make possible the programmed production of better proportioned, more desirable plants of these new hybrids.

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The Inheritance of Fusarium Wilt Resistance in Watermelon, *Citrullus lanatus* (Thunb.) Mansf.[1]

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Abstract. The inheritance of Fusarium wilt resistance was studied using 3 varieties of watermelon. 'Summit' and 'Charleston Gray' differed in their level of resistance. 'Summit, carrying a high level of resistance, acted in a completely dominant manner to the susceptible variety 'New Hampshire Midget'. On the other hand, 'New Hampshire Midget' was partially dominant to the moderately resistant variety 'Charleston Gray'. The relationship of 'Summit' to 'Charleston Gray' was not clear, although the data suggested dominance of 'Summit' to 'Charleston Gray'.

Three gene models were postulated, a one-locus multiple-allelic model and 2 two-locus models. The deviations from regression (model adequacy) for the multiple-allelic model and for one of the 2-locus models were nonsignificant as shown by the Chi-square test. The coefficient of determination (R^2) for the multiple-allelic model was .973 with 5 genetic parameters and for the 2-locus model, R^2 = .970 with 4 genetic parameters. Neither model was considered superior to the other.

It was suggested that a variety such as 'Summit', with a high level of resistance, inherited in a completely dominant manner, could be of value in obtaining F_1 hybrids highly resistant to Fusarium wilt, as well as aid in the development of highly resistant open-pollinated varieties.

FUSARIUM wilt of watermelon was first described by Smith in 1899 (10). He considered the Fusarium fungus as one of the most destructive soil-inhabiting "parasites." Since the organism may persist in soils for many years, a lengthy rotation is usually necessary when susceptible varieties are used. Even when land was readily available, it was recognized that resistant varieties would soon be needed.

Resistance to Fusarium wilt was found in the stock citron by Orton (4) and used by him to develop the first wilt-resistant watermelon variety, 'Conqueror' (5).

Numerous varieties carrying resistance to Fusarium wilt have been released since 'Conqueror' in 1911. The level of resistance, however, may differ among varieties. Stevenson (11) tested 32 varieties for resistance in Indiana and placed them in 3 groups. Schenck (8) used 4 groups to categorize varieties tested in Florida.

The inheritance of Fusarium wilt resistance in watermelon has not been adequately studied. Porter (6, 7) considered resistance as a recessive character from the reaction of F_1 hybrids to wilt and from data obtained during the development of the resistant variety 'Klondike R-7'. Walker (12), while studying the effect of temperature on the development of Fusarium wilt in watermelon, observed that the F_1 hybrids between 3 strains of citron and 2 susceptible varieties appeared to be as susceptible as the susceptible parents.

On the other hand, Welch and Melhus (13) found the F_1 hybrid between resistant 'Iowa Belle' and a susceptible parent to be resistant. Bennett (2) studied the inheritance of wilt resistance using a Russian melon (resistant) and 'Early Fordhook' (susceptible) parent. He concluded resistance was due to several factors, some of which were cumulative in effect. Ivanoff and Albritton (3) suggested that resistance of an intermediate to dominant nature may be obtained in an F_1 hybrid if a highly resistant variety is used as a parent, provided that more than one factor for resistance is present and is derived from different origin. No data were given, however.

The objectives of this study were: 1) to determine if the factors for resistance in the variety 'Summit' are different from those in 'Charleston Gray'; and, 2) to determine the mode of inheritance of resistance in the 3 varieties studied.

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Materials and Methods

Three varieties, 'Summit' (S), 'Charleston Gray' (CG) and 'New Hampshire Midget' (N), were used to study the inheritance of Fusarium wilt resistance. Preliminary tests had indicated differential responses among the varieties to inoculation; 'Summit' was highly resistant, 'Charleston Gray' moderately resistant and 'New Hampshire Midget' susceptible. Three families of crosses were made: 'Summit' × 'New Hampshire Midget' (Family 1), 'Charleston Gray' × 'New Hampshire Midget' (Family 2) and 'Summit' × 'Charleston Gray' (Family 3). Five greenhouse tests were conducted during 1966 and 1967. Each test contained the 2 parent varieties, labeled P_1 and P_2; the F_1, the F_2 and the 2 backcross generations, labeled BCP_1 and BCP_2, from each of the 3 families. Plants were grown in 4-inch plastic pots which were placed in glazed pots and, in turn, were immersed in a temperature-controlled water bath maintained at 26°C ± 2°. The experimental design was a randomized complete block containing 4 to 6 replications depending upon the test. Each tank constituted a replication and each pot a treatment.

Inoculum was prepared by using a mixture of 2 pathogenic isolates (F-9D and F-9F) of *Fusarium oxysporum* f. sp. *niveum* (E. F. Sm.) Snyder and Hansen. Cultures were grown in 250 ml of a modified Richards solution (14) in 1-liter Erlenmeyer flasks placed on a rotary shaker for 5 days. The culture liquid was filtered with a Büchner funnel and discarded; the mycelial mat was resuspended in 3 volumes of deionized water and blended for 30 seconds in a Waring Blendor. Each flask produced enough inoculum for 15 four-inch pots.

When the plants were at the 3–4 true-leaf stage, approximately 2½ wk following seeding, roots were wounded by running a sharp knife between the 2 plants in each pot. Fifty ml of mycelial suspension per pot was poured into the furrow. Soil moisture was maintained at a level which promoted rapid vegetative growth. Plants were observed daily and recorded as diseased on the day when distinct wilting occurred.

A disease index, that reflected both the extent and rapidity of disease development, was computed. The highest value of 10 was given to plants diseased on the day the first 'New Hampshire Midget' plant showed symptoms. The index was decreased by one unit for each succeeding day the plant did not show symptoms to a lower limit of zero for any plant not showing symptoms within 10 days of the first diseased plant in 'New Hampshire Midget'.

The disease-index data showed a distinct bimodal distribution with very few readings occurring for X = 2 or 3. Thus, individuals with a disease-score rating of X>2 were classified as "diseased" and the percentage of diseased individuals per entry was recorded. The arcsin transformation on the percentages was used to stabilize the variance. This transformation theoretically gives a constant variance of $\sigma^2 = 0.25/n$ or, using the harmonic mean (n_h) for n, $\sigma^2 = (.02791)$.

Three basic genetic models were postulated and tested against the observed results. In every case each genotype was assumed to have some inherent tendency to develop the disease; this tendency being measured by the proportion of the individuals having a disease score greater than 2. For convenience, this might be referred to as the *propensity* score. In the case of segregating generations, assuming no selection of any kind favoring one genotype over another, the expected propensity scores were calculated as the weighted averages of the propensities for the component genotypes where the weighting was determined by expected segregation ratios for the particular model. The ancestry of the 3 parental lines was such that all were assumed to be homozygous.

Model 1. Multiple-allelic, single-locus model. The 6 possible genotypes for a 3-allele model and their respective genetic values are given in Table 1.

Table 1. Parental and F_1 genotypes and genetic values as defined by Model 1.

Generation	Genotype	Genetic value
Summit	A^1A^1	u
Charleston Gray	A^2A^2	u + $2d_1$
New Hampshire Midget	A^3A^3	u + $2d_2$
Summit × Charleston Gray (F_1)	A^1A^2	u + d_1 + h_1
Summit × New Hampshire Midget (F_1)	A^1A^3	u + d_2 + h_2
Charleston Gray × New Hampshire Midget (F_1)	A^2A^3	u + d_1 + d_2 + h_3

d_1 is the additive effect of replacing an A^1 allele with an A^2 allele, and d_2 is the additive effect of replacing A^1 with the A^3 allele; h_1, h_2 and h_3 are the dominant deviations of A^1A^2, A^1A^3, and A^2A^3 respectively from the averages (midparent) of the corresponding homozygous genotypes. The matrix of coefficients for the generations of Model 1 is shown in Table 2.

Table 2. Matrix of coefficients of genetic parameters for Model 1.

Generation[a]	Genetic parameters					
	u	d_1	d_2	h_1	h_2	h_3
S	1	0	0	0	0	0
CG	1	2	0	0	0	0
N	1	0	2	0	0	0
(S × CG) F_1	1	1	0	1	0	0
(S × N) F_1	1	0	1	0	1	0
(CG × N) F_1	1	1	1	0	0	1
(S × CG) F_2	1	1	0	1/2	0	0
(S × N) F_2	1	0	1	0	1/2	0
(CG × N) F_2	1	1	1	0	0	1/2
(S × CG) × S	1	1/2	0	1/2	0	0
(S × CG) × CG	1	3/2	0	1/2	0	0
(S × N) × S	1	0	1/2	0	1/2	0
(S × N) × N	1	0	3/2	0	1/2	0
(CG × N) × CG	1	3/2	1/2	0	0	1/2
(CG × N) × N	1	1/2	3/2	0	0	1/2

[a]S = Summit, CG = Charleston Gray, N = New Hampshire Midget.

Model 2. Two-locus model with parent 'Summit' having 2 alleles for resistance at both loci, and parent 'Charleston Gray' having alleles for resistance at only 1 locus, *A* (Table 3). The loci were assumed not to interact.

d_1 is the additive effect of replacing *A* with *a;* d_2 is the additive effect of replacing *B* with *b*. The dominance

Table 3. Parental and F_1 genotypes and genetic values as defined by Model 2.

Generation	Genotype	Genetic value
Summit	*AABB*	u
Charleston Gray	*AAbb*	u + $2d_2$
New Hampshire Midget	*aabb*	u + $2d_1$ + $2d_2$
Summit × Charleston Gray (F_1)	*AABb*	u + d_2 + h_2
Summit × New Hampshire Midget (F_1)	*AaBb*	u + d_1 + d_2 + h_1 + h_2
Charleston Gray × New Hampshire Midget (F_1)	*Aabb*	u + d_1 + $2d_2$ + h_1

deviations for the *A* locus and *B* locus are labeled h_1 and h_2 respectively. The genetic values for the remaining 3 genotypes are clear from the definitions.

Since the basic version of this model will be shown to have a significant lack of fit to the observed results, the complete matrix of coefficients is not given.

Model 3. Two-locus model with the 2 resistant parents having genes for resistance at different loci. This version of the 2-locus model postulated that 'Summit' had alleles for resistance at 1 locus, *A,* and 'Charleston Gray' had alleles for resistance at the other locus, *B*. The genetic values for 6 of the 9 genotypes are shown in Table 4.

Table 4. Parental and F_1 genotypes and genetic values as defined by Model 3.

Generation	Genotype	Genetic value
Summit	*AAbb*	$u + 2d_2$
Charleston Gray	*aaBB*	$u + 2d_1$
New Hampshire Midget	*aabb*	$u + 2d_1 + 2d_2$
Summit × Charleston Gray (F_1)	*AaBb*	$u + d_1 + d_2 + h_1 + h_2$
Summit × New Hampshire Midget (F_1)	*Aabb*	$u + d_1 + 2d_2 + h_1$
Charleston Gray × New Hampshire Midget (F_1)	*aaBb*	$u + 2d_1 + d_2 + h_2$

The definition of the parameters is the same as for Model 2. Note that genotype *AABB* with value u is no longer one of the parental genotypes. Table 5 contains the matrix of coefficients for the 15 generations involved in Model 3.

Standard least-squares multiple regression techniques were used for estimating the parameters and testing the adequacy of the models. All analyses and tests were conducted using the arcsin-transformed variables expressed in radians. The coefficient of determination, R^2 = (sum of squares due to regression/corrected entry sum of squares), was used as the measure of goodness of fit of the model to the observed results. All tests of significance were based on the chi-square distribution utilizing the expected error variance of $\sigma^2 = 0.25/n_h = .02791$. (The observed error variance estimated by the "generations × replications" mean square was slightly but not significantly smaller, $s^2 = .024510$ with 56 degrees of freedom.)

Results and Discussion

Tests of heterogeneity of response for the 5 inoculation tests conducted during this study were nonsignificant. Thus, the data from all 5 tests were pooled for testing the genetic models and estimating the genetic parameters.

Preliminary analyses of variance showed the differences among 'generations', with 14 degrees of freedom, to be

Table 5. Matrix of coefficients of genetic parameters for Model 3.

Generation[a]	Genetic parameters				
	u	d_1	d_2	h_1	h_2
S	1	0	2	0	0
CG	1	2	0	0	0
N	1	2	2	0	0
(S × CG) F_1	1	1	1	1	1
(S × N) F_1	1	1	2	1	0
(CG × N) F_1	1	2	1	0	1
(S × CG) F_2	1	1	1	1/2	1/2
(S × N) F_2	1	1	2	1/2	0
(CG × N) F_2	1	2	1	0	1/2
(S × CG) × S	1	1/2	3/2	1/2	1/2
(S × CG) × CG	1	3/2	1/2	1/2	1/2
(S × N) × S	1	1/2	2	1/2	0
(S × N) × N	1	3/2	2	1/2	0
(CG × N) × CG	1	2	1/2	0	1/2
(CG × N) × N	1	2	3/2	0	1/2

[a]S = Summit, CG = Charleston Gray, N = New Hampshire Midget.

Table 6. Genetic models with coefficients of determination (R^2) and tests of deviations from regression (model adequacy).

Model	Description[a]	No. parameters	R^2	Deviations from regression		
				d.f.	Chi-square	Probability (%)
		Model 1				
1a	Arbitrary dominance	5	.973	9	11.24	25–50
1b	Complete dominance of A^1 to A^2 (S, CG)	4	.973	10	11.45	25–50
1c	Complete dominance of A^1 to A^2 (S, CG) Complete dominance of A^1 to A^3 (S, N)	3	.966	11	14.34	10–25
1d	Complete dominance of A^1 to A^2 (S, CG) Complete dominance of A^1 to A^3 (S, N) Alleles additive, A^2–A^3 (CG, N)	2	.956	12	18.59	5–10
1e	Complete dominance of A^1 to A^2 (S, CG) Complete dominance of A^1 to A^3 (S, N) Complete dominance of A^3 to A^2 (N, CG)	2	.940	12	25.25	1– 2.5
		Model 2				
2a	Arbitrary dominance	4	.939	10	25.72	<1
		Model 3				
3a	Arbitrary dominance	4	.970	10	12.58	25–50
3b	Complete dominance of *A* to *a* (S, N)	3	.964	11	14.89	10–25
3c	Complete dominance of *A* to *a* (S, N) *B*-locus additive (CG, N)	2	.926	12	31.31	<1
3d	Complete dominance of *A* to *a* (S, N) Complete dominance of *b* to *B* (N, CG)	2	.601	12	167.96	<1

[a]S = Summit, CG = Charleston Gray, N = New Hampshire Midget.

Table 7. Estimates of genetic parameters, d_i (additive) and h_i (dominant), and Chi-square tests of each equaling zero.

Family[a]	Alleles involved	Genetic parameter	Estimate of parameter	Tests of parameters = 0	
				Chi-square	Probability (%)
		Model 1a			
S × N	A^1, A^3	d_2	.57	170.17	<1
		h_2	−.47	30.71	<1
CG × N	A^2, A^3	$d_3 = d_2 - d_1$	.47	115.70	<1
		h_3	.18	4.34	2.5– 5
S × CG	A^1, A^2	d_1	.10	5.18	1 – 2.5
		h_1	−.14	2.85	5 –10
		Model 1c			
S × N	A^1, A^3	d_2	.57	253.82	<1
		h_2	$-d_2$	—	—
CG × N	A^2, A^3	d_3	.49	131.31	<1
		h_3	.17	4.26	2.5– 5
S × CG	A^1, A^2	d_1	.08	5.38	1 – 2.5
		h_1	$-d_1$	—	—
		Model 3a			
S × N	*A*, *a*	d_1	.54	201.06	<1
		h_1	−.41	39.35	<1
CG × N	*B*, *b*	d_2	.44	134.53	<1
		h_2	.24	13.41	<1
S × CG	All	$d_3 = d_1 - d_2$	.10	4.92	2.5– 5
		$h_3 = h_1 + h_2$	−.17	3.72	5 –10
		Model 3c			
S × N	*A*, *a*	d_1	.49	385.53	<1
		h_1	$-d_1$	—	—
CG × N	*B*, *b*	d_2	.37	118.60	<1
		h_2	.00	—	—
S × CG	All	d_3	.12	15.75	<1
		h_3	$-d_1$,0	—	—

[a]S = Summit, N = New Hampshire Midget, CG = Charleston Gray.

highly significant. The generation mean values ranged from .15 radians (2.3% individuals diseased) to 1.36 radians (95.8% individuals diseased). In all cases, the genetic models studied accounted for highly significant proportions of the 'generations' (entries) sum of squares and, with the exception of Model 3d, the degree of fit was very high, as reflected in the coefficient of determination, R^2, in Table 6.

The adequacy of the various models is indicated by the levels of significance of the deviations from regression as shown by the Chi-square test in Table 6. The most general version of each model is listed as the *a* submodel in each case. The models indicated by successive letters are simplified models obtained by imposing restrictions on the level of dominance. Since the most general version of Model 2 showed highly significant deviations from regression it was construed as being inadequate and dismissed from further examination. The deviations from regression were not significant for Models 1 and 3 until at least 2 restrictions had been imposed. Consequently, both were accepted as plausible genetic models.

Estimates of the individual parameters for Models 1a, 1c, 3a and 3c and tests of significance of their deviation from zero are shown in Table 7. A significant difference from zero of one of the *d* parameters implies that the parental lines carrying the corresponding alleles are genetically different. A significant difference from zero of one of the *h* parameters would indicate some degree of dominance of one allele over the other or, conversely, that the 2 alleles do not act additively. The test for complete dominance of one allele over another is the test of the hypothesis that the appropriate *h* parameter is equal to plus or minus the additive parameter (d_i), and are obtained by taking differences between sums of squares due to regression for the appropriate alternative models shown in Table 8. As a result, each test is conditional on the restrictions imposed in the previous model. The results of these tests are summarized in Table 8.

Gene action can be clarified by an examination of the inheritance in each of the 3 crosses according to Models 1a and 3a. In both models the differences due to additive effects between parents (d_i) were significant in all cases (Table 7) implying that all parental lines are different.

Table 8. Chi-square test of dominant parameters (h_i) equaling additive parameters (d_i).

Restriction tested		Source of alleles[a]	Sum of squares obtained from	Chi-square (1 d.f.)	Probability (%)
		Model 1			
$h_1 = -d_1$	A^1 completely dominant to A^2	(S, CG)	1a–1b	0.21	50–75
$h_2 = -d_2$	A^1 completely dominant to A^3	(S, N)	1b–1c	2.88	5–10
$h_3 = +d_3$	A^3 completely dominant to A^2	(N, CG)	1c–1e	10.92	<1
		Model 3			
$h_1 = -d_1$	*A* completely dominant to *a*	(S, N)	3a–3b	2.36	10–25
$h_2 = +d_2$	*b* completely dominant to *B*	(N, CG)	3b–3d	153.07	<1

[a]S = Summit, CG = Charleston Gray, N = New Hampshire Midget.

Thus, only the level of dominance in each case will be discussed.

Model 1a—multiple alleles with arbitrary dominance. Family 1 ('Summit' × 'New Hampshire Midget', $A^1 - A^3$ relationship)—The dominance effect $h_2 = -0.47$ was highly significantly different from zero (Table 7), but was not significantly different from $-d_2$ (Table 8). These results imply that A^1 is completely dominant to A^3. The relationship of 'Summit' to 'New Hampshire Midget' (A^1 to A^3), Model 1a, is shown diagramatically in Fig. 1, top.

Family 2 ('Charleston Gray' × 'New Hampshire Midget', $A^2 - A^3$ relationship)—The dominant effect, $h_3 = 0.18$, was significantly different from zero (Table 7), and highly significantly different from $+d_3$ (Table 8) implying some degree of partial dominance of A^3 to A^2 or, 'New Hampshire Midget' was partially dominant to 'Charleston Gray' (Fig. 1, middle).

Family 3 ('Summit' × 'Charleston Gray', $A^1 - A^2$ relationship)—The dominance effect $h_1 = -0.14$ was not significantly different from either $-d_1$ (Table 8) or zero (Table 7). Although complete dominance of A^1 to A^2 was suggested by the data, there was not enough power in the test to make a firm statement on this point (Fig. 1, bottom).

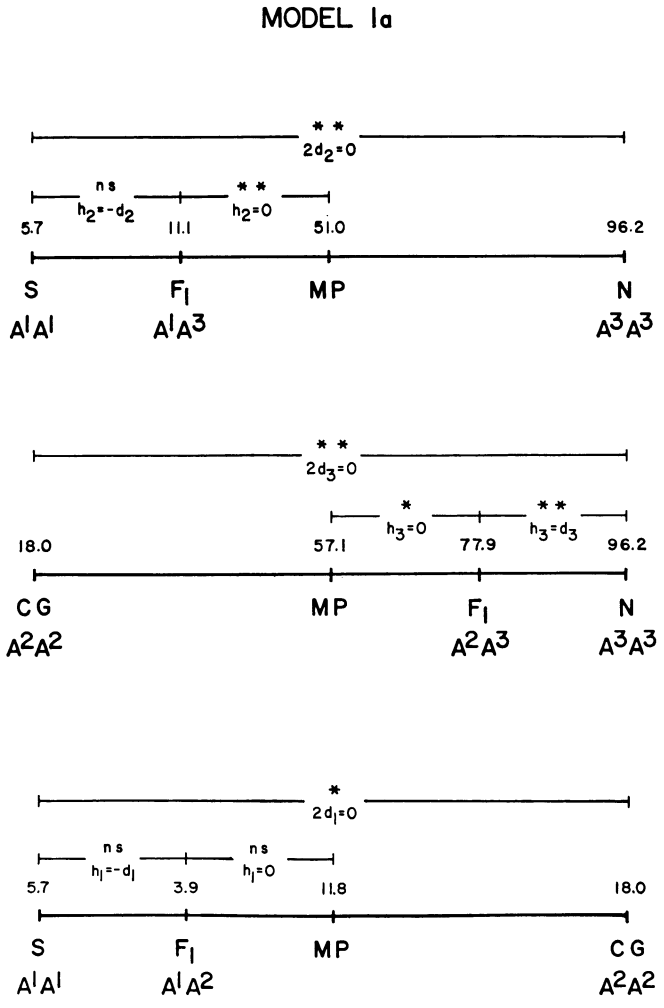
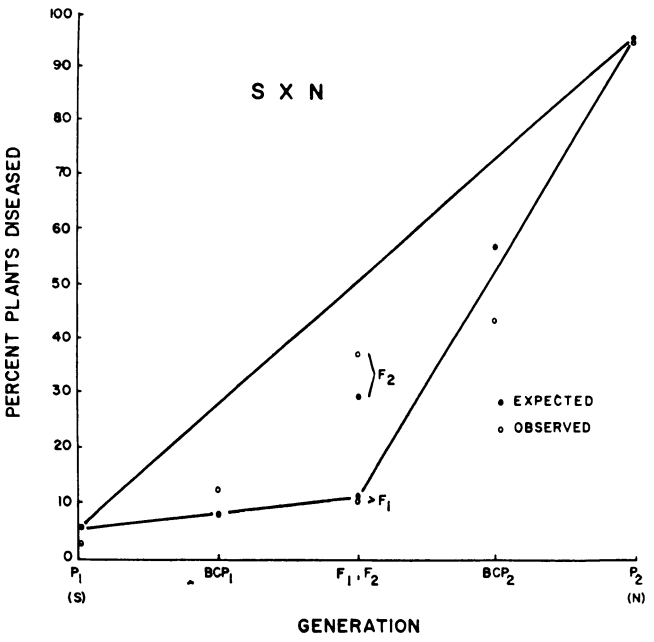
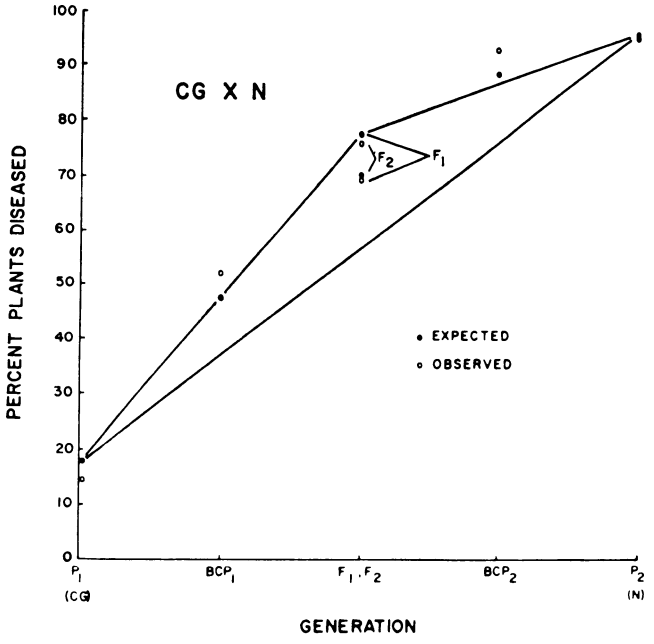


Fig. 1. Model 1a, genetic relationship by family. Top—Summit (S) × New Hampshire Midget (N), middle—Charleston Gray (CG) × New Hampshire Midget (N), and bottom—Summit (S) × Charleston Gray (CG). Numbers indicate the expected per cent diseased plants. P_1, P_2, F_1 and MP are not scaled proportional to the disease percentage.

ns, *, ** = nonsignificance, significance (5% level) and high significance (1% level), respectively, of the null hypothesis given by the equalities immediately below the symbols.





The expected and observed generation values (P_1, P_2, F_1, F_2, BCP_1 and BCP_2) for Model 1a are shown diagramatically by family in Fig. 2 ('Summit' × 'New Hampshire Midget', top, 'Charleston Gray' × 'New Hampshire Midget', middle, and 'Summit' × 'Charleston Gray', bottom). The main discrepancy appeared to be that the observed F_1, in Family 2 ('Charleston Gray' × 'New Hampshire Midget'), was lower than the F_2. The level (per cent diseased plants) of the backcrosses suggested the F_1 was much higher than was observed here.

Model 3a – 2-locus inheritance. (See MATERIALS AND METHODS for complete description.) Family 1 ('Summit' × 'New Hampshire Midget', *A-a* relationship)—The dominance deviation, $h_1 = -0.41$, was highly significantly different from zero (Table 7), but was not significantly

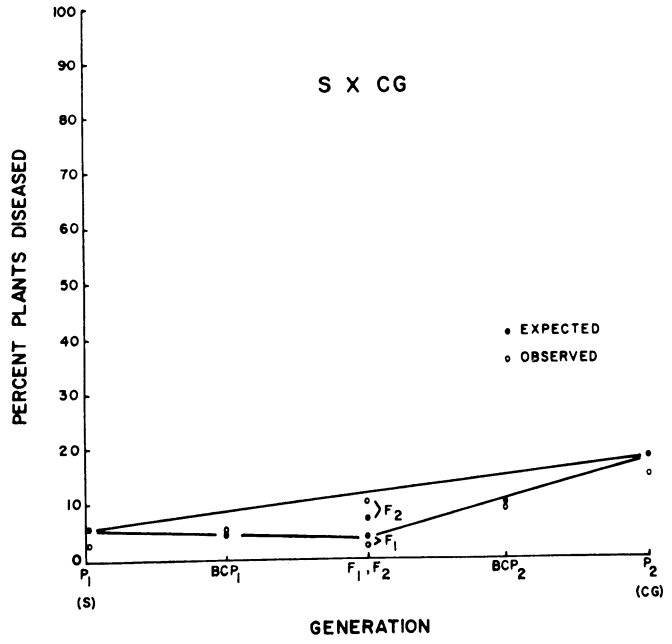


Fig. 2. Per cent diseased plants expected and observed in the P_1, P_2, F_1, F_2, BCP_1 and BCP_2 for Model 1a. Top, left—Summit (S) × New Hampshire Midget (N), bottom, left—Charleston Gray (CG) × New Hampshire Midget (N), and above—Summit (S) × Charleston Gray (CG). Slight distortion from the expected (BCP_2) resulted from fitting model in arcsin $\sqrt{\%}$ (radians) and then decoding the expected values back to per cent.

different from $-d_1$ (Table 8). Therefore, the hypothesis that gene *A* was completely dominant to gene *a* and that the variety 'Summit' was completely dominant to 'New Hampshire Midget' is acceptable. The relationship of 'Summit' to 'New Hampshire Midget' *(A-a)* Model 3a is shown diagramatically in Fig. 3, top.

Family 2 ('Charleston Gray' × 'New Hampshire Midget', *B-b* relationship)—The dominance effect, $h_2 = 0.24$, was highly significantly different from zero (Table 7), and also from $+d_2$ (Table 8). Thus gene *b* acted in a partially dominant manner to its allele *B*, or the susceptible variety 'New Hampshire Midget' was partially dominant to 'Charleston Gray', the resistant variety (Fig. 3, middle).

Family 3 ('Summit' × 'Charleston Gray', *A-a, B-b*)—According to Model 3, the cross of 'Summit' and 'Charleston Gray' involves 2 loci and the F_1, being a double heterozygote, will deviate from the midparent value by the sum of the 2 dominance effects, $h_3 = h_1 + h_2$. Individually, both dominance effects have been shown to be significantly different from zero but in opposite directions so that the combined effect, $h_3 = -.17$ does *not* differ significantly from zero. Even though the F_1 of S × CG was as resistant as the more resistant parent, there was insufficient power in the test to declare the deviation from the midparent significant (Fig. 3, bottom).

The observed and expected genetic values in radians and percentage diseased plants by generation is contained in Table 9.

In summary, all estimates of the additive parameters (d_i) are significantly different from zero in both models. In Model 1, the estimate of h_2 is significantly different from zero but not from $-d_2$ so that complete dominance of A^1 over A^3 appears tenable. There was insufficient power in the test to allow any concrete conclusion regarding the level of dominance of A^1 over A^2; i.e., h_1 was not significantly different from either zero or $-d_1$. On the other hand, h_3 was significantly different from both zero and d_3 implying partial dominance of A^3 over A^2. In Model 3, *A* appears to be dominant to *a*, and *b* is partially dominant to *B*.

Thus, irrespective of gene model, it can be concluded that the resistant variety 'Summit' acted in a completely dominant manner to 'New Hampshire Midget', that the susceptible variety 'New Hampshire Midget' acted in a partially dominant manner to the resistant variety 'Charleston Gray', and that the relationship of 'Summit' to 'Charleston Gray' was not clear, but the data suggested that 'Summit' would behave in a dominant manner to 'Charleston Gray'.

The 2 hypothesized complete gene models showed a high degree of fit to the observed results: Model 1a gave $R^2 = .973$ with 5 parameters and Model 3a gave $R^2 = .970$ with 4 parameters (Table 6). Since both Models 1 and 3 could be reduced to 3 parameters with no significant loss of fit, either model should serve equally as well in explaining the inheritance of Fusarium wilt resistance in the varieties 'Summit' and 'Charleston Gray'; e.g., multiple-allelic system or a 2-locus model of the type

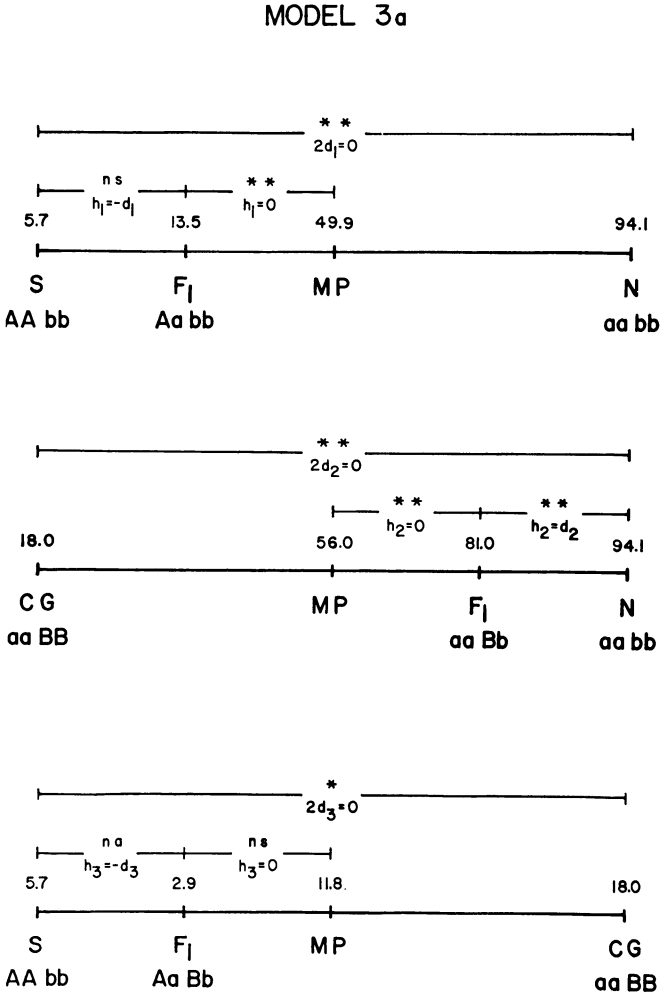


Fig. 3. Model 3a, genetic relationship by family. Top—Summit (S) × New Hampshire Midget (N), middle—Charleson Gray (CG) × New Hampshire Midget (N), and bottom—Summit (S) × Charleston Gray (CG). Numbers indicate the expected per cent diseased plants. P_1, P_2, F_1 and MP are not scaled proportional to the disease percentage.

ns, *, ** = nonsignificance, significance (5% level) and high significance (1% level), respectively, of the null hypothesis given by the equalities immediately below the symbols. (na = test not applicable).

Table 9. Expected and observed genetic values (in radians and percentage individuals diseased) for Models 1a, 1c, 3a and 3c.

Generation[a]	Expected								Observed	
	Model 1a		Model 1c		Model 3a		Model 3c			
	Radians	%	Radians	%	Radians	%	Radians	%	Radians	%
S	.24	5.7	.26	6.5	.24	5.7	.32	9.9	.17	2.8
CG	.44	18.0	.42	16.9	.44	18.0	.44	18.1	.39	14.7
N	1.37	96.2	1.39	96.8	1.33	94.1	1.32	93.7	1.36	95.8
(S × CG) F_1	.20	3.9	.26	6.5	.17	2.9	.14	1.9	.15	2.3
(S × N) F_1	.34	11.1	.26	6.5	.38	13.5	.32	9.9	.33	10.5
(CG × N) F_1	1.08	77.9	1.08	77.9	1.12	81.0	1.14	82.2	.99	69.7
(S × CG) F_2	.27	7.1	.30	8.7	.25	6.4	.26	6.6	.32	10.1
(S × N) F_2	.57	29.4	.54	26.6	.58	30.1	.57	29.1	.66	37.2
(CG × N) F_2	.99	70.3	.99	70.3	1.00	70.9	1.01	71.4	1.06	76.2
(S × CG) × S	.22	4.7	.26	6.5	.21	4.2	.23	5.2	.23	5.1
(S × CG) × CG	.32	9.8	.34	11.2	.30	9.0	.29	8.1	.30	8.9
(S × N) × S	.29	8.2	.26	6.5	.31	9.2	.32	9.9	.36	12.4
(S × N) × N	.86	57.1	.83	54.0	.85	56.6	.82	53.4	.72	43.7
(CG × N) × CG	.76	47.5	.75	46.7	.78	49.3	.79	50.2	.81	52.0
(CG × N) × N	1.23	88.7	1.24	89.2	1.22	88.4	1.23	88.6	1.30	93.1

[a]S = Summit, CG = Charleston Gray, N = New Hampshire Midget.

earlier described for Model 3. However, acceptance of Model 3 as the model for the inheritance of Fusarium wilt resistance implies that there are 2 genotypes which were not observed in this study, *AABB* and *AaBB*. Their predicted genetic values based on the parameter estimates obtained were negative, −0.64 and −0.50 radians, respectively, which may be untenable values; i.e., zero radians corresponds to zero per cent diseased. This suggests: 1) that the 2-locus model is not correct, 2) that some degree of interaction between the loci would have to be incorporated to allow the genotypes to take more reasonable values, or 3) a threshold boundary exists which limits ability to recognize differences, zero diseased plants being such a threshold. Perhaps *AABB* and *AaBB* are superresistant genotypes which further investigation could identify.

It should be emphasized that the difference between 2 parents ascribed to 1 or 2 alleles may be due to *sets* of loci differentiated by 1 or 2 alleles and the resulting dominant and additive effects would be the average for the *sets* (1).

It should be noted that varieties other than those tested may respond differently than reported herein for 'Summit', 'Charleston Gray' and 'New Hampshire Midget'. Moreover, the presence of isolates other than those used in this study could cause a different response within these varieties (9) just as different environmental conditions during development of the disease might do.

On the basis of these results, and with these precautions in mind, the use of a highly resistant variety such as 'Summit' carrying a completely dominant source of resistance could be useful in F_1 hybrid production, as well as in the development of highly resistant open-pollinated varieties. Hybridization with susceptible varieties of good combining ability to produce F_1 hybrids highly resistant to Fusarium wilt should be possible.

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