INHERITANCE OF RESISTANCE TO POWDERY MILDEW IN CUCUMIS MELO

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ABSTRACT

INHERITANCE OF RESISTANCE TO POWDERY MILDEW
IN CUCUMIS MELO

by Richard Roland Harwood

A detailed study of the inheritance of resistance to powdery mildew (Erysiphe cichoracearum D.C.) in the cantaloupe variety Seminole showed resistance to be governed by two partially dominant genes. A greenhouse seedling screening was used to determine levels of resistance, with the degree of mildew infection being rated visually on a 5-category scale. The scale was adjusted to near-linearity, but the population distributions were found to be anormal. A partitioning method based on non-parametric statistics was used to separate environmental from genetic components of variance. This method was especially effective in handling low levels of resistance where environmental effects caused an overlap of the two parents.

Genetic comparison of several sources of powdery mildew resistance revealed the existance of genes different from the previously reported Pm^1 and Pm^2. The U. S. Department of Agriculture Plant Introduction 124111 which was used as a source of resistance to race 2 of the mildew in California contained a single dominant gene giving excellent resistance in Michigan. This gene was designated Pm^3. The two genes in Seminole were found to be different from Pm^1, Pm^2, or Pm^3 and were designated as Pm^4 and Pm^5. Other genes giving good resistance were noted, but their designation was delayed pending further analysis of their relationship to the reported genes. Evidence was presented for the existance of genes

giving very low levels of resistance which added to that of the major genes.

The genes Pm^1 and Pm^2 were found to act in an epistatic manner, with Pm^1 giving good resistance in Michigan and Pm^2 giving no resistance. Pm^1 and Pm^2 acting together gave increased resistance. Preliminary screening with mildew race 2 in California indicated that resistance was controlled by the epistatic interaction of Pm^1 and Pm^2.

INHERITANCE OF RESISTANCE TO POWDERY MILDEW IN *CUCUMIS MELO*

By

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INTRODUCTION

A study was begun in 1964 at the Michigan Agricultural Experiment Station to determine the inheritance of resistance to powdery mildew (Erysiphe cichoracearum D.C.) of the cantaloupe variety Seminole. The early results of this study were reported by Harwood (8), Harwood and Markarian (7), and by Markarian and Harwood (16).

Subsequent work brought about the refinement of technique and the verification of results. The scope of the study was then broadened to include the observation of representative genetic material from the known sources of mildew resistance.

The results reported here are not conclusive concerning all of the genetic factors which influence powdery mildew resistance in Cucumis melo. It is hoped they will serve as an outline of the methods and genetic materials for subsequent work designed to reach this end.

The work is reported in four parts: The sources of genetic material used; the development of a resistance evaluation technique; the analysis of resistance in Seminole and the genetic evaluation of resistance from other known sources.

LITERATURE REVIEW

Controlled Pollinations

The technique for hand pollination of muskmelons is one of the first problems which must be considered before starting a muskmelon genetic study. Muskmelons are rather difficult to work with, both from the standpoint of the induction of female flowers and the setting of fruit. A knowledge of the pattern of fruit set is an essential prerequisite to the manipulation of the plant. Rosa (24) described the basic pattern of fruit set and suggested a method of pruning to increase set. Wolf and Hartman (33) improved upon this technique. Whitaker and Pryor (29) reported some increase in set using growth regulators. The most recent analysis of muskmelon fruit set patterns was that of McGlasson and Pratt (18). Successful hand pollinations may range from 10% to 70%.

The Pathogen

A review of the morphology and host range of the pathogen, Erysiphe cichoracearum D.C. was done by Harwood (8). The features of fungal morphology and behavior which especially pertain to the current work are its dissemination by wind-blown asexual conidia and its temperature sensitivity for multiplication.

The races of mildew seem to be rather uniform in distribution. Race 2 or at least a race similar to it in pathogenicity appears across the entire southern United States. Response in Costa Rica has been reported as being similar (19). Mildew in Michigan and New York is similar to race 1 in pathogenicity, thus resistant material from any source gives resistance

in these areas.

Screening

The basic screening technique used was patterned after a greenhouse technique developed by Pryor et. al. (23) and was reported by Markarian and Harwood (16).

Genetics of Resistance

The first important commercial variety of muskmelons having resistance to powdery mildew was PMR 45, which was released in 1934 (13). Jagger et. al. (12) reported its resistance as being due to a single dominant gene which they named Pm^1. In 1938 a new biotic form of powdery mildew was reported (11) which was known as race 2. Since that time several varieties having resistance to this new race have been released. A study of the resistance to race 2 by Bohn and Whitaker (3) has revealed the presence of a single dominant gene Pm^2 and several epistatic modifiers.

A study in Egypt (26) has indicated the presence of two genes in native Egyptian material, each of which gave good powdery mildew resistance. The relationship of these genes to those in American material is not known.

Genetic Material

The current study was originally concerned with the resistance of the Florida variety Seminole (32) and the relationship of the genes responsible to Pm^1 and Pm^2. It soon became apparent that the inheritance of resistance in the U.S.D.A.-California material was more complex than supposed. The pedigrees of the material were traced (Figures 1 and 2) in an attempt to more objectively select material for evaluation in determing the genetics of resistance in the major sources.

Figure 1. Powdery mildew resistant muskmelons of the southeastern U.S.

A. Pedigree of Florida varieties and others such as Delta Gold (Louisiana) and Edisto (South Carolina) having the same source of resistance. Resistance was derived from PI 124112 (Pm^4, Pm^5) and probably from Louisiana 8-2 and 7-1 as well as some "minor" genes being derived from Smith's Perfect and others.

B. Pedigree of the Florida variety Floridew and its source of resistance in PI 223637.

POWDERY MILDEW RESISTANT MUSKMELONS

OF THE SOUTHEASTERN U.S.

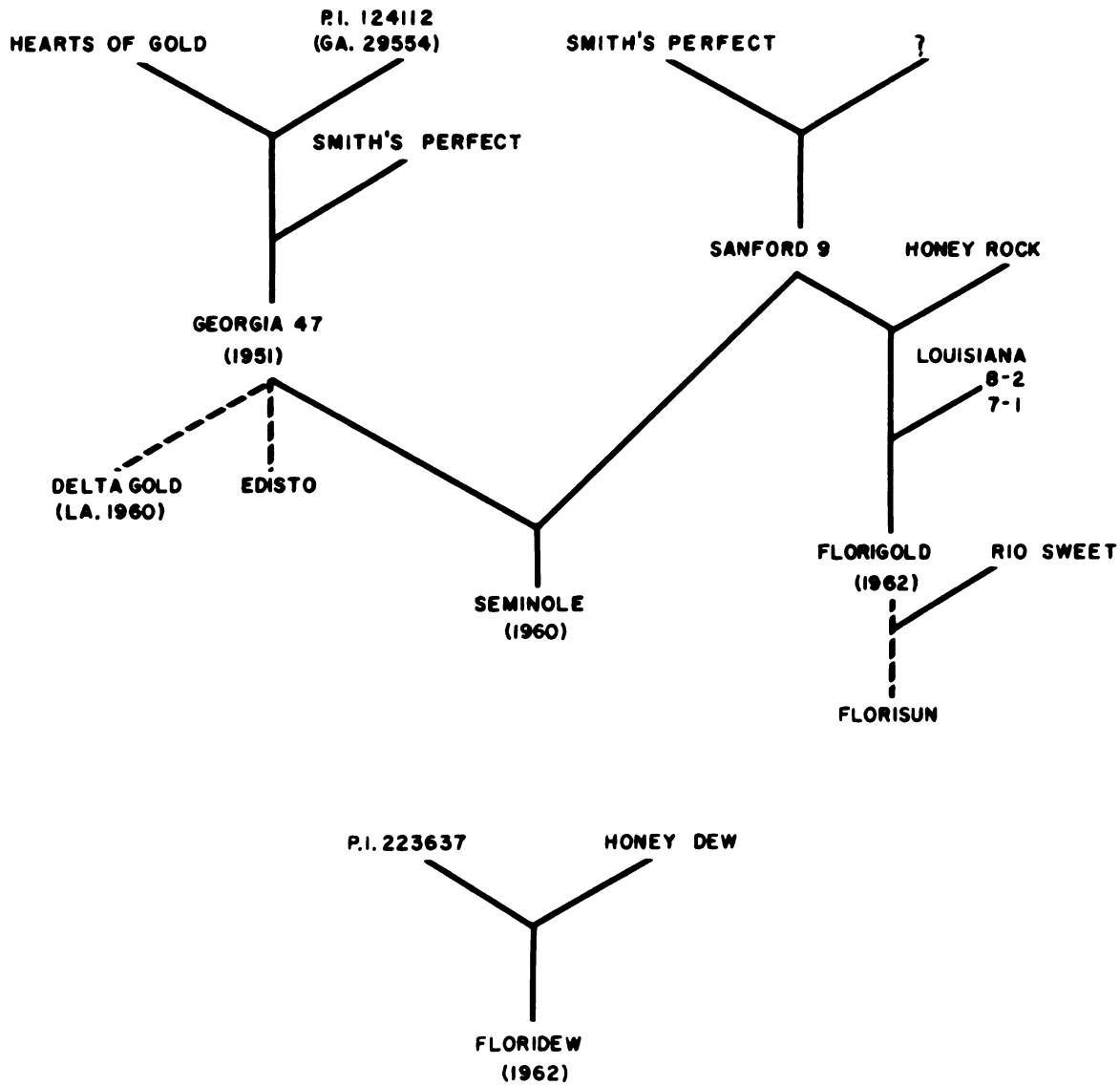
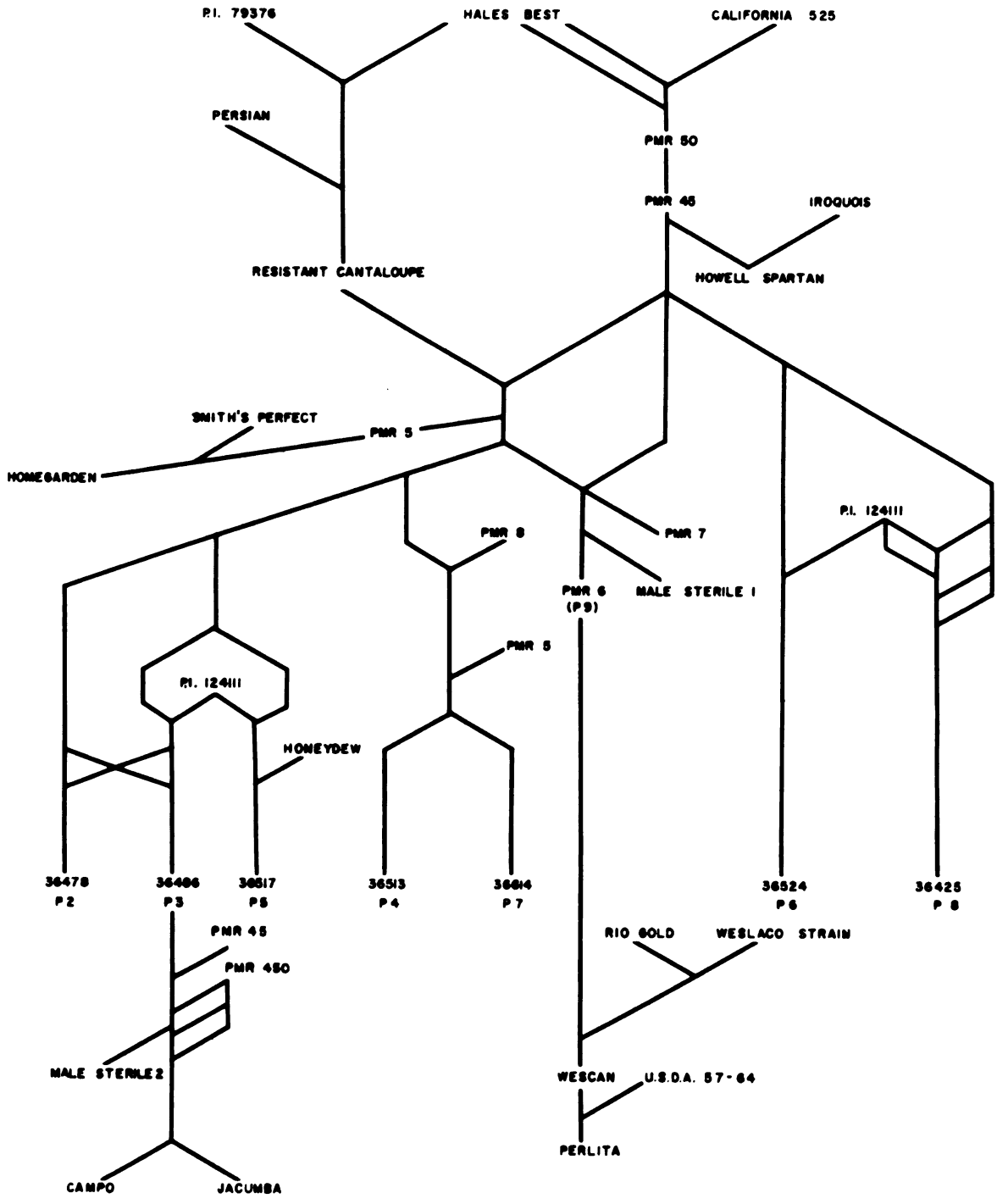


Figure 2. Muskmelon powdery mildew resistance derived from California Sources.

The known sources and their probable gene contributions were California 525, Pm^1, PI 79376, Pm^1, Pm^2, and PI 124111, Pm^3. Varieties developed from this material at other stations include Howell Spartan at Michigan, Homegarden at Louisiana and Wescan and Perlita at Texas.

MUSKMELON POWDERY MILDEW RESISTANCE

DERIVED FROM CALIFORNIA SOURCES



AFTER BOHN 1961

The outline which provided the basic information on the California material was published by Bohn (4). This information is supplemented in the present report by the pedigrees of material derived from the California material by the major muskmelon breeding centers.

The first powdery mildew resistant variety was PMR 45 (13). This was followed in 1943 by the race 2 resistant PMR #5 (31), also referred to as U.S.D.A. #5. In 1946 the varieties PMR #6 and #7 were released (30). In 1949 a source of male sterility (ms^1ms^1) was released (5) having the same pedigree as #5, #6, and #7 and the same mildew resistance. In 1951 the variety Georgia 47 was released, having derived its resistance from a Plant Introduction line closely related to the one from which later California material derived its resistance (28). There is some confusion on this point, since the source of resistance listed in the release was given as PI 29554, whereas the U.S.D.A. summary (27) lists the source as PI 124112. This variety has served as a source of resistance for many of the lines developed for the southeastern U.S. such as Edisto (9) in 1957 and Delta Gold and Seminole (32) in 1960.

Varieties subsequently developed from the California material included Rio Gold (6) in Texas, Homegarden (10) in Mississippi, Wescan (12) and Perlita (20) in Texas. Other California melons included Male Sterile #2 (2) and Campo and Jacumba (1). The variety Howell Spartan has PMR 45 as a parent (17). The Varieties Floridew (15) and Florisun (14) have resistance derived from Louisiana lines which probably originated in Georgia 47.

Information on the PI lines which were furnished by the U.S.D.A. plant collection center at Experiment, Georgia, is contained in a U.S.D.A. Cucumis melo summary (27).

METHODS

Crosses

The methods used in making the crosses and in screening for resistance were essentially the same as reported for the earlier portion of the study (8). All plants used in making crosses were grown in the greenhouse in 8" clay pots. They were pruned to a single main runner which was trained on a stake to a height of three feet. Laterals were allowed to develop beginning at the 4th and 5th node. These were pruned, in turn, at the 3rd or 4th node to enhance growth of the female or the perfect flower at the first and second nodes of the laterals. Since the flowers at these nodes are the strongest and the most likely to set fruit after pollination (24), the training and pruning of the plant was designed to maximize their growth and development.

Crosses were made by emasculating the perfect flowers of the female parent the day before opening. The corollas were left intact in this process. Insect control in the greenhouse was good, but the corollas were held closed both before and after pollination by #1 Tip Top paper clips as further precaution against pollen contamination. Whenever possible the crosses were planned so as to use either a gynoecious or a monoecious female parent. The two major advantages to this approach are the elimination of emasculation and its source of pollen contamination, and the increased percentage of successful pollinations on the stronger female flowers produced. The overall estimate of successful pollinations under greenhouse conditions in the summertime on this type of plant was in excess of 75%. This is well above results reported for field crosses and was an important factor in making the 500 crosses for this study.

Screening

The screening for resistance was essentially the same as used previously (7,8). The seedlings were grown in 3 ft. x 50 ft. greenhouse soil benches in the winter when house temperatures could be lowered as desired. The seeds were planted directly into the soil in rows running across the bench, with 150 rows per bench and 20 seeds per row. The temperature was held at 75 F. until the primary leaves were 1/2 inch in diameter, then lowered to 60 F. for the remainder of the screening. Inoculation was achieved by breath-blowing of conidia from heavily infected source plants over the seedlings at the time the temperature was lowered. Ratings of the degree of infection were made three weeks after inoculation using the previously reported 5-category rating scale (Markarian and Harwood (16), Harwood (8)). In screenings conducted during the months of October and November when there was abundant sunshine, greenhouse air temperature of 60-65 F. seemed optimum for mildew development and at the same time limited seedling growth. In December and January screenings when there were few days of sunshine, it was necessary to maintain daytime air temperature between 70 and 73 F. in order to have satisfactory mildew development. This was attributed to the fact that under the influence of direct sunshine, leaf temperatures may average 5-8 F. higher than air temperature when there is little air movement in the greenhouse. In cloudy weather the leaf temperature may be lower than ambiant air temperature because of radiation. Since the growth of mildew is dependent upon the microclimate of the leaf surface, factors which affect this microclimate become important in the resistance evaluation process.

Evaluation of Mildew Infection

A major difficulty with evaluation of plant material for amount of fungal growth occurs with the absence of a satisfactory quantitative method for measuring the amount of mildew present. It would be possible, although tedious, to measure the surface area affected, but this would not account for differences in the type of fungal growth. Most pathologists and geneticists have therefore resorted to visual evaluation of the degree of infection (23). Some, such as Bohn and Whitaker (3) have used microscopic evaluation to detect minor differences in degree and type. Two major difficulties are encountered when microscopic evaluation is attempted. First, the method is slow and requires detailed evaluation of each plant, which limits the number which can be handled. Secondly, environmental variation may be sufficiently great to limit the effectiveness and need for detailed evaluation.

In adopting an evaluation method consideration should be given both to the magnitude of the genetic and environmental effects and to numbers of plants required. A third and important factor is the adaptation of the method to statistical analysis. A major difficulty with a rating scale whereby plants are assigned to categories by a visual evaluation is that the scale of the categories is not necessarily mathematically linear nor even continuous. This, of itself, precludes the use of means, standard deviations, etc. in analysis of the results. When such a scale is used, the categories are generally placed into more or less arbitrary groupings and labeled "resistant", "moderately resistant", and "susceptible." Although this method is useful in many cases, it has obvious statistical weaknesses.

Adjustment of the Rating Scale

In a standard type of evaluation of a metrical character, for example the height of plants in a homogeneous population, measurements of the height of each plant are made. The scale used is linear and continuous by nature of the measuring device used. If the individual heights are plotted on a frequency diagram it could be expected that the variation would be normally distributed about a mean value. If this same population of plants were rated according to a short, medium, high type of classification in such a manner that the rating procedure was entirely objective, the rating categories could be adjusted to a continuous and linear scale by adjusting their values so that variation was distributed normally. This was done in determining the criteria for the 0-4 rating scale used on the melon seedlings. Several homogeneous populations totaling about 5,000 individuals were rated several times using different criteria for establishing the categories. Where the populations showed abnormal distributions, categories were added, deleted or altered in such a way as to adjust the distributions to normality or as near as possible. Importance was given to the identification of distinctive criteria upon which to base the various categories in order to achieve a maximum of objectivity in the ratings.

The problem of locating homogeneous populations which fell in the center of the rating scale arose in the early screenings. As a result, heavy emphasis was placed on the parental populations which could not be expected to show true normal distributions in this case, because they had mean values close to the end categories of 0 and 4. This indicated that there was an accumulation of individuals at the end points of the scale, which

might have fallen further out if the scale had been extended. Environmental variation was thus not a random effect, but was determined partially by the limitation of the scale. These parental distributions, then, could only be used as indicators of approximate linearity of scale because of the large proportion of their numbers falling in the open-interval categories.

With the screening of large F_3 populations of the Seminole x Delicious 51 cross, several F_3 families were seen which were probably homozygous and which did fall in the central region of the rating scale (Table 4). Homozygosity was determined by selecting those populations whose variance was less than the average of P_1, P_2 and their F_1. A test for symmetry and kurtosis in this population, following the method reported in Snedecor (25) shows a value of $g_1 = 1.17$ for the moment of the third power of deviation about the mean. This gives a t value of 5.086, including highly significant asymmetry in the distribution. Likewise the g_2 value of 3.01 with its t of 6.689 shows highly significant kurtosis.

If these deviations were caused by abnormalities in the scale, corrections should be possible by adjustment of the rating values. A close analysis of the F_1 and F_3 populations 28, 31, 33, 40 and 45 shows them all to be skewed to the lower values. Most of them also show positive kurtosis. With the means of these populations falling from values of 0.83 to 3.19, the asymmetry covers the whole range of scale. A correction of scale for one population would increase asymmetry in others. It would thus appear that this asymmetry is not solely a function of abnormality of scale. Since these populations contain different genetic bases for mildew resistance it

can be assumed that the environmental effects are different for different genes. The distribution of these F_3 populations does indicate, however, that the scale is sufficiently linear to permit the use of means and standard deviations even though the distributions are not normal. The genetic analysis must therefore be based on non-parametric methods which make use of order statistics.

Uniformity of Screening

The uniformity of screening is shown by the representative data of Table 1. The populations of Delicious 51 were single row populations which were planted every 10 rows in the benches as a check for mildew infection. Each population was comprised of approximately 20 plants. These data were taken from the single benches having the greatest variability. As can be seen, the coefficients of variation for these small groups are 15% and 11% for the 1965 and 1966 screenings respectively. In the segregating populations of the different crosses of Gynoecious (USDA 5 x Seminole) each population totaled about 200 individuals, giving a much smaller standard deviation and coefficient of variation.

These data attest to the uniformity of the screening for both homozygous and segregating populations. This uniformity is seen to hold from year to year as well as within a single screening. On the basis of these results the data were totaled over all screenings for the analysis of Seminole resistance.

Table 1 Frequency Distribution of Resistance Ratings
(Number of plants per category)

Population	0	1	2	3	4	Mean	Variance	Average of means
Del. 51 (1965)				4	14	3.78	0.160	
"				7	12	3.63	0.145	
"				5	11	3.68	0.214	3.62 ± .56
"				2	16	3.88	0.980	cv = .154
"			3	10	7	3.20	0.460	
"				4	13	3.76	0.180	
"				9	11	3.55	0.248	
"				9	8	3.47	0.265	
" Total			3	50	92	3.61	0.280	
Del. 51 (Nov.'66			1	13	4	3.16	0.194	
" Bench 6)			1	16	3	3.10	0.248	
"		1	1	18	1	2.91	0.276	
"			1	10	6	3.29	0.325	3.35 ± .37
"				12	6	3.33	0.222	cv = .110
"			1	8	8	3.41	0.268	
"			1	5	14	3.65	0.328	
"					23	4.0	0	
" Total		1	6	82	65	3.37	.403	3.37 ± .63
Gyn. (USDAxSEM)	123	27		7	37	1.01	2.495	
"	117	38	1	7	41	1.10	2.514	
"	121	59	2	2	57	1.23	2.618	1.17 ± .109
"	114	43	10	1	54	1.13	2.418	cv = .093
"	111	39	3	6	55	1.32	2.855	
"	87	41	1	6	38	1.23	2.572	

GENETIC ANALYSIS OF SEMINOLE RESISTANCE

In an early analysis of the Seminole x Delicious 51 crosses (8), an empirical method was used to partition the variance of the populations using frequency distributions. This method was similar to that proposed by Powers (22) in dealing with what he called Type I data. No consideration was given to normality of the distributions or to the means or standard deviation.

The method used here is somewhat similar to the Power's Type III method of partitioning variance where classes are grouped according to the means and variances.

The mean values of the Seminole x Delicious 51 and Seminole x Gynoecious crosses (Table 2) were used to determine mean values for each genotype in a two-gene model (Table 3). The Delicious 51 x Seminole F_3#10 had been previously selected as being homozygous for one of the genes for resistance in Seminole. It was, in turn, crossed to the susceptible Gynoecious parent, and the F_2 and backcross populations produced. The proposed genotypes and their observed mean values are listed in Table 3. These observed values were then used to give mean values for the genotypes in the Delicious 51 x Seminole F_1 backcrosses to both parents. In the backcross to Seminole, for instance, only the value for the genotype aaBb was needed. Since an observed value was available for the mean of the backcross population, the mean of this genotype could be determined. The same was done for the backcross to Delicious 51.

With the means for these genotypes determined, an F_2 was constructed as a test of the hypothesis. This was done by multiplying the mean for each genotype by its expected F_2 frequency, then totaling the values to arrive at

Table 2 Frequency Distribution of Resistance Ratings for Seminole Crosses
(Number of plants per category)

Population	Rating					Mean	Variance
	0	1	2	3	4		
Seminole	158	25	15	0	0	.277	.354
Delicious 51	0	1	6	82	65	3.37	.403
Del.xSem. F_1	27	41	81	1	0	1.37	.607
Del.xSem. F_2	332	166	388	184	111	1.65	1.296
(Del.xSem.) Del.B_1	12	30	95	58	25	2.13	1.026
(Del.xSem.) Sem. B_1	154	12	72	19	0	.86	1.150
Del.x Sem. F_3(Total)	960	2130	2979	1557	1105	1.97	1.376
Del. x Sem. F_3 10	6	36	69	8	0	1.66	.459
Gyn.x F_3 10 F_1	1	46	53	39	0	1.93	.630
Gyn.x F_3 10 F_2	2	22	33	106	10	2.58	.682
Gyn. (Gyn.xF_3 10)B_1	0	31	14	103	3	2.52	.700

Table 3 Observed and Computed Means for the Genotypes of the Seminole Crosses

Population	Genotype	Mean
Del. x Sem. F_3 10	AA	1.68
Gyn. x F_3 10 F_1	Aa	1.93
Gyn. x F_3 10 F_2		2.58
	AA	1.68
	Aa	1.93
	aa	3.37
Gyn. (Gyn. x $F_3$10)		2.53
	Aa	1.93
	aa	3.37
(Del.x Sem.) Sem.		.86
	AABB	.28
	AaBB	1.10*
	AABb	.90*
	AaBb	1.37
(Del.x Sem.) Del.		2.13
	AaBb	1.37
	Aabb	1.93
	aaBb	2.00*
	aabb	3.37
Del.x Sem. F_2		1.65
	AABB	.28
	AaBB	1.10
	AABb	.90
	AAbb	1.68
	AaBb	1.37
	Aabb	2.31
	aaBB	1.99
	aaBb	2.00
	aabb	3.37

*Computed values

an overall mean. The results are as follows:

	Calculated mean	Observed mean
(Gyn x $F_3$10) F_2	2.23	2.58 ± .109
(Del x Sem) Sem	.91	.86 ± .109
(Del x Sem) Del	2.17	2.13 ± .109
(Del x Sem) F_2	1.59	1.65 ± .109

Any deviations from the observed are well within the limits of error. The bimodal aspect of the F_2 distribution lends further support to the two-gene hypothesis. The model shows gene A to have 70% dominance and gene B to be somewhat more completely dominant.

The environmental effect computed from the average of the variances of the F_1 and both parents was 0.454. The observed genetic variance in the F_2 was thus 0.942. The calculated genetic variance of the F_2 as determined from the means of the component genotypes by the Powers method, was 0.499. This discrepancy will be discussed later.

The heritability in the F_2 as calculated from the Mather model was 67%.

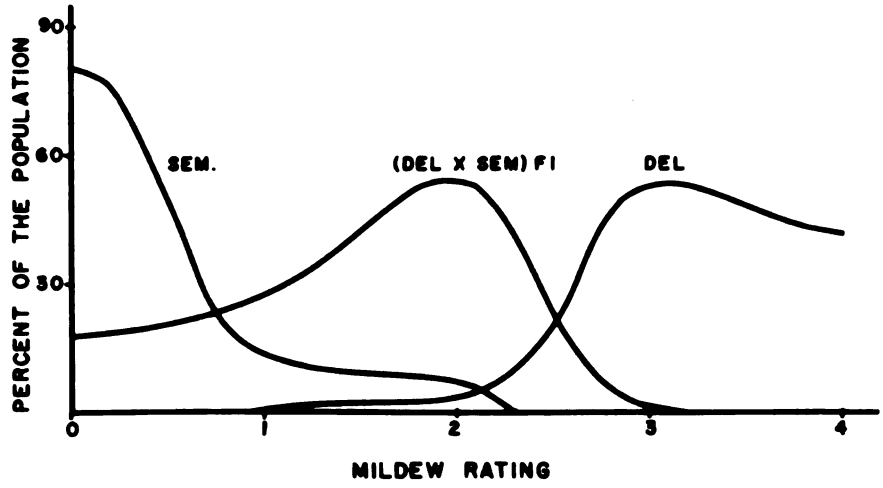
An F_3 population of 8731 individuals was screened. This came from 62 F_2 plants which were rated for mildew both in the seedling and the mature plant stage in the greenhouse. The totals are given in Table 2 and plotted in Figure 3. The 62 F_2 plants do not represent a completely unbiased sample of the F_2 population, however, as some incompatibility was present in the material so that some plants were difficult to self or produced few viable seeds. Consequently, more of the plants of these particular phenotypes were chosen so that all of the genetic material would be represented in the F_3. The individual F_3 families which showed less variance than

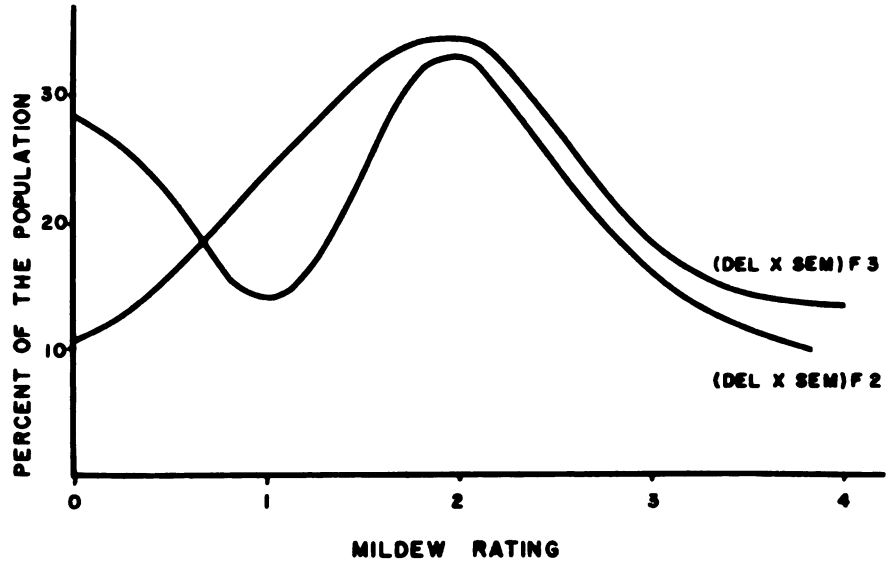
Figure 3. Populational distributions of the crosses of Delicious 51 x Seminole.

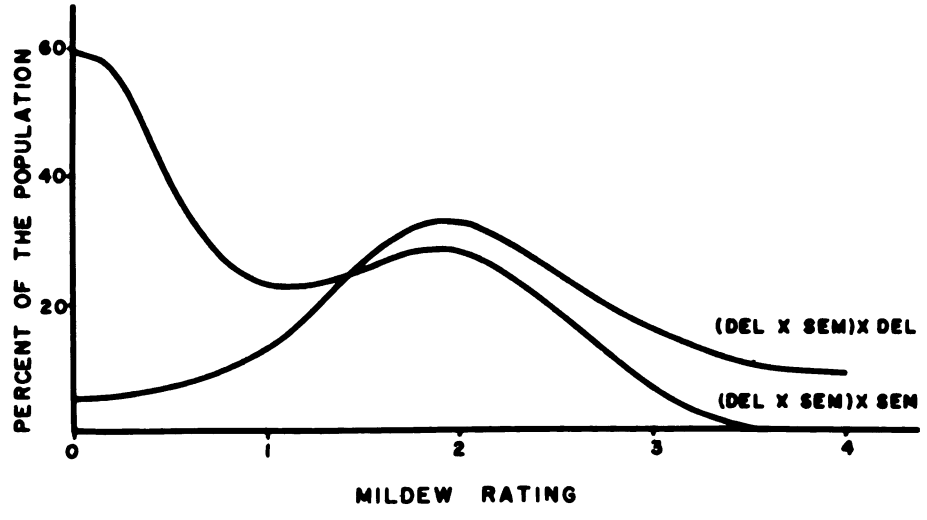
A. Resistant parent Seminole, susceptible parent Delicious 51, and their F_1, Delicious 51 x Seminole.

B. Delicious 51 x Seminole F_2 and F_3. The F_2 shows the 2-gene bimodal distribution.

C. Backcrosses of the F_1 to the resistant and to the susceptible parent. The backcross to the resistant parent shows the bimodal effect of two genes but the backcross to the susceptible merely shows a skewness toward the susceptible.


PERCENT OF THE POPULATION
90
60
30
SEM.
(DEL X SEM) F1
DEL
0
1
2
3
4
MILDEW RATING


PERCENT OF THE POPULATION
30
20
10
(DEL X SEM) F 3
(DEL X SEM) F 2
0
1
2
3
4
MILDEW RATING


PERCENT OF THE POPULATION
60
40
20
(DEL X SEM)X DEL
(DEL X SEM)X SEM
0
1
2
3
4
MILDEW RATING

the mean of the parents and F_1 were assumed to be homozygous. These are listed in Table 4, beginning with the most resistant. The mean values cover the entire rating scale. As has previously been demonstrated (Table 1), the expected standard deviation in mean values of large populations is about $\pm$ 0.109. Since there are so many different values for these F_3 populations, it would seem likely that there are additional genetic factors involved. There is, in fact, a good deal of evidence to support this hypothesis.

It is the opinion of Dr. W. Bohn* of the U.S.D.A. laboratory at La Jolla, California, that there are numerous "minor" genes for mildew resistance scattered throughout the Cucumis melo species. Dr. Henry Munger* at Cornell has noted that crosses of Delicious 51 to a resistant source seem to have slightly higher resistance than do crosses of Iroquois x resistant. This same effect was noticed in field screenings in East Lansing in 1965, where the Delicious 51 x PMR 45 F_1 had no mildew at all, while the Iroquois x PMR 45 F_1 was rated at 1/2, having a few isolated mildew colonies late in the season. The Gynoecious x PMR 45 F_1 also was rated at 1/2. These differences were not evident in a subsequent greenhouse screening, although the screening technique had not been refined. Iroquois and Delicious 51 by themselves have shown no difference in resistance in the field, but in the greenhouse Delicious 51 was rated at 3.5 while Iroquois, which was adjacent to it in the screening, was rated at 3.8.

In the Delicious 51 x Seminole F_3 several of the populations were rated significantly lower in resistance than was Delicious 51 (Table 4). This

*Personal Communication.

Table 4 Frequency Distribution of Resistance Ratings for Homozygous Seminole x Delicious 51 F_3 Populations (Number of plants per category)

Population	0	1	2	3	4	Mean	F_2 Rating	Variance
F_3 # 47	147	34	2			.21	0/0*	.184
30	112	91	14			.55	0/0	.425
56	57	65	16			.70	1/0	.441
50	38	144	3			.81	0/0	.187
45	34	136	5			.83	0/0	.195
31	13	166	31	7		1.15	1/0	.312
36		114	79			1.40	2/0	.242
40	4	106	84	1		1.42	3/0	.294
20		51	153			1.75	2/0	.188
59	12	11	167	9	1	1.88	0/0	.345
34	1	5	197	6	1	2.0	1/0	.090
55		5	39	20		2.23	3/1	.336
33		8	6	178	21	2.99	3/3	.276
28	1	3	16	156	30	3.02	4/3	.325
46			6	166	32	3.12	3/2	.245
21				60	130	3.57	4/4	.222
38			4	53	110	3.60	4/3	.281
54		4	5	37	167	3.72	4/3	.360
53		1	1	36	154	3.79	4/3	.209
61			1	9	179	3.94	4/2	.066
52	7	39	129	35	4	1.95	1/0	.554

*Seedling rating/Mature plant rating

would seem to indicate segregation of a more susceptible type. The fact that only one population was seen having as high a resistance as Seminole indicates an additional factor or factors in its resistance also. If only the two genes were responsible for its high resistance, four of the 62 F_2 plants could be expected to give Seminole type resistance in their F_3 progeny. These additional minor genes, one each in Seminole and in Delicious 51, would also account for the many different levels of resistance in the homozygous F_3 populations.

There were, in addition to the homozygous F_3 populations, several such as #52 (Table 4) whose variance was slightly higher than the homozygous, but which was not as high as for a population with segregation for a major gene. These populations were probably homozygous for gene B but contained a segregating minor gene.

One additional indicator of increased genetic variance was seen in the previously mentioned discrepancy between observed and calculated genetic variance in the F_2. The calculated variance was based on a two-gene model, while that observed for the F_2 was significantly higher because of the added effect of the minor genes in both Seminole and Delicious 51.

GENETIC SURVEY OF RESISTANCE SOURCES

In an attempt to evaluate the available genetic material having mildew resistance, a series of crosses was made between the resistant sources in question and the California sources. The plan was to look for susceptible types in the test-crosses in a test for allelism. Obtaining homozygous sources of resistance proved to be the greatest difficulty. The sources were checked for homozygosity by making their F_1 s with Gynoecious and then testing these for uniformity. Of the 35 PI lines having resistance, only 5 had plants homozygous for resistance. Resistant F_1s from the segregating lines were selfed to provide homozygous individuals for crossing at a later date.

The pedigree of the California mildew program as adapted from Bohn (4) is presented in Figure 2. Some of the varieties produced from different breeding have been included in the diagram.

Resistance of PMR 45 to race 1 of mildew has been reported (12) as being a single dominant gene, Pm^1. This has been verified in the current study (Table 5). The PMR 45 backcross to Gynoecious segregated 1:1, indicating the presence of a single dominant gene. Bohn (3) reported inheritance to race 2 of the mildew in P_2 - P_9 as being controlled by a single dominant gene and two or three epistatic modifiers. This single dominant gene was named Pm^2. Data from the current study indicate that this is probably partially correct.

Table 5 Frequency Distribution of Resistance Ratings of California Type Resistance (Number of plants per category)

Population	0	1	2	3	4	Ratio	Chi^2	P
USDA 5	18							
Gynoecious x USDA5 F_1	91	1						
Gyn.(Gyn.xUSDA5)B_1	95	3	8	72	18	1:1	0	1.0
PMR 45	20							
Gyn.(Gyn.xPMR 45)B_1	33			1	42	1:1	1.316	.2
Gynoecious			2	8	3			
Gyn. x Wescan F_1	91							
Gyn. x Wescan F_2	116	2		46		3:1	.813	.3
Gyn.(PMR 45 x Wescan)	152							
Gyn.(USDA5 x Wescan)	117							
Gyn.(Gyn.x Wescan)	54	2	2	55	2	1:1	.039	.8
Gyn. x Perlita F_1	120							
Gyn. x Perlita F_2*	117		1	22	3			
Gyn.(PMR 45 x Perlita)	135							
Gyn.(USDA5 x Perlita)	139							
Gyn.(Gyn.x Perlita)B_1*	95	7		55	24	1:1	2.92	.07
Gyn. x PI 124111 F_1	111	12						
Gyn. x PI 124111 F_2*	57	122	28	11				
Gyn.(USDA5 x PI 124111)	22	8	3	5		3:1	.316	.5
Gyn.(Gyn.x PI124111)	102	48	1	149	1	1:1	0	1.0

*These populations received poor screening because of low incidence of mildew.

Table 6 Frequency Distribution of Resistance Ratings of Sources other than California Material (Number of plants per category)

Population	0	1	2	3	4	Ratio	Chi^2	P
PMR 45 x Seminole F_1	46							
USDA5 x Seminole F_1	45							
Gyn(USDA5 x Sem.)	673	247	17	29	282			
Gyn(PMR 45 x F_3#10)	71	5	7	63	1			
Gyn(USDA5 x F_3#10)	92	9	12	48	1			
Gyn. x PI 234607 F_1	166							
Gyn. x PI 234607 F_2	127	26	3					
Gyn(PMR 45xPI 234607)	142							
Gyn(USDA5xPI 234607)	146							
Gyn(Gyn.xPI 234607)	244	66	10	117	6	3:1	5.95	.02
Gyn. x PI 236355 F_1	96							
Gyn. x PI 236355 F_2	138	16	4	10		15:1	.875	.3
Gyn(PMR 45xPI 236355)	65	8		3				
Gyn(USDA5xPI 236355)	153							
Gyn(Gyn.xPI 236355)	84	24	26	4		3:1	.785	.3
Gyn. x Bellgarde F_1	77							
Gyn. x Bellgarde F_2	34	39	9	12		3:1	.354	.5
Gyn(PMR 45xBell.)	82	20	2	9	25	3:1	.196	.6
Gyn(USDA5xBell.)	89	34	5	19	20	3:1	.169	.6
Gyn(Gyn.xBell.)	7	53	1	53	12	1:1	.286	.6
Gyn. x PI 179901 F_1	100							
Gyn(PMR 45 x PI 179901)	117							
Gyn(USDA5 x PI 179901)	128							
Gyn(Gyn.x PI 179901)	98	44	5	24		3:1	5.86	.02

In evaluations in Michigan resistant material from any other area has been resistant. The race of mildew present is probably race 1 or at least a race similar to it in its pathogenicity.

Test for Allelism With Pm^1 and Pm^2

In the study of the genetics of resistance of different sources the test for allelism with Pm^1 and Pm^2 were conducted by making the testcross Susc(Pm^1 or Pm^2 source x resistant source being evaluated). The variety PMR 45 was used as a source for Pm^1 and PMR 5 as a source for Pm^2. The results of these tests conducted with the varieties Wescan and Perlita and the PI lines 234607 and 179901, all having resistance to race 2, predictably showed no segregation when tested against PMR 5 (Table 5 and 6), indicating the presence of Pm^2. They also showed no segregation with PMR 45. This indicated the presence of Pm^1 or of a gene so closely linked to Pm^1 that no crossovers were seen in 1000 testcross individuals with these varieties. Since Wescan and Perlita derived their resistance indirectly from PMR 5 it can be assumed that this variety also contains Pm^1.

On this basis, the cross Gyn(Gyn x USDA 5) was screened here in East Lansing, giving a nearly perfect 1:1 ratio as shown in Table 5. This would seem to indicate the presence of a single dominant gene. The 95 plants which were completely clean of mildew were removed from the screening bench and planted in peat pots. They were then hardened for two weeks for shipment to California. During this period about half of them showed signs of mildew growth. Several of the plants died in shipment, but those remaining, when tested in California to race 2 of the mildew,

segregated 24 susceptible - 21 resistant*. The susceptibles were said to have PMR 45 type resistance and the resistant ones USDA 5 type. It can thus be concluded that USDA 5 resistance to race 2 is governed by two genes, Pm^1 and another dominant gene hypostatic to Pm^1. This second gene gave no resistance by itself, at least in Michigan, but in combination with Pm^1 gave increased resistance to race 1 and a moderate level of resistance to race 2. A second, but unlikely hypothesis is that Pm^2 acts alone but gives no resistance to the Michigan race of mildew.

In comparing all other material with PMR 45 and USDA 5, then, the test was against the gene Pm^1 only. The second gene in USDA 5 could not be distinguished in a cross to PMR 45 by screening with race 1.

The Texas varieties Wescan and Perlita, having been derived from PMR 6, showed no segregation when tested against PMR 45 and both showed 1:1 segregation in the Gynoecious backcross. They are both resistant to race 2, so it can be concluded that they contain Pm^1 and its epistatic modifier.

The PI 124111, when tested against USDA 5, segregated 3:1, indicating the presence of two separate genes in the F_1. These genes are Pm^1 and a different independent gene in 124111. This gene by itself gives a level of resistance in Michigan equal to that of any other source, and in addition, gives at least some resistance to race 2 (27).

*The California screening was done by Mr. Joseph Principe in the U.S. Department of Agriculture laboratory of Dr. G. W. Bohn at La Jolla.

The variety Campo was tested also, but unfortunately the screening as indicated by the Delicious 51 check row was not severe enough to be reliable. The P_3 line has been reported by Bohn (3) as having the best resistance to race 2 of all of the California lines. It seems likely that this line contains Pm^1, its epistatic modifier and the gene from PI 124111.

Seminole, as shown from the pedigree in Figure 1, derived its resistance from Georgia 47. The screening results of the cross Gyn(USDA 5 x Sem) were presented in detail in Table 1 and in summary in Table 6. On the basis of a two-gene hypothesis for Seminole and Pm^1 in USDA 5, the following genotypes could be expected in equal proportions in the testcross progeny:

Pm_1^1 pm_1^1 Aa Bb
Pm_1 pm_1^1 aa Bb
Pm^1 pm_1^1 Aa bb
Pm^1 pm^1 aa bb

pm_1^1 pm_1^1 Aa Bb
pm_1 pm_1^1 aa Bb
pm^1 pm_1^1 Aa bb
pm^1 pm^1 aabb

Since the first four contain Pm^1, their expected mean is zero. The second four are exactly the same as expected in the cross Delicious 51 (Del x Sem). The observed mean for this cross was 2.13. This gives an expected mean for the above testcross population of 1.07. The observed value (Table 1) was $1.17 \pm .109$. We can thus conclude that genes A and B of Seminole are not linked to Pm^1.

The Del 51 x Sem F_3 #10 when crossed to USDA 5 and the F_1 testcrossed to Gynoecious, would give the following genotypes in equal proportions:

Pm_1^1 pm_1^1 Aa
Pm^1 pm^1 aa
pm_1^1 pm_1^1 Aa
pm^1 pm^1 aa

The mean of the Pm^1 types, as before, is zero (as seen from the PMR 45 x Gyn F_1 - Table 5), and the mean of the second two types is the same as that of the cross Gyn(Gyn x F_3 10) which is 2.52. The expected mean for this population thus is 1.26. The observed mean is 1.12 and for the similar PMR 45 testcross, 1.44, both of which are well within the limits of variability of the calculated value. This further indicates the non-allelic relationship of gene A and Pm^1. Seminole is reported to have PI 124112 in its pedigree. This source is probably of a different genotype from PI 124111, since Seminole has no one major gene for mildew resistance.

Other material tested showed additional genes for resistance (Table 6). PI 234607 from South Africa showed no segregation with PMR 45 or USDA 5, but showed evidence of having two genes by the 3:1 ratio in its backcross. The resistant plants for all major genes fall in categories 0 and 1 and for the susceptible allele, in 2 and 4.

PI 236355 from England showed a similar pattern, with some evidence that one of its genes might be linked to Pm^1. The second gene in both cases may or may not be similar to that in PI 124111.

The Bellgarde crosses showed the presence of a single gene not linked to Pm^1. The PI 179901 showed linkage or allelism to Pm^1, but its backcross indicates the presence of at least two dominant genes.

SUMMARY

The distribution of environmental variance of mildew infection in Cucumis melo does not follow that expected for a normal distribution. The statistics used in the analysis of mildew ratings must therefore be adapted to a non-parametric distribution.

The screening of a wide range of genetic types has shown variation in the seedling response to mildew infection. Some types for instance are less prone to cotyledonary infection than others, even though the mature plant or its progeny may be more susceptible. Minor adjustments in the rating scale may be necessary for evaluation of these types.

A study of the inheritance of resistance to powdery mildew in the variety Seminole has shown resistance to be governed by two "major" and probably one "minor" gene. Gene "A" was shown to have 70% dominance and gene B somewhat more complete dominance. Their effects are partially additive. The symbols Pm^4Pm^4 are proposed for the dominant alleles of gene A and Pm^5Pm^5 for those of gene B.

The race 2 resistant variety PMR 5 and those derived from it were shown to contain the gene Pm^1. The gene Pm^2 gave a high level of resistance in combination with Pm^1. Pm^2 appears to act in an epistatic manner with Pm^1 to give resistance to race 2.

A single dominant gene giving a high level of resistance was found in the PI 124111. This gene was shown to be not linked to either Pm^1 or Pm^2. The symbols Pm^3Pm^3 are proposed to designate its dominant allele.

Other genes giving resistance in Michigan were identified but their naming is to be delayed pending a more complete analysis of their relation-

ship to Pm^3, Pm^4 and Pm^5.

From the evaluation of the available genetic material giving resistance to powdery mildew, conclusions may be drawn concerning the suitability of the material as a source of resistance in a Michigan breeding program. It has been found (16) that low levels of resistance as determined by a greenhouse seedling evaluation give effective "commercial" resistance in the field. The genes Pm^4 and Pm^5 would thus be suitable. It is more convenient and desirable, however, to use the more potent genes. Pm^1, for instance, gives a much cleaner segregation in backcross progeny and is much easier to select as well as giving somewhat better field resistance. Pm^2 is rather difficult to handle. Very close evaluation is required for its detection in the presence of Pm^1. Since there is some merit to breeding for resistance to race 2 as a precautionary measure, the selection of Pm^3 might be wise. This gene should be rechecked by itself for resistance to race 2 before it is used.

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