THE INHERITANCE OF ALTERNARIA LEAF SPOT RESISTANCE IN MUSKMELON *Cucumis melo* L.

by

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INTRODUCTION

The fungus, Alternaria cucumerina (E. & E.) J. A. Elliot, has been prevalent on various cucurbit species throughout the world especially in America and Europe since 1894 (4, 9). The most frequently reported host is muskmelon, Cucumis melo L. (9). The cantaloupe rust, or "blight" as it is called, has inflicted serious injury to the industry. The disease defoliates the vines and thereby diminishes yield and lowers melon quality by premature ripening.

Due to the rapid growth of muskmelon vines frequent spraying is required to protect new foliage. It may be impracticable to control the disease with various fungicide sprays. Therefore, it seems necessary to investigate the inheritance of resistance to the fungus and to breed resistance into new varieties.

Extensive genetic studies of resistance to rust in other crops have been conducted (1, 6, 7, 19), whereas those of muskmelon diseases have been rather limited. This might be partially due to the complicated expression of resistance to alternaria leaf spot in muskmelon (20).

An attempt was made in this study to establish a reliable sampling method to determine the number of genes involved in resistance, the type of gene action, heritability and the linkage relationship between the genes contributing to resistance and such morphological characters as marginal vein length.

REVIEW OF LITERATURE

Symptoms of Disease Infection

Leaf blight in muskmelon was first reported by Blinn (2) in the United States, in 1905, under the name of Macrosporum cucumerinum E. & E. Later, Brisley (3) proved that the disease was primarily caused by M. cucumerinum and secondarily by Alternaria cucumerina (E. & E.) J. A. Elliot which is normally a saprophytic fungus. However, at the present, muskmelon rust or "blight" is reported by the name of the secondary fungus.

The first symptoms usually appear on the leaves nearest the center of the hill (4). The spots increase rapidly in number and in size on susceptible plants, later spreading to leaves toward the tips of the vines (4). The infected areas are noticed first on the upper leaf surface as spots which are circular, 0.5 mm in diameter, watersoaked, turning yellow and later brown in color, surrounded by light green halos (4, 8). Lesions develop distinct concentric rings, giving a target-board appearance, but under conditions highly favorable for development of the pathogen these rings are often lacking (4, 8). On the lower leaf surface rings seldom occur and the lesion margins are indistinct (4). Individual lesions enlarge to 5-20 mm and become light to dark brown (4, 8). Marginal necrosis and coalescence of lesions are common, resulting later in large, torn and ragged areas which seldom approach a shot-hole appearance (8). The foliage is often curled downward and vines may be almost completely defoliated on susceptible varieties. Tomiyama (18) described a general case of disease resistance.

The browning of infected cells in resistant varieties occurs more rapidly than in the susceptible ones. Browning of the cells means the hypersensitive death of host cells. The susceptible cells infected by a parasite keep alive for a longer time than the resistant ones. Therefore hypersensitive death of the host cells is closely related to the major gene resistance in plants. Sun-scalded or over-ripe fruits are occasionally invaded by the pathogen (8) but they almost never become diseased unless the plants are grown under conditions of malnutrition. Especially the lack of minor elements, or in highly acid or strongly alkaline soil (4) this is true. Leclerg (11), however, denies the above claims that they are not caused by any organism, but by ultraviolet rays. There is no evidence of root, hypocotyl, or stem infection on plants (8). The pathogen on cucurbits rarely infects young, vigorously growing plants (4).

Genetic and Other Factors Related to Rust Resistance

The inheritance of resistance to alternaria leaf spot in muskmelon has not been previously studied in detail. There are a number of reports indicating that the exact number of genes involved was unknown or undetermined. The breeding behavior of resistance is partially known, but reports are inconsistent. The genetic explanations were assumed to be multiple genes, polyploidy, or physiologic races of the causative organism, in addition to the effects of environment (20). However, no physiologic races of the causative organism have been reported and the host is believed to be diploid instead of polyploid. Therefore, the remaining multiple genes and the effects of environment seem to be the

important factors involved in the susceptibility or resistance to the fungus.

On the other hand, the knowledge of genetics of rust resistance has been applied to and advanced in other crops. Flor (6) studied the inheritance of rust reaction in flax by crossing all possible combinations of the 11 varieties used to differentiate physiologic races of rust. He tested the F_2 plants with 24 races that had been identified from North American and South American collections of the pathogen. He suggested that genotypes of the 16 rust-differential flax varieties possess 22 pairs of rust-conditioning factors of which at least 19 are distinct and other varieties have additional rust-conditioning factors distinct from those possessed by the 16 differentials. Among them seven of the differential varieties have rust-conditioning factors in the LL series, six in the MM series, and five in the NN series. Each factor in the LL series appears to be distinct. Gough and Williams (7) reported that the resistance of each of the two durum wheat varieties Acme and Mindum to stem rust was conditioned by at least three incompletely dominant genes. The genes which conditioned higher levels of resistance were epistatic to those which conditioned lower levels. In another report on wheat Berg, Gough, and Williams (1) found that at least three dominant genes condition the resistance to stem rust in each of the two wheat varieties, Marquis and Kota. The genes also showed the same epistatic condition as in the other example in wheat. Williams and Pound (19) reported that in radish a single dominant gene governs the resistance to Albugo candida, white rust, and that resistance is modified by minor genes which are affected by environment.

Tomiyama (18) assumed a generalized genetic model of disease resistance in plants. He proposed that there are two different genetic types of resistance in various crops. One type is controlled by specific single genes in which the varieties having this type of resistance are usually highly resistant to a specific race of pathogens. However, they are susceptible to other races of the same pathogen. The other type of resistance is polygenic (quantitative). The varieties having this type of resistance are usually not so highly resistant and not stable. In other words they are easily affected by environmental conditions. The former type of resistance is called "major gene resistance", and the latter "field resistance". He further assumed that the factors limiting field resistance are classified as one in which plants become more susceptible at high and low sugar levels.

It is believed to be important to study linkage between the genes contributing to quantitative and to qualitative characters from the standpoint of plant breeding. The first case of apparent linkage between polygenes and a major gene was reported by Sax (16). He crossed two varieties of Phaseolus vulgaris, one having large colored seeds and the other with small white seeds. Seed size was a continuously variable character, but pigmentation was proved to be due to a single gene difference. The F_2 gave a ratio of 3 colored to 1 white-seeded plant. The colored F_2 plants were further classified by means of F_3 progenies into homozygotes and heterozygotes. As in the parents the gene producing colored seeds is associated with large seeds and that for white with small ones.

Rasmusson (15) investigated the variation of flowering time and the flower pigmentation in three crosses of the garden pea, Pisum sativum. The pigmentation, color versus white of the flower was governed by a single dominant gene. In the crosses linkage between the gene contributing to pigmentation and polygenes to flowering time varied from one cross to another. His work was interpreted as contradictory evidence to Sax's mentioned above by Mather (13). However, it seems that the arrangement of positive and negative polygenes on chromosomes vary from one variety to another and from one species to another. The work by Currence (5) gave results which might support the concept of linkage between a major gene and polygenes. He crossed a tomato strain, Lycopersicum esculentum, homozygous for DPOS (d = dwarf plant recessive to D the standard plant; p = pubescent recessive to P for smooth fruit; o = pear shape fruit recessive to O = round fruit and s = compound inflorescence is recessive to S = simple inflorescence) with one homozygous for dpos located on the first chromosome. The strains had different dates of fruit ripening. The parents and F_1 showed the following dates:

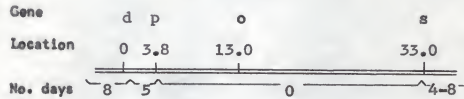
P_1 D-P-O-S (27.4 $\pm$ 0.77 days) x P_2 d-p-o-s (46.4 $\pm$ 0.62 days)

DPOD / spos

(25.6 $\pm$ 0.47 days)

The progeny of the F_1 backcrossed to dpos segregated phenotypically in the range from DPOS (24.4 $\pm$ 0.25 days) to dpos (43.8 $\pm$ 0.40 days). For example, DPos was earlier by 13.9 days than dpos and Dpos by 9.1 days.

From the results he presented, the following relationship appeared between the location of the four genes and the distribution of polygenes for ripening time:



The effects of different regions of chromosome I on ripening time.

It was found that the first chromosome had an effect of approximately 19 days on the time of fruit ripening. By repeated backcrossing and selecting for ddppooss a strain was developed that gave additional evidence of linkage. In this case the linkage relationship was between a major gene, or genes contributing to one character and polygenes to another. Moreover, the major genes were completely dominant. Therefore, linkage could be rather simply tested by using F_2 and F_3 or backcross progenies. Mather (13) proposed a method for testing linkage in quantitative inheritance studies of a character. However, there is no appropriate method for the linkage test in case that each of two characters are controlled by incompletely dominant genes, polygenes, or both.

Variable Expression of Disease Resistance of a Plant

There are some plants which show a great variation of degree of infection to causative organisms. Kahn and Libby (10) found in rice that the upper leaf surfaces were twice as susceptible to the blast fungus Pricularia oryzae as the lower surfaces and that as the age of the plant increased leaves or portions thereof progressively decreased in susceptibility to infection. A similar case (19) was also reported where white rust resistance in radish was exhibited differently on cotyledons and secondary leaves and ages. In such cases it is sometimes difficult to evaluate the precise resistance of a plant.

MATERIALS AND METHODS

The original objective of this study were to determine the genetic basis of resistance in muskmelon, Cucumis melo L., to Alternaria cucumerina (E. & E.) J. A. Elliot and to evaluate the varietal potential of each in muskmelon as germ plasm sources for a breeding program. Alternaria leaf spot and powdery mildew are the two important foliage diseases of muskmelon in this area. The latter has been controlled by the use of resistant varieties for many years.

Five commercial varieties were used. The genetic basis for resistance to alternaria leaf spot was unknown in any of them. In this study they were classified according to the degree of resistance to field infection as follows:

Table 1. The varieties used with line designations and resistance.

Variety	Line designation	Resistance
Heart of Gold	P_1	highly resistant
PMR 6	P_2	highly susceptible
Golden Gate 45	P_3	intermediate
PMR 45	P_4	intermediate
Cranshaw	P_5	resistant

P_1 was found to be the most resistant variety to alternaria and was selected as the common resistant parent in the study and was crossed with the other four varieties. Development of populations of these genetic lines began in the horticultural greenhouse in the spring of 1963 and was completed at the Ashland horticultural farm in the summer of the same year. The four different crosses and the genetic lines involved in each cross are listed in Table 2.

Table 2. Four crosses and the genetic lines involved in each cross.

Cross	Genetic lines
$P_1 \times P_2$	P_1, P_2, F_1, F_2, BC_1 and BC_2
$P_1 \times P_3$	P_1, P_3, F_1 and F_2
$P_1 \times P_4$	P_1, P_4, F_1 and F_2
$P_1 \times P_5$	P_1, P_5, F_1 and F_2

To breed these lines, 5 plants of each parental variety were first planted in the greenhouse to produce the F_1 seed. Ten F_1 plants together with the same number of each parent were grown in the field to develop all genetic lines of the four crosses for the following year. Two to 5 melons (about 300 seeds) per line were obtained by selfing or crossing and bulked within each line. Plants of the parent varieties for inbreeding as well as for genetic experiments were grown from the original seed stocks.

It was assumed that by evaluation of the genetic variance, means and types of gene action of the lines that an estimation could be made of the number of genes involved in resistance and the nature of inheritance could be determined.

Length of the base marginal leaf vein from the point of the petiole junction to the first branch vein (Plate I) appeared to be related to and was studied to determine if the genes for resistance were closely linked with those for a vein length. If so, one would be able to distinguish resistant from susceptible plants phenotypically with more accuracy and selection could be made at an early stage of development. The vein length genes seemed to be closely linked with the resistant genes when the parent varieties were observed.

Seed of all lines from each of the four crosses were planted in the field with a spacing of 3 feet within the row, 6 feet between rows and 6 hills per row. In order to detect and evaluate all recombinants in the segregating populations, all lines were assigned to rows to permit a calculation of theoretical gene frequencies. Consequently, the total numbers of individuals were as follows: 24 plants for each parent

EXPLANATION OF PLATE I

Leaves showing different length of the base marginal vein from the point of petiole junction to the first branch vein.

(Top) The longest vein, Heart of Gold

(Bottom left) The intermediate vein, PMR 45

(Bottom right) The shortest vein, PMR 6

PLATE I

and F_1, 96 plants each for BC_1 and BC_2 and 288 plants for each of the F_2 lines. Numbers were reduced slightly by field mice which damaged some germinating seeds. Plants were thinned to one plant per hill at 4 leaf stage. Since 2 to 6 seeds were planted per hill, thinning was practiced to leave the plant on the most eastern and southern sides so that any zygotic elimination could not occur. All genetic lines from each cross were randomized within each of 4 replications. The crosses from $P_1 \times P_5$ originally had the same number of replications as all others but were reduced to only one replication by powdery mildew since both parents were susceptible.

All experimental plants were exposed to natural field infection by the fungus since they were planted in an area which had been in muskmelons for several years that were infected with alternaria.

Collection and Recording of Data

The degree of resistance of an individual to the pathogen was measured by the number of disease lesions present on leaves and were classified into four different groups based on lesion size. Readings were taken on the right half of three sample leaves per plant. The infection rating of an individual was made by averaging the total number of disease lesions of the three leaves sampled. However, use of the lesion size was discontinued later because it was found that there was a high positive correlation between the number and size of the lesions.

Seven different sampling methods were evaluated in an effort to find a reliable one for measuring the genetics of resistance to

alternaria leaf spot. A field row of P_{2}, consisting of 6 plants was divided into 480 six-inch squares (Plate II) and eighteen leaves were sampled (Table 3). A total of 54 leaves were sampled in each of the methods from the 3 replications. Thus each pooled sample was used to compare the seven different sampling methods. The node sampling method (Method 7) was selected as a standard and was used throughout the study.

Measurement of vein length was made on both sides of the three sampled leaves in order that error sampling variance would not interfere with measurements of correlation of resistance and vein length. The average vein length of the three sample leaves was used to represent an individual plant. In order to reduce experimental error due to variation of sampling time, all individuals (774 leaves) within a block were sampled on the same day and the four blocks of a cross were observed on each of four successive days.

Table 3. Seven different leaf sampling methods.

Method	Explanation
1	Random sampling of the entire area.
2	Random sampling from the middle 1/2 of the area lengthwise.
3	Random sampling from the middle 1/3 of the area lengthwise.
4	Random sampling from the middle 1/6 of the area lengthwise.
5*	Random sampling 3 leaves from a 2 square foot area in the center of a plant.
6*	Random sampling of 3 leaves from a 1 square foot area in center of a plant.
7*	Sampling 3 leaves from the 7th, 8th and 9th nodes of the main vine of each plant.

* Six plants provide the same total number.

EXPLANATION OF PLATE II

(Top) The total sampling area.

(Bottom) Close-up of sampling area.

PLATE II

Statistical and Genetical Analyses

Analyses of variance were made for all experimental data relating to resistance and vein length from 1963 and 1964 seasons. Comparisons of difference between two mean values were made by the t-test (14, 17). Levels used for significance were 1 and 5 per cent for both F- and t-values.

Linear regression coefficients for the two characters among the parental varieties studied in 1963 were determined by the standard method (17). In order to make a valid determination of the degree of linkage in the F_2 generation, a special t-test for comparing regression coefficients of the parents pooled with those of the F_2 was developed by the following equation (14):

$$t = (b_1 - b_2)/s_{b_1 - b_2}$$

where $$s^2_{b_1 - b_2} = s^2_E \left[\frac{1}{\sum_{i=1}^{n_1} (X_{1i} - \bar{X}_1)^2} + \frac{1}{\sum_{j=1}^{n_2} (X_{2j} - \bar{X}_2)^2} \right]$$

and $$s^2_E = \frac{\sum_{i=1}^{n_1} (Y_{1i} - \hat{Y}_{1i})^2 + \sum_{i=1}^{n_2} (Y_{2j} - \hat{Y}_{2j})^2}{n_1 + n_2 - 4}$$

where b_1 is a regression coefficient of the pooled parental population, b_2 is that of the F_2 population, and n_1 and n_2 are the population size, respectively. The t-value has $n_1 + n_2 - 4$ degrees of freedom. This method may be used for testing the degree of linkage in the F_2 generation on the conditions that the gene action involved in two characters is additive with incomplete dominance. If the conditions are accepted,

the degree of linkage in the F_2 generation will be determined by finding a region of rejection, α , where the calculated t-value meets the closest t-value in the table.

Correlation coefficient analyses for resistance and vein length were made from data of each parental variety in 1963 and from the pooled parental variety and F_2 populations from each cross in 1964.

A simple linear regression equation was derived from the mean resistance and vein length values of the parents.

To afford valid comparisons among the seven different leaf sampling methods, the coefficient of variation was calculated as follows:

$$CV = s / \bar{x}.$$

In percentage form this becomes

$$100\ CV = 100(s / \bar{x}) \text{ per cent (14).}$$

where s is a standard error and $\bar{x}$ the mean value for the population.

The additiveness for genic effects was first tested by the original mean measurements from all lines of a cross. If the effects of a number of such genes are additive on the average the mean measurements from the lines will be approximately the same as the expected mean measurements as follows (13): in the absence of certain extreme forms of interactions, $M = 1/2(P_1 + P_2)$, $BC_1 = 1/2(P_1 + F_1)$, $BC_2 = 1/2(P_2 + F_1)$ and $F_2 = 1/4(P_1 + P_2 + 2F_1)$. Where M is mid-parent, BC_1 and BC_2 are the first backcross generations from crosses of the F_1 to parents, or of BC_1 from $F_1 \times P_1$, or reciprocally and BC_2 from $F_1 \times P_2$ or reciprocally.

When original mean measurements of a character for lines of crosses were not near the expected, data were transformed to other scales in

order to measure the additive genic effects. For example, Y' = $\log_{10}$ Y or Y' = (Y + $\log_{10}$ Y)/2, where Y was the original measurement and Y' the new measurement by transformation of scaling.

After vein length measurements of lines from two crosses were transformed to the full or half logarithmic scales estimation of heritability of resistance and vein length was made by the following formula developed by Lush (12):

$$h^2 = \frac{\sigma_G^2}{\sigma_P^2} \quad \text{where} \quad \sigma_E^2 = \frac{\sigma_{P_1}^2 + \sigma_{P_2}^2 + \sigma_{F_1}^2}{3} \quad \text{and}$$

$$\sigma_P^2 = \sigma_G^2 + \sigma_E^2 \qquad \text{Hence,} \quad h^2 = \frac{\sigma_G^2}{\sigma_G^2 + \sigma_E^2}$$

h^2 is the estimated heritability; σ_E^2 the environmental variance; σ_G^2 the genic variance; and σ_P^2 the phenotypic variance.

An estimation of the number of genes involved in each character and cross was made by the following formula:

$$r = \frac{D^2}{8(\sigma_{F_2}^2 - \sigma_{F_1}^2)}$$

where r denotes the estimated number of genes involved and D is the difference between the two parent means from the cross.

EXPERIMENTAL RESULTS

Sampling for Resistance

Data in Table 4 show that there is a parallel between size of the sampling area and the coefficient of variation where the node sampling

method (Method 7) was employed. A good CV percentage range for any biological experiment is 10-20 per cent. Methods 5 and 6, where 1 and 2 square feet were sampled, respectively, appear to be permissible.

Table 4. Coefficients of variation for comparing sampling methods.

Sampling method	Standard error	Mean value	CV (%)
1	29.5	114	25.9
2	26.1	118	22.1
3	24.2	115	21.0
4	29.9	137	21.8
5	26.4	132	20.0
6	30.4	161	18.9
7	28.7	166	17.3

Differences Among Parents and the F_1s

Four F_1s were compared with the parent varieties for degree of resistance to alternaria in 1963.

Analysis of variance of the number of the disease lesions is shown in Table 5. Significant differences at the 1 per cent level were found for both genetic lines and replications. Table 5 includes the variations of mean values and the standard errors for resistance and marginal vein length of all lines.

In Table 6, means of the number of disease lesions show P_5 and P_1 to be the most resistant, P_4 and P_3 intermediate, and P_2 highly susceptible. Means of all four F_1s are distributed between the respective

Table 5. Analysis of variance of the number of the disease lesions among five varieties and the F_1s in 1963.

Source	d.f.	S. S.	M. S.
Lines	8	59651	7456**
Blocks	3	2330	777**
Error	24	3809	159
Total	35	65790	-

**Indicates significance at the 1 per cent level.

Table 6. Mean values and standard errors for the number of the disease lesions and vein length for parents and F_1 progeny.

Lines	No. of disease lesions $\bar{X} \pm s$	Vein length $\bar{X} \pm s$
P_1	18.6 ± 17.9	27.6 ± 5.2
P_2	156.0 ± 46.4	11.9 ± 1.6
F_1 (P_1 x P_2)	56.5 ± 38.0	18.0 ± 2.9
P_3	47.5 ± 30.8	16.1 ± 1.8
F_1 (P_1 x P_3)	38.8 ± 26.6	22.1 ± 2.3
P_4	46.3 ± 25.7	12.6 ± 1.6
F_1 (P_1 x P_4)	39.8 ± 28.0	18.4 ± 2.3
P_5	13.8 ± 10.1	27.2 ± 3.3
F_1 (P_1 x P_5)	16.8 ± 14.0	33.6 ± 3.6

* 1964 data.
$\bar{X}$ is mean value; and s is standard error.

parents in every case and standard errors for all lines are large. By comparing differences in number of disease lesions between parents, between female parent and F_1, and between F_1 and male parent in each cross by the t-test all differences between P_1 and P_2, P_3, P_4 and P_5 are highly significant except for P_1 and P_5. Differences between P_1 and the three F_1s are nonsignificant. However, there was a significant difference at the 5 per cent level between P_1 and the F_1 of $P_1 \times P_2$. Differences between all F_1s are similar to the immediate above except that the change in significance is from 5 to the 1 per cent level in the same cross (Table 7). In other words, the test indicates that all F_1s may be localized approximately around the arithmetic means, though the magnitudes toward either female or male parent are different from one cross to another. It is also shown in Table 6 that the leaves of P_1 and P_5 have longer veins than those of the other three varieties. The three F_1s of $P_1 \times P_2$, $P_1 \times P_3$, and $P_1 \times P_4$ are similarly distributed about the arithmetic means between the parents, with exception of the F_1 ($P_1 \times P_5$), which does not fall midway between the parents apparently due to some degree of dominance. The standard errors for vein lengths are smaller than those for the number of disease lesions.

The linear relation was calculated from the mean values of 5 parent varieties by a regression equation using the number of disease lesions and vein length measurements (Table 6).

The degree and direction of correlation were determined by the following equation:

$$Y = -0.0768X + 22.21$$

where Y is the estimated vein length and X the number of disease lesions.

The linear correlation and the regression line are shown in Fig. 1. A correlation coefficient, r = -0.6462, was calculated from the data. The above data suggest a negative relationship between the two characters in the five parental varieties.

The Inheritance of Resistance to Alternaria Leaf Spot

$P_1 \times P_2$ Cross. This cross was composed of plantings of P_1, P_2, F_1, F_2, BC_1 and BC_2. It can be seen in Table 8 from the analysis of variance of the data that differences due to lines are significant at the one per cent level and that those among blocks are significant at the 5 per cent level. Frequency distribution of the number of disease lesions is shown in Fig. 2. The F_1 mean is closer to P_1 than to P_2 which agrees with data in Table 6, whereas, the F_2 mean is near the arithmetic mean (87.9) between the parents. It may be concluded from the data that heterosis is present in the F_1 and is decreased in the F_2. The BC_1 mean deviated slightly from the expected arithmetic mean. Results indicate that the heterotic effect is mainly caused by P_1. The F_1 variance is approximately that of the expected arithmetic mean between the parents while the F_2 variance is high. The F_2 population covered the entire range of the parents, indicating a segregation of genes which control the number of disease lesions. The BC_1 and BC_2 variances are considerably high but resistance is genetically controlled in P_1 and P_2.

The estimated number of the incompletely dominant genes (r) involved in this cross is 1.964 or for practical purposes 2. The per cent heritability in this cross is 62.5.

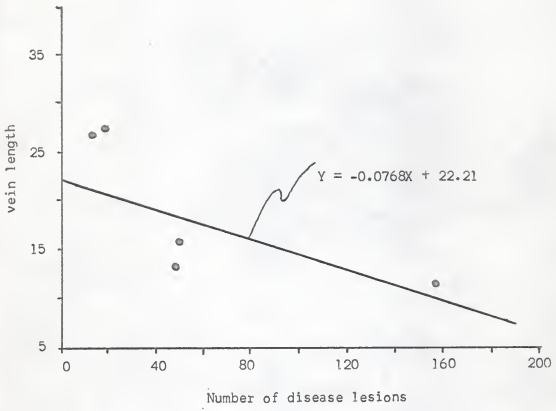


Fig. 1. Correlation diagram of the number of disease lesions and vein length of the five parental varieties.

Table 7. The t-values for differences in the number of disease lesions between parents, female parent and F_1, and F_1 and male parent.

Between parents	t-value	Between female parent and F_1	t-value	Between F_1 and male parent	t-value
P_1:P_2	10.913**	P_1:F_1 (P_1 x P_2)	3.304*	F_1:P_2	5.958**
P_1:P_3	4.203**	P_1:F_1 (P_1 x P_3)	1.533	F_1:P_3	0.906
P_1:P_4	4.712**	P_1:F_1 (P_1 x P_4)	2.068	F_1:P_4	0.570
P_1:P_5	1.918	P_1:F_1 (P_1 x P_5)	0.354	F_1:P_5	0.968

** significant at the 1 per cent level; $t_{.01,6} = 3.707$.

* significant at the 5 per cent level; $t_{.05,6} = 2.447$.

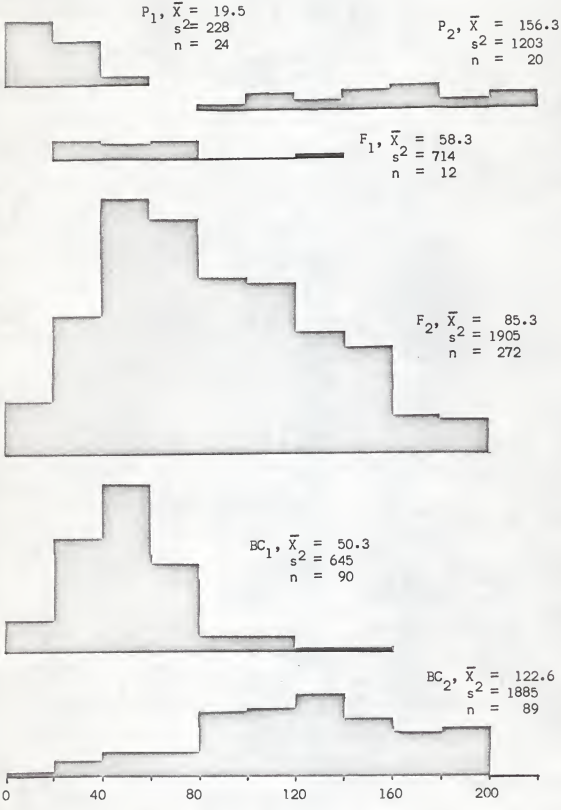


Fig. 2. Frequency of distribution of the numbers of disease lesions on leaves of six lines in the P_1 x P_2 cross.

Table 8. Analysis of variance of the number of the disease lesions among the six lines of $P_1 \times P_2$ cross in 1964.

Source	d.f.	S. S.	M. S.
Lines	5	51437	10287**
Blocks	3	1638	546*
Error	15	2380	159
Total	23	55455	

**Indicates the significance at 1 per cent level.
*Indicates the significance at 5 per cent level.

$P_1 \times P_3$ Cross. The lines of this cross are P_1, P_3, F_1 and F_2. Analysis of variance of the data shows both differences due to lines and blocks as being significant at the 1 per cent level, but with the former showing a higher significance. Frequency distribution of the number of disease lesions is shown in Fig. 3. A similar tendency of incomplete dominance in the F_1 can be observed here, too. However, the F_1 mean is much closer to P_3 than to P_1. Dominance is toward the larger number of the disease lesions; moreover, the magnitudes between the P_1 and the F_1 or between the F_1 and P_3 are different from the former cross due to the shortened genetic range. The F_2 mean is likewise contiguous to the expected arithmetic mean. The P_1 variance was increased from that in the former cross due to the lag of time of sampling. However, the variance which normally is about the mean between the parents in quantitative inheritance studies is smaller than that of either of the parents. Similarly, the F_2 variance did not fully overlap the whole range of the parents.

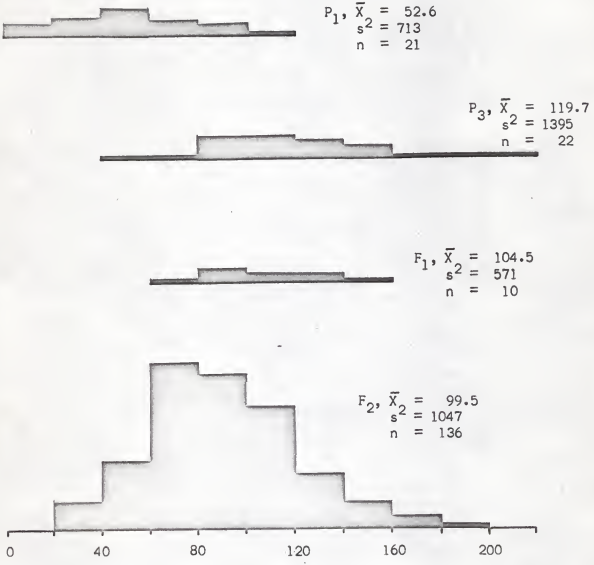


Fig. 3. Frequency of distribution of the number of disease lesions on leaves of the four lines in the $P_1 \times P_3$ cross.

The difference in number of gene pairs between the parental varieties is estimated as: r = 1.182. It can be regsrded as a difference of one pair of the genes governing resistance between the parents. The per cent heritability is 14.7.

Table 9. Analysis of variance of the number of the disease lesions among the four lines of $P_1 \times P_3$ cross in 1964.

Source	d.f.	S. S.	M. S.
Lines	3	9863	3288**
Blocks	3	2075	692**
Error	9	730	81
Totsl	15	12668	-

** Indicstes the significance at 1 per cent level.

$P_1 \times P_4$ Cross. This cross consisted of P_1, P_4, F_1 and F_2 generations of the parents. Analysis of varisnce of the data is presented in Tsble 10. It is appsrent from the dsta that the parentsl influence on expression of resistance is significant st the 1 per cent level whereas the influence of blocks is nonsignificant. Frequency of distribution of the number of disease lesions is shown in Fig. 4. The F_1 mean of this cross also shows a psrtial dominance influence from P_4. This tendency wss detected in a previous experiment in 1963. The F_2 mesn is closer to the expected arithmetic mean than to thst of the F_1 although some dominance is still evident in the populstion. The F_1 varisnce is smaller thsn for the parents. The F_2 vsriance is high and individuals in the

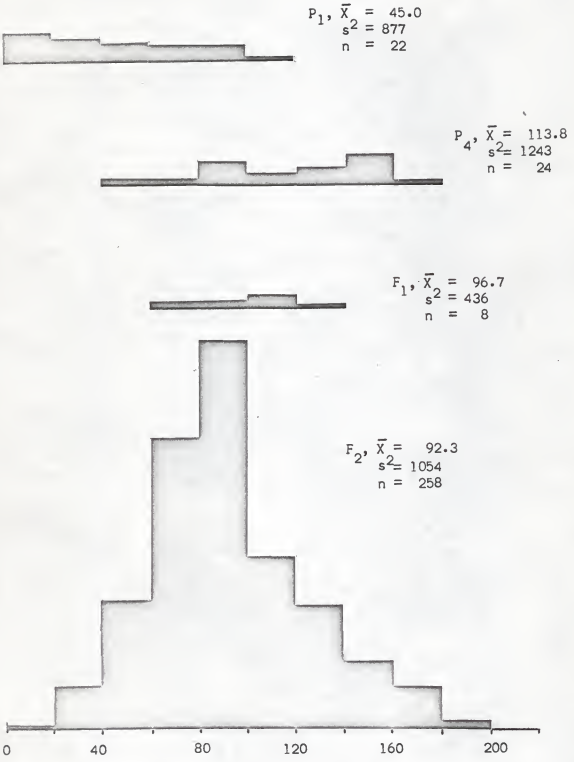


Fig. 4. Frequency of distribution of the numbers of disease lesions on leaves of plants of the four lines in $P_1 \times P_4$ cross.

population cover the entire range of the parents which indicates a segregation of genes governing resistance.

The estimated difference in number of gene pairs from this cross is r = 0.971, or one pair of genes. The per cent heritability in this cross was estimated as 19.2.

$P_1 \times P_5$ Cross. Lines of this cross consisted of P_1, P_5, F_1 and F_2. Frequency of distribution of the number of disease lesions is presented in Fig. 5. The order of means of the P_1 and P_5 is inverted from that in the 1963 experiment (Table 5), but the difference is also nonsignificant in this experiment. The F_1 mean is equal to that of the F_2. The F_1 variance is smaller than that of either of the parents. Generally, all lines in this cross are closely localized with nearly the same means and variances. The estimated number of gene pairs, r, is 0.264. Its nearest integer is 0. The estimated per cent heritability is 15.4.

Table 10. Analysis of variance of the number of the disease lesions among the four lines of $P_1 \times P_4$ in 1964.

Source	d.f.	S. S.	M. S.
Lines	3	12362	4121**
Blocks	3	2283	761
Error	9	1963	218
Total	15	16608	-

** Indicates significance at 1 per cent level.

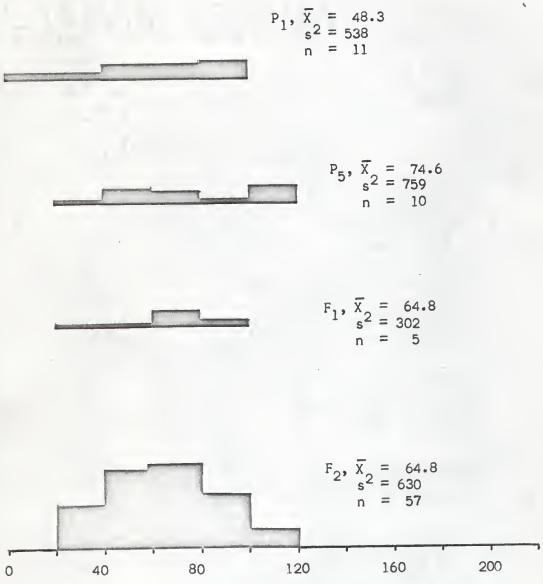


Fig. 5. Frequency of distribution of the number of disease lesions of the four lines in $P_1 \times P_5$ cross.

Inheritance of Vein Length

Results of the analyses of variance of data are shown in Tables 11, 12 and 13. It is clear from data in the tables that the lines are highly significant at 1 per cent level in all crosses analyzed, while blocks are all nonsignificant. Mean values and standard errors for the lines of four crosses were first calculated from the original data. However, it was found that the expected means of F_1, F_2, BC_1 and BC_2 of two crosses, $P_1 \times P_2$ and $P_1 \times P_3$ all fell near the expected geometric rather than arithmetic means. In other words, the gene action involved in vein length of plants from the two crosses is geometric or multiplicative. The original data from plants of the cross $P_1 \times P_2$ were transformed to a common logarithmic scale and that of the cross $P_1 \times P_3$ were transformed to the scale, $Y = (Y + \log_{10} Y)/2$, whereas the data from the other two crosses, $P_1 \times P_4$ and $P_1 \times P_5$ were not transformed because of the presence of additive gene effects (Table 14). The P_1 mean is always larger than those of the four male parents. The F_1 and F_2 means of all crosses fall closely to the expected means except for that of the F_2 of the cross $P_1 \times P_3$. BC_1 and BC_2 means of the $P_1 \times P_2$ cross are also located close to the expected means. The F_2 variances indicate a segregation of genes contributing to vein length. The BC_1 and BC_2 variances of the $P_1 \times P_2$ cross are similarly larger than the F_1s.

The number of gene pairs involved in the four crosses were estimated and the results are shown in Table 15. The larger the number of gene pairs, the higher the heritability value, except that the number of estimated major gene pairs is zero, that is, in the case of polygenes.

Table 11. Analysis of variance of marginal vein length among the six lines of $P_1 \times P_2$ cross in 1964.

Source	d.f.	S. S.	M. S.
Lines	5	530	106**
Blocks	3	25	8.3
Error	15	43	2.9
Total	23	598	-

** Indicates significance at 1 per cent level.

Table 12. Analysis of variance of marginal vein length among the four lines of $P_1 \times P_3$ cross in 1964.

Source	d.f.	S. S.	M. S.
Lines	3	426	142**
Blocks	3	2	0.667
Error	9	12	1.333
Total	15	440	-

** Indicates significance at 1 per cent level.

Table 13. Analysis of variance of marginal vein length among the four lines of $P_1 \times P_4$ cross in 1964.

Source	d.f.	S. S.	M. S.
Lines	3	337	112.3**
Blocks	3	16	5.33
Error	9	19	2.11
Total	15	372	

** Indicates significance at 1 per cent level.

Table 14. Mean values and standard errors for vein length for lines of the four crosses.

Cross	Mean values and standard errors					
	Female parent	Male parent	F_1	F_2	BC_1	BC_2
$P_1 \times P_2$ [(a)]	4.366 ± 1.045	0.797 ± 0.526	25.360 ± 0.742	2.532 ± 1.046	3.393 ± 0.969	1.878 ± 0.933
$P_1 \times P_3$ [(b)]	17.704 ± 2.529	9.108 ± 1.019	13.401 ± 1.418	12,517 ± 2.979	-	-
$P_1 \times P_4$	25.3 ± 3.48	12.6 ± 1.64	18.4 ± 2.27	20.4 ± 4.80	-	-
$P_1 \times P_5$	35.5 ± 4.32	27.1 ± 3.18	33.6 ± 5.23	28.0 ± 4.73	-	-

(a) Stands for transformation of scaling to $\log_{10} Y$.

(b) Stands for transformation of scaling to $(Y + \log_{10} Y)/2$.

Table 15. Genetic range, estimated number of gene pairs and per cent heritability for the four crosses.

Cross	Genetic range	Estimated number of genes		Per cent heritability
		Original	Transformed	
$P_1 \times P_2$	15.0	2.604	2.927	41.6
$P_1 \times P_3$	14.5	2.263	1.993	44.7
$P_1 \times P_4$	12.7	1.118	-	63.0
$P_1 \times P_5$	3.4	0.321	-	15.6

Linkage Relationship Between Resistance and the Vein Length

The distribution of individuals of each P_1, P_2 and F_1 from the $P_1 \times P_2$ cross is shown in Fig. 6. It can be observed that the P_1 individuals are distributed in a standing oval with a mean number of disease lesions of 19.5, and vein length 26.9 mm. The P_2 individuals are distributed in a flat plane with means of 156.3 and 12.3 mm, respectively. The F_1 individuals are distributed between the parental areas in an intermediate shaped area with means of 58.3 and 18.2 mm, respectively, indicating some dominance toward resistance and shorter vein length. In Fig. 7 the distribution of F_2 progeny from the cross is presented. It can be observed that the F_2 population displayed a somewhat negative relationship between the two characters in the parental populations (Fig. 6). The population expanded in distribution area toward greater resistance and shorter vein length and at the same time extended toward greater susceptibility and longer veins indicating a recombination of genes which offset the two characters. The BC_1 progeny are distributed in the summed area of the P_1 and F_1 and the BC_2 progeny in that of the F_1 and P_2 (Fig. 8).

From the two crosses, $P_1 \times P_3$ and $P_1 \times P_4$, data in Figs. 9 and 11 show that the distribution of P_1, P_2 and F_1 individuals are similar to those in the $P_1 \times P_2$ cross. However, the genetic ranges for resistance are much reduced and the progenies of both F_1s are slightly shifted by dominance toward susceptibility and longer veins. The difference of progeny distribution between $P_1 \times P_3$ and $P_1 \times P_4$ crosses is that vein length of plants from the former cross is shorter than that of the latter. Subsequently, shape

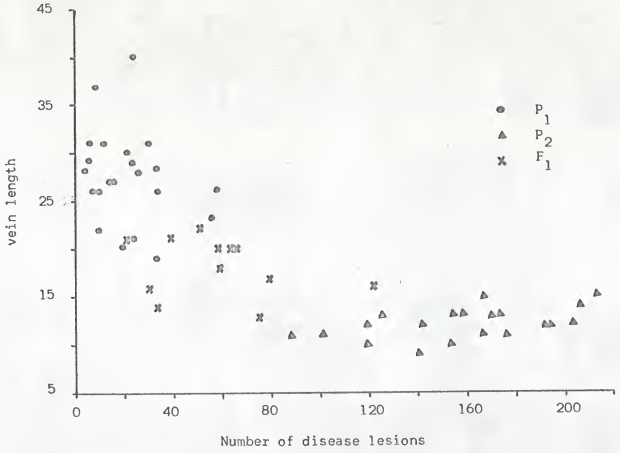


Fig. 6. Correlation diagram of the number of disease lesions and vein length of P_1, P_2 and F_1 lines of the $P_1 \times P_2$ cross.

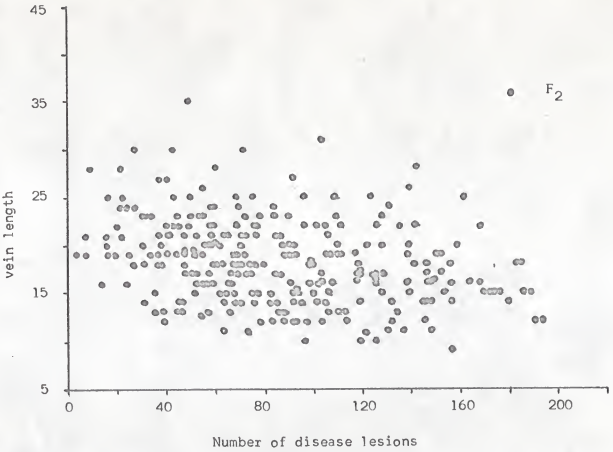


Fig. 7. Correlation diagram of the number of disease lesions and vein length of F_2 line of the $P_1 \times P_2$ cross.

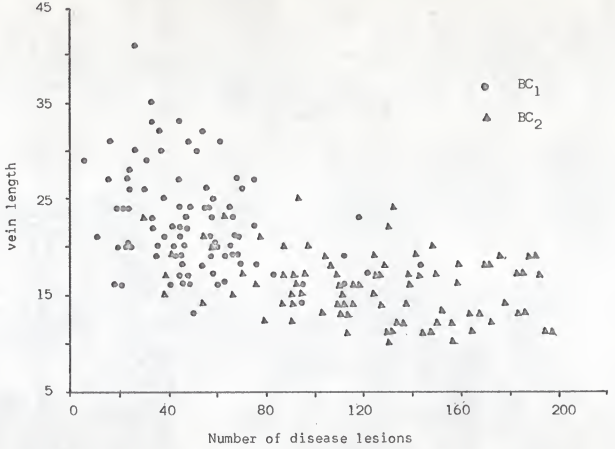


Fig. 8. Correlation diagram of the number of disease lesions and vein length of BC_1 and BC_2 lines of the $P_1 \times P_2$ cross.

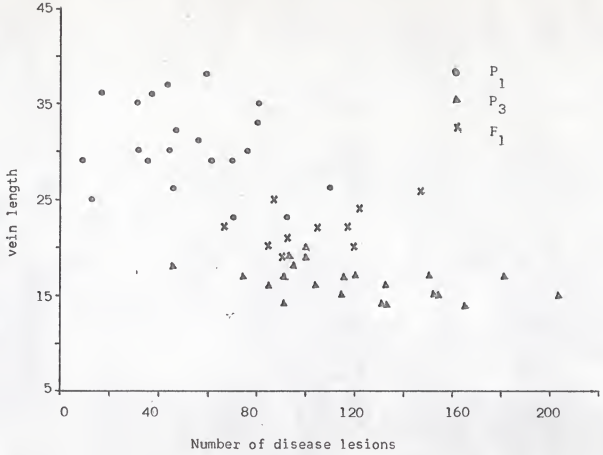


Fig. 9. Correlation diagram of the number of disease lesions and vein length of P_1, P_3 and F_1 lines of the $P_1 \times P_3$ cross.

of the distribution area of the F_2 progeny is restricted by the genetic ranges of plants which have different numbers of incompletely dominant genes. However, in the two recombination areas the F_2 individuals are more widely distributed than those of the $P_1 \times P_2$ cross (Figs. 10 and 11).

The distributions of P_1, P_5 and F_1 individuals from the $P_1 \times P_5$ cross are shown in Fig. 13. Individuals from the lines are distributed in a similar fashion to those in the three previous crosses. The F_1 shows a slight dominance for resistance and longer veins but about two thirds of the areas are overlapped showing less difference in the magnitude for the two characters. Distribution of the F_2 progeny from this cross is nearly the same as for those of the early generations, but the size of the F_2 area is somewhat increased (Fig. 14).

The correlation figures presented above explain the relationship between the two characters to some extent, but to evaluate the phenomena statistically, correlation analyses were made from the 1964 data, too.

Results of correlation coefficient analyses of the four crosses are shown in Table 16.

Table 16. Correlation coefficients of the pooled parents and the F_2 progeny.

Cross	r for parents*	r for F_2*
$P_1 \times P_2$	-0.8177	-0.3260
$P_1 \times P_3$	-0.7364	-0.1844
$P_1 \times P_4$	-0.6896	-0.1775
$P_1 \times P_5$	0.0433	0.2849

* r = correlation coefficient.

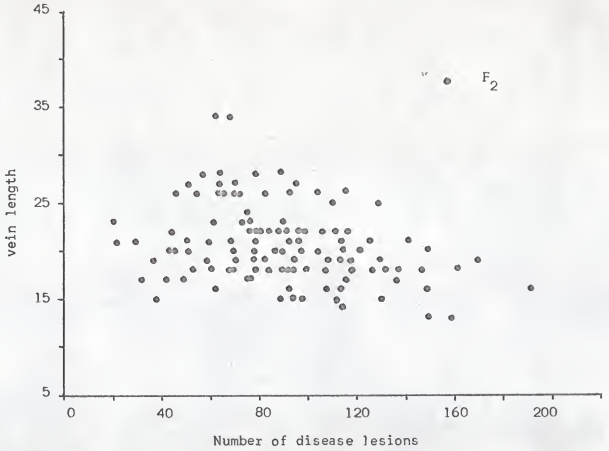


Fig. 10. Correlation diagram of the number of disease lesions and vein length of F_2 line of the $P_1 \times P_3$ cross.

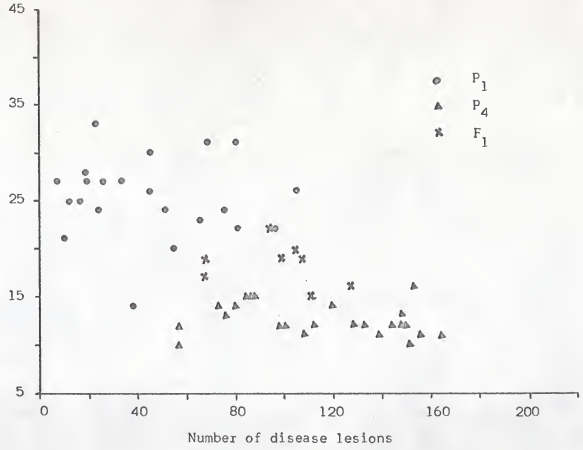


Fig. 11. Correlation diagram of the number of disease lesions and vein length of P_1, P_4 and F_1 lines of the $P_1 \times P_4$ cross.

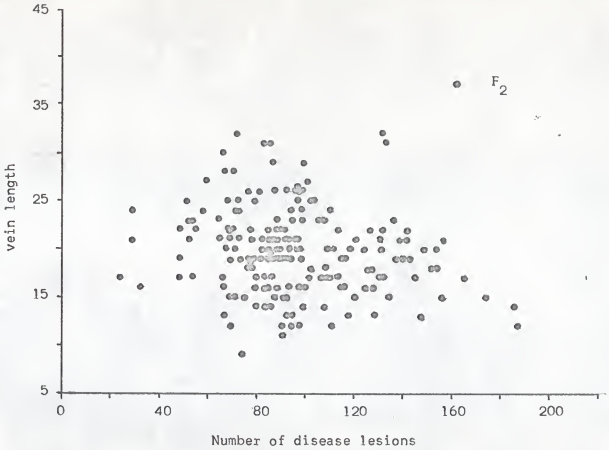


Fig. 12. Correlation diagram of the number of disease lesions and vein length of F_2 line of the $P_1 \times P_4$ cross.

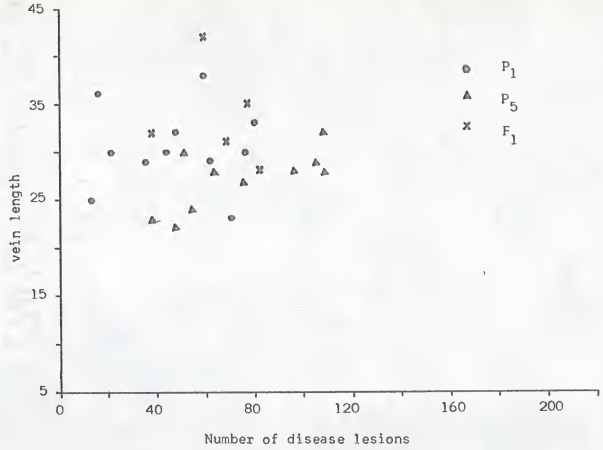


Fig. 13. Correlation diagram of the number of disease lesions and vein length of P_1, P_5 and F_1 lines of the $P_1 \times P_5$ cross.

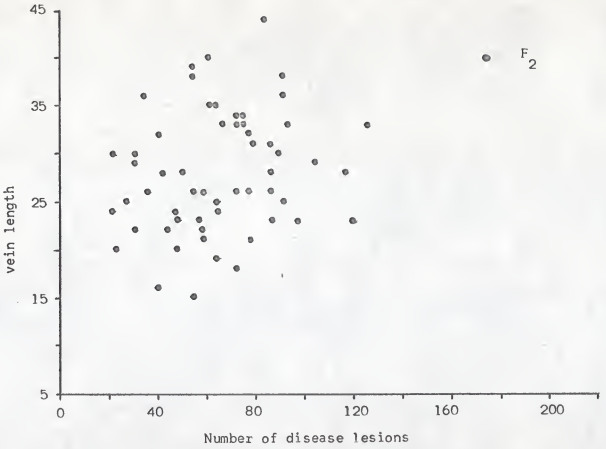


Fig. 14. Correlation diagram of the number of disease lesions and vein length of F_2 line of the $P_1 \times P_5$ cross.

It can be seen from data in Table 16 that the degree of correlation of the F_2s from the first three crosses was reduced to less than half of those of their parents, whereas the results of the cross $P_1 \times P_5$ were reversed. Among all crosses, $P_1 \times P_2$ revealed the strongest correlation in the parentals as well as in the F_2 populations. Degree of reduction of the relationship in the first three crosses is the most remarkable in the $P_1 \times P_3$ cross, the next in the $P_1 \times P_4$ cross, and the smallest in the $P_1 \times P_2$ cross. In the $P_1 \times P_5$ cross the parental correlation coefficient is nearly equal to zero on the positive side, but the F_2s increased. It seemed that the increased degree of relationship in the F_2 from the $P_1 \times P_5$ cross might be due to the small sample size.

Table 17 shows results of the special t-test for detection of linkage.

Table 17. Regression coefficients for the parents and F_2s pooled.

Cross	b for parents*	b for F_2*	t-value	
$P_1 \times P_2$	-0.0934	-0.0330	$t_{312} = 4.6938$	$t_{.001,\infty} = 3.2905$
$P_1 \times P_3$	-0.1247	-0.0234	$t_{175} = 4.5102$	$t_{.001,\infty} = 3.2905$
$P_1 \times P_4$	-0.1038	-0.0262	$t_{300} = 4.0312$	$t_{.001,\infty} = 3.2905$
$P_1 \times P_5$	0.0633	0.0534	$t_{74} = 0.2240$	$t_{.82,60} = 0.2284$

* b = regression coefficient.

It is apparent from data in the table that t-values of the first three crosses are greater than the table values at $\alpha = 0.001$. In other words, the probability that the correlation between resistance and vein length

in the pooled parent data and that between these traits in the F_2 data are samples from the same population parameter is less than one in a thousand. Therefore, there is no linkage of the genes for the characters. On the contrary, in the cross $P_1 \times P_5$ the calculated t-value was found to be .82 or 82 per cent of the total number of genes differentiating the parental varieties for the two characters are linked. Since no major genes were previously estimated for either resistance or vein length in this cross all genes must be polygenes.

DISCUSSION

Data summaries from six experiments reveal a number of consistent significant host reactions and characters which will support definite genetic conclusions. Other results are less reliable but permit speculation on the genetic inheritance of alternaria leaf spot resistance. In addition, other genetic data reveal interactions and correlations between marginal vein length, sampling techniques and resistance.

All F_1 and F_2 means are approximately intermediate between the parents in both resistance and marginal vein length. However, gene action is different for resistance which is governed by arithmetic or additive gene effects than for vein length which is governed by geometric or multiplicative gene effects. All the F_2, BC_1 and BC_2 means for resistance are close to the expected arithmetic means. Thus it appears that resistance is controlled by incompletely dominant genes. This agrees with findings of Gough and Williams (7) in their studies of the durum wheat varieties Acme and Mindum where resistance to stem rust was conditioned by at least three incompletely dominant genes.

Marginal vein length means of the F_1, F_2 and BC_1 and BC_2 are closer to the geometric than arithmetic means although fitness is within the error variances. By full or half transformation of scaling to a common logarithm of the two crosses $P_1 \times P_2$ and $P_1 \times P_3$ gave satisfactory means from the segregating populations except for the F_2 mean from the $P_1 \times P_3$ cross. Cause for this variation could be due to sampling error, gametic or zygotic elimination, or coupling. In other words, a survey of vein length data indicates gene substitution is all geometrical rather than arithmetic. Therefore, the genes contributing to vein length would also be classified as incompletely dominant. The estimated number of major gene pairs causing differences in resistance vary from zero to two among progeny of the crosses. Similar results have been reported for other crops.

It was reported that resistance to flax rust was conditioned by a three dominant gene series (6) and resistance to wheat stem rust in durum wheat was reported to be by at least three dominant genes and at least three incompletely dominant genes, and in radish a single dominant gene.

Data from this study indicate that there may be a direct correlation between the genetic range and the number of genes involved. In one of the crosses, $P_1 \times P_5$, the estimated number of gene pair differences was 0.264 and the genetic range in number of lesions was 26.3. There appears to be no major gene differences between the parents. It may be assumed that alleles control differences in resistance and that probably linked polygenes contribute to the small genetic range of 26.3. Other evidence

of the existence of polygenes is that all parental varieties studied possess a wide variation in number of disease lesions. Parental variations were not included in the formula for estimation of the number of the major genes. This alone makes it difficult to give a definite genetic explanation of resistance (20). Gene analysis of results from this study of resistance agrees with the assumption given by Tomiyama (18) that there are two different genetic types of resistance. One where major genes contributed to a higher level of resistance and the other where polygenes give unstable low levels of resistance. The resistance of radish to white rust was governed by a single dominant gene and minor genes which modify it (19). Results from the study of alternaria resistance indicate that a pair of genes control an average of 68 lesions. However, crosses were not made between P_2, P_3, P_4 and P_5, hence the gene analogs are unknown.

The estimated numbers of gene pairs which control marginal vein length differed between crosses and required different scales (Table 14). Full and half transformation to a common logarithm from crosses $P_1 \times P_2$ and P_1 and P_3 fit more closely to the expected ones. Progeny means from the other two crosses $P_1 \times P_4$ and $P_1 \times P_5$ are close to the arithmetic means due to none or one major gene pair difference. There is an interesting relation among the crosses in that apparently when the number of gene pairs increases, the dosage per gene pair decreases. However, data from this study are inadequate to provide definite proof of this conclusion. Marginal vein length appears to be partially under the control of polygenes, but genetical evidence is inadequate for proof of the observation.

Linkage between the genes contributing to resistance and vein length was studied. The study of linkage between genes contributing to quantitative and qualitative characters is important from the standpoint of plant breeding. This concept has actually been established by Sax (16), Rasmusson (15) and Currence (5). They reported instances where qualitative characters were governed by completely dominant genes; therefore the F_2 and F_3, or testcross generations were needed to determine gene linkage. Mather (13) proposed a method for testing linkage in quantitative inheritance of a character by using F_2 and F_3 or BC_1 and BC_2 generations. However, a completely satisfactory method for linkage testing in quantitative inheritance of two characters has not been developed. An attempt to test linkage by using the parents and F_2 generations was made in this study. Difference between parental and F_2 regression coefficients was compared by a special t-test and the probability of a rejection region in the t-test was calculated as linkage percentage in the F_2 generation. Results show that there is no linkage of genes contributing the resistance and marginal vein length in the three crosses but there was 82 per cent linkage in the cross of $P_1 \times P_5$ (Table 17). It may be interpreted that the major genes which contribute to resistance and vein length are located on different chromosomes and may segregate and recombine, whereas some of the polygenes involved for both characters are closely linked on a chromosome and in the coupling phase. In this case the method for testing gene linkage was used under the assumption that the two characters might be controlled by incompletely dominant genes and polygenes.

Heritability of resistance tends to increase with the number of major gene pairs. However, polygenic effects in the crosses remain

approximately the same. This causes the heritability to decrease when a cross has one gene pair difference. On the contrary, the heritability of vein length is different from resistance. Heritability is apparently inversely proportional to the number of major gene pairs which control vein length. This may be due to the fact that the dosage per gene pair decreases as the number of gene pairs increase.

Resistance to the disease causal organism of alternaria leaf spot in muskmelon plants varies greatly with leaf age. As leaf age increases, resistance decreases. In general, the situation is the reverse in other crops. There are examples reported by Kahn and Libby (10) in rice and by Williams and Pound (19) in radish. In such cases it is difficult to estimate exact gene effects. It has been reported that in muskmelon this seems to be one of the factors (20) that has delayed genetic studies of resistance to alternaria leaf spot.

Sampling of leaves from specific nodes on the vine throughout this study gave the smallest coefficient of variation among the seven different methods. It is believed that the sampling method minimized error variance giving the greatest genetic variance.

SUMMARY AND CONCLUSION

This study was conducted to determine the inheritance of resistance to Alternaria cucumerina (E. & E.) J. A. Elliot, marginal leaf vein length in muskmelon Cucumis melo L., the linkage relationship between genes controlling resistance and vein length.

The study was conducted at the Ashland horticultural farm at Kansas State University during 1963-1964.

From the results of the experiments it is concluded that the widest range of resistance in progeny from the crosses is controlled by two allelic pairs of incompletely dominant genes and polygenes. The resistance largely increases with the number of the major genes whose effects are arithmetic or additive. The existence of polygenes was indirectly detected in this study. The analog of the major genes involved in the crosses is still unknown. The heritability of resistance varies with the number of the major gene pairs and it is low when resistance is controlled by polygenes only.

It was found that the longest marginal vein length on leaves of plants in the crosses is controlled by three allelic pairs of incompletely dominant genes and polygenes. The dosage of a pair of major genes contributing to vein length is inversely correlated to the number of gene pairs. The gene effects declined to geometric rather than arithmetic effects within the error variances. The existence of polygenes was also indirectly detected and the analog of the major genes is still unknown. Heritability decreases as the number of major genes increase, and has a low per cent of polygen control.

It is concluded that there is no linkage relationship between the major genes controlling resistance and those controlling the vein length while there is a strong linkage of some of the polygenes which modify each of the two characters. Therefore, it is clear that vein length is not a visible and dependable marker for resistance.

There is a great variation in the degree of infection by the fungus within an individual muskmelon plant. An experiment was conducted to develop a suitable sampling method for the genetic study. Results indicate that sampling specific nodes on the vine was the best of the seven methods and was used throughout the study.

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THE INHERITANCE OF ALTERNARIA LEAF SPOT RESISTANCE IN MUSKMELON *Cucumis melo* L.

by

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Muskmelon, Cucumis melo L., is the host which is most seriously injured by the fungus Alternaria cucumerina (E. & E.) J. A. Elliot. This disease causes damage by defoliating the vines and thereby causing premature ripening which reduces yield and fruit quality. Control with fungicide sprays may be impracticable because of the rapid growing nature of the vine which requires frequent sprayings.

The inheritance of resistance to alternaria leaf spot in muskmelon has not been previously studied in detail. There are a number of reports indicating that the exact number of genes involved was unknown or undetermined. The breeding behavior of resistance is partially known but reports are inconsistent.

In 1963-1964 six experiments involving the crossing of five commercial varieties of muskmelon were designed to determine the number of genes involved in the resistance to the fungus, the type of gene action, heritability, the linkage of genes contributing to resistance and other visible characters such as marginal vein length and to develop a suitable sampling method.

From results of the inheritance experiments it was found that the widest genetic range from the crosses was governed by two allelic pairs of incompletely dominant genes and polygenes. Resistance largely increases with the number of the major genes whose effects are arithmetic or additive. Heritability varies with the number of major gene pairs and it is low when resistance is controlled by only polygenes.

From the inheritance experiments it was found that the widest genetic range of vein length in the progeny was controlled by three

allelic pairs of incompletely dominant genes and polygenes. The dosage of a major gene pair contributing to vein length was inversely correlated with the number of gene pairs. Gene effects declined to geometric effects. Heritability decreased as the number of major genes increased and is low under the control of the polygenes.

There was no linkage between major genes which control resistance and those which control vein length. However, there was strong linkage of polygenes which modify each of the characters. Therefore, it is clear that vein length is not a dependable phenotypic character for screening plants for resistance.

A suitable sampling method was developed to reduce variability due to the degree of infection of individual plants. It was the node sampling method where leaves from specific nodes were sampled.

A further study is needed to determine the analog of major genes contributing to resistance found in this study.