watermelon mosaic virus. *Phytopath.* 53:600-602.
19. \_\_\_\_\_ and R. E. Webb. 1966. High temperature masking of mosaic symptoms in muskmelons. *Phytopath.* 56:146.
20. Freitag, J. H. 1952. Seven virus diseases of cucurbits in California. *Phytopath.* 42:8.
21. Gilbert, J. C. 1959. Disease resistant cucumbers for Hawaii. *Hawaii Farm Science* 7:1-12.
22. Greber, R. S. 1969. Viruses infecting cucurbits in Queensland. *Qd. J. Agric. Anim. Sci.* 26:145-171.
23. Grogan, R. G., D. H. Hall, and K. A. Kimble. 1959. Cucurbit mosaic viruses in California. *Phytopath.* 49:366-376.
24. Komuro, Y. 1956. Studies on a mosaic disease of squash in Japan. Its symptoms, host range and transmission. *Ann. Phytopath. Soc. of Japan* 21:162-166.
25. \_\_\_\_\_. 1957. Studies on a mosaic disease of squash in Japan. II. Physical properties and identification of its casual virus, with special reference to the relationship to the virus from mosaic plants of white gourd, oriental pickling melon and watermelon. *Ann. Phytopath. Soc. Japan* 22:220-224.
26. \_\_\_\_\_. 1962. Virus diseases of cucumber and watermelon caused by melon mosaic virus. *Ann. Phytopath. Soc. Japan* 27:31-36.
27. \_\_\_\_\_. 1966. Identification of viruses affecting vegetables and ornamentals in Japan. *Ann. Phytopath. Soc. Japan* 32:114-117.
28. Kooistra, E. 1969. The inheritance of resistance to Cucumis virus 1 in cucumber (Cucumis sativus L.). *Euphytica* 18:326-333.
29. Lastra, R. 1968. Occurrence of cucurbit viruses in Venezuela. *Plant Disease Repr.* 52:171-174.

30. Leppik, E. E. 1966. Searching gene centers of the genus Cucumis through host-parasite relationships. *Euphytica* 15:323-328.
31. Lindberg, G. D., D. H. Hall, and J. C. Walker. 1956. A study of melon and squash mosaic viruses. *Phytopath.* 46:489-495.
32. McLean, D. M. and H. M. Meyer. 1961. A survey of cucurbit viruses in lower Rio Grande Valley of Texas: Preliminary report. *Plant Disease Reprtr.* 45:137-139.
33. Millrath, G. M. and M. R. Nelson. 1968. Watermelon mosaic virus 2 and cucurbit latent virus: A new evaluation. *Phytopath.* 58:687-689.
34. Milne, K. S. and R. G. Grogan. 1968. Identification of strains of watermelon mosaic virus by serology and other characteristics. *Phytopath.* 58:1060.
35. \_\_\_\_\_ and \_\_\_\_\_. 1969. Characteristics of watermelon mosaic virus strains by serology and other properties. *Phytopath.* 59:809-818.
36. \_\_\_\_\_, \_\_\_\_\_, and K. A. Kimble. 1969. Identification of viruses affecting cucurbits in California. *Phytopath.* 59:819-828.
37. Molnar, A. and K. Schmelzer. 1964. Beitrage zur Kenntnis des Wassermelonen-mosaik-virus. *Phytopath. Z.* 51:361-384.
38. Morton, D. E. and R. E. Webb. 1963. Symptoms associated with watermelon mosaic virus in South Georgia. *Plant Disease Reprtr.* 47:772-773.
39. Namba, R. and S. Y. Higa. 1970. Transmission of watermelon mosaic virus by green peach aphids subjected to light or darkness. *J. Econ. Entomol.* 63:98-101.
40. Nelson, M. R. 1962. Effect of mosaic viruses on cantaloupes. *Phytopath.* 52:363-364.
41. \_\_\_\_\_, R. M. Allen, and D. M. Tuttle. 1962. Distribution, prevalence and importance of some cantaloupe virus diseases in Southwestern Arizona. *Plant Disease Reprtr.* 46:667-671.
42. \_\_\_\_\_, J. A. Laborde, and H. H. McDonald. 1966. Cucurbit viruses on the west coast of Mexico. *Plant Disease Reprtr.* 50:947-950.

43. Pérez, J. E. 1963. A strain of squash-mosaic virus and other cucurbit viruses found in Puerto Rico during 1958-1962. Univ. of Puerto Rico J. of Agric. 47:141-153.
44. Porter, R. H. 1929. Reaction of Chinese cucumbers to mosaic. Phytopath. 19:85.
45. \_\_\_\_\_. 1932. The reaction of cucumbers to types of mosaic. Iowa State Coll. J. of Sci. 6:95-129.
46. Provvidenti, R. and W. T. Shroeder. 1970. Epiphytotic of watermelon mosaic among Cucurbitaceae in central New York in 1969. Plant Disease Reprtr. 54:744-748.
47. Purcifull, D. E. and J. R. Edwardson. 1967. Watermelon mosaic virus: tubular inclusions in pumpkin leaves and aggregates in leaf extracts. Virology 32:393-401.
48. Schmelzer, K. and D. Milicic. 1966. Zur Kenntis der Verbreitung des Wassermelonen-mosaik-virus in Europe und seiner Fahigkeit zur Beldung von Zellinschlusskorpern. Phytopath. Z. 57:8-16.
49. Shanmugasundaram, S., M. Ishii, J. C. Gilbert, and H. Nagai. 1969. Cucurbit virus studies in Hawaii. Plant Disease Reprtr. 53:70-74.
50. Shifriss, O., C. H. Myers, and C. Chupp. 1942. Resistance to mosaic virus in Cucumber. Phytopath. 32:773-784.
51. Simons, J. N. 1958. Virus diseases affecting vegetables in South Florida. Proc. Florida Hort. Soc. 71:31-34.
52. Sitterly, W. R. 1973. Breeding for disease resistance in cucurbits. Ann. Rev. of Phytopath. 11:471-491.
53. Skotland, C. B., R. L. Clark, and R. E. Webb. 1963. Watermelon mosaic virus in Washington. Plant Disease Reprtr. 47:774-775.
54. Sowell, G., Jr. and J. W. Demski. 1969. Susceptibility of watermelon cultivars to watermelon mosaic virus 2. Plant Disease Reprtr. 53:208-209.
55. Thomas, W. 1971. The incidence and economic importance of watermelon mosaic virus. New Zealand J. Agric. Res. 14:242-247.
56. Toba, H. H. 1962. Studies on the host range of watermelon mosaic virus in Hawaii. Plant Disease Reprtr. 46:409-410.
57. \_\_\_\_\_. 1963. Vector-virus relationships of the watermelon mosaic virus and the green peach aphid, Myzus persicae (Sulz.). J. Econ. Entomol. 56:200-203.

58. Van Regenmortel, M. H. V. 1960. Zone electrophoresis and electron microscopy of a watermelon mosaic virus in South Africa. *Virology* 12:127-130.
59. \_\_\_\_\_, J. Brandes, and R. Bercks. 1962. Investigations on the properties of watermelon mosaic virus. *Phytopath. Z.* 45:205-216.
60. Walker, M. N. 1933. Occurrence of watermelon mosaic. *Phytopath.* 23:741-744.
61. Wasuwat, S. L. and J. C. Walker. 1961. Inheritance of resistance to cucumber mosaic virus. *Phytopath.* 51:423-428.
62. \_\_\_\_\_ and \_\_\_\_\_. 1961. Relative concentration of cucumber mosaic virus in a resistant and a susceptible cucumber variety. *Phytopath.* 51: 614-615.
63. Webb, R. E. 1961. Distribution of some cucurbit viruses in some Southwest melon producing areas. *Plant Disease Repr.* 45:851-853.
64. \_\_\_\_\_. 1963. Local lesion host for some isolates of watermelon mosaic virus. *Plant Disease Repr.* 47:1036-1038.
65. \_\_\_\_\_. 1965. Luffa acutangula for separation and maintenance of watermelon mosaic virus 1 free from watermelon mosaic virus 2. *Phytopath.* 55:1379-1380.
66. \_\_\_\_\_. 1967. Cantaloupe breeding line B66-5: highly resistant to watermelon mosaic 1. *HortScience* 2:58-59.
67. \_\_\_\_\_. 1971. Watermelon mosaic viruses 1 and 2 in squash on the Atlantic Seaboard. *Plant Disease Repr.* 55:132-135.
68. \_\_\_\_\_ and G. W. Bohn. 1962. Resistance to cucurbit viruses in Cucumis melo. *Phytopath.* 52:1221.
69. \_\_\_\_\_ and H. A. Scott. 1964. Relations of 10 isolates of watermelon mosaic virus (WMV). *Phytopath.* 54:749.
70. \_\_\_\_\_ and \_\_\_\_\_. 1965. Isolation and distribution of watermelon mosaic viruses 1 and 2. *Phytopath.* 55:895-900.
71. \_\_\_\_\_, G. W. Bohn, and H. A. Scott. 1965. Watermelon mosaic viruses 1 and 2 in southern and western cucurbit production areas. *Plant Disease Repr.* 49:532-535.
72. Whitaker, T. W. and G. W. Bohn. 1954. Mosaic reaction and geographic origin of accessions of Cucumis melo L. *Plant Disease Repr.* 38:838-340.

73. Whitaker, T. W. and G. N. Davis. 1962. Cucurbits, Botany, Cultivation and Utilization. World Crops Books, Interscience Publ., Inc. New York. 249 pp.
74. Zabala, S. and J. C. Ramallo. 1969. Mosaic of cucurbits. Rev. agron. Noroeste Argent. 6:197-208.