Quantitatively Assessed Resistance to Bacterial Leaf Spot in Pepper That is Simply Inherited

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ABSTRACT

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Leaves were infiltrated with cells of *Xanthomonas campestris* pv. *vesicatoria* to characterize resistance in a single plant selection of PI 163189 of *Capsicum annuum*. This selection (189-5) contained the *Bs*1 gene for hypersensitivity to race 2 of the bacterial spot pathogen and was detected by rapid necrosis after inoculation of leaves with concentrated inoculum (5×10^8 cfu/ml). A second type of resistance was detected with strains of races 1 and 3 after inoculation of leaves with suspensions containing 2×10^3 cfu/ml and was quantitatively assessed by lesion numbers and lesion diameters 2 wk after inoculation. This resistance could not be detected with the concentrated inoculum. Multiplication of strains of races 1 and 3 ceased in resistant leaves from 2 to 5 days after inoculation; and by 14 days after inoculation, bacterial populations were 10^3–10^4 times fewer than in leaves of the susceptible cultivar, Early Calwonder. In studies of the inheritance of the resistance to races 1 and 3 in populations from the cross, 189-5 X Florida VR-4 (a susceptible pepper cultivar), low numbers and small diameters of lesions were controlled by the same gene. The mean number of lesions in the F_1, F_2, and backcross populations varied according to the bacterial strains used for inoculation. Lesion numbers in heterozygotes were few with the weakly aggressive strain, XV 77-3A of race 3, but many with the strongly aggressive strain, XV 82-8 of race 1. The same additive gene appeared to control resistance to the strain of race 1 and concomitantly, to the strain of race 3. The resistance seems to be a slow form of hypersensitivity that is best assessed by quantitative means, but that is simply inherited.

Bacterial leaf spot, caused by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye, is the most destructive foliar disease of bell pepper (*Capsicum annuum* L.) in warm humid climates. Control of the disease is difficult with chemical sprays because of resistance of the pathogen to streptomycin (31) and to copper (22). Control by sanitation in areas such as Florida is impossible because the pathogen is endemic (19), and wind-blown rains spread the bacterium from field to field. However, control of the disease by genetic resistance seems possible because many accessions of pepper have been identified that contain resistance (27,28).

Three independent single genes for resistance have been identified from the wild collections of pepper (7,8,20). These have been given the designations of *Bs*1, *Bs*2, and *Bs*3, and screening for these genes is typically done by inoculation with concentrated inoculum (10^8 cfu/ml) and observing the plants for rapid necrosis (hypersensitivity). The *Bs*1 and *Bs*3 genes are weak genes as described by Vanderplank (32), because these genes are not effective against some races that already exist in the field. For example, the *Bs*1 gene is effective against race 2 and the *Bs*3 gene

against race 1; but neither gene is effective against race 3 (15). Plants with the gene *Bs*2 have resistance to all known races in the field, but mutants of the pathogen have been found in culture that defeat the *Bs*2 gene (Stall, *unpublished*).

The problems with single-gene resistances have led us to turn to quantitatively assessed resistance in the hope that it would be more durable. The resistance in PI 163189 is one that is thought to be durable (17,24) and was used in breeding resistant bell pepper (3). Useful genetic gains were made by selecting among inbred backcross progenies, but that work was discontinued and seed is no longer available (Borchers, *personal communication*). The line was found to be heterogeneous for hypersensitivity to race 2, but no plant was hypersensitive to race 1 (9). Other evidence from F_2-derived random F_3 lines of a cross between plants of PI 163189 and susceptible pepper implicated a two-gene model for resistance to race 2, namely a dominant gene linked to *Bs*1 and a second, possibly independent, dominant gene (1).

Plants of PI 163189 were resistant to all three races of *X. c.* pv. *vesicatoria* in field plantings (15), but hypersensitivity (HR) after inoculation with high concentrations of bacteria occurred only with strains of race 2. This reaction was inherited independently from resistance to races 1 and 3 (15). Furthermore, resistance to races 1 and 3 could be detected in artificial inoculations using low concentrations of inoculum and assessing the reactions quantitatively as numbers and sizes of lesions (29). The purpose of this paper is to report the analysis of a quantitatively assessed resistance in pepper and to report the inheritance of the resistance in PI 163189.

MATERIALS AND METHODS

Plant culture. Seeds of PI 163189 were obtained from the Southern Regional Plant Introduction Station, Experiment, GA. Inbred progenies of a single plant of PI 163189, designated as 189-5, homozygous for rapid HR to race 2 strains and resistant to strains of races 1 and 3, were obtained in this work. Seeds were on hand for the susceptible control, Early Calwonder (ECW), and its near-isogenic cultigens, Early Calwonder 10R (ECW-10R) and Early Dalwonder 30R (ECW-30R), with the *Bs*1 and *Bs*3, respectively. Seeds of Florida VR-4 (VR-4), which contains the *Bs*1 gene, were obtained from Dr. A. A. Cook, Plant Pathology Department, University of Florida, Gainesville (6).

Seedlings were transplanted to steamed peat-vermiculite mix in 10-cm plastic pots. The plants were arranged in rows of eight plants, which were randomized according to cultivar. They were placed on a bench in a greenhouse with temperatures ranging from 20 to 35 C. Plants were watered as required and treated four times during the experiments with approximately 0.4 g per pot of soluble 20:20:20 fertilizer.

Inoculum preparation. Two strains of race 1 (XV 80-5 and XV 82-8), one strain of race 2 (XV E3), and two strains of race 3 (XV 69-1 and XV 77-3A) were used as inoculum in this study. Pathogenicity of all strains was tested in plants of the susceptible cultivar, Early Calwonder. Races were confirmed by the presence or absence of HR in pepper lines with genes *Bs*1 and *Bs*3 (ECW-10R and ECW-30R).

The strains had been stored in sterilized tap water, and when recovered they were kept in 15% glycerol at −20 C between tests. Inocula were prepared from late log-phase nutrient broth cultures derived from single colonies on nutrient agar plates. After centrifugation of cultures at 1,000 *g*, the bacterial pellets were suspended in sterile tap water and standardized photometrically to 0.3A at 600-μm wavelength, which approximated a density of 5×10^8 cfu/ml. These suspensions were either used directly or serially diluted to approximately 10^3 cfu/ml for inoculations. The latter concentration was confirmed by replicated colony counts from 0.05-ml subsamples spread on nutrient agar plates. All inoculations were by infiltration of intercostal leaf tissues of fully expanded leaves (21).

Bacterial populations. Plants of 189-5, ECW, and ECW-10R were inoculated with strains of race 1 (XV 80-5), race 2 (XV E3), and race 3 (XV 69-1). Inoculum concentrations of 1.4×10^3, 2.2×10^3, and 0.8×10^3 cfu/ml of the strains of races 1, 2, and 3, respectively were infiltrated into an area of 4 to 5 cm^2 in each of three leaves per plant. Leaves were collected periodically after inoculation, and bacterial populations in the inoculated areas were determined from a 1-cm^2 sample. Samples were triturated in 0.5 ml of sterile tap water, the suspension serially diluted 10-fold when appropriate in sterile tap water, and 0.05-ml subsamples of the final dilution were spread on nutrient agar plates. Colonies of *X. c.* pv. *vesicatoria* were counted after 2 to 3 days of incubation at 30 C, and means were converted to $\log_{10}$ values. There were three replicates of each assay and the experiment was repeated. Means of the six assays were designated as populations of bacteria in the inoculated areas.

Electrolyte leakage. Each of three fully expanded leaves below the first fork of the stem of a plant was inoculated with strains of race 1 (XV 80-5), race 2 (XV E3), and race 3 (XV 69-1) standardized to a concentration of 5×10^8 cfu/ml. Inocula were infiltrated into a 4- to 5-cm^2 area of leaf with each strain. Each strain was placed in each of the three leaves per plant. After inoculation, the plants were kept in a growth chamber held at 30 C. Six disks of tissue, each of 0.5 cm^2 in size were recoved from the inoculated area of each leaf at 9–15-hr intervals for 60 hr after inoculation and placed in test tubes containing 3.0 ml of sterile deionized water. A piece of plastic mesh was inserted into the tubes to keep the disks submerged. Conductivity (μmhos) of the suspending solution was recorded immediately and again after vacuum infiltration at 63 cm Hg for 60 sec and agitation for 1 hr at 30 C. The difference in conductivity between the two readings was taken to represent the influence of bacteria on host tissue. There were three replicates, and the experiment was repeated. Means of the three replicates of one experiment are reported.

Quantitative assessment of disease. The number of lesions within the perimeter of a cork borer of 2 cm^2 imprinted on the leaf was counted at each inoculation site while viewed with a dissecting microscope at a magnification of 2.5×. The diameters of five random lesions per site, or of all lesions where fewer than five existed, were measured with a graduated eyepiece in the microscope.

Inheritance of quantitatively assessed resistance. A single plant of the homozygous selection, 189-5, was cross-pollinated with a single plant of VR-4. The VR-4 plant was used because VR-4 appeared to be more susceptible to race 1 in the field than was ECW, another susceptible parent (15). A single F_1 plant was self-pollinated to yield F_2 seeds and also cross-pollinated with an additional plant of both parents to give backcross seeds. Populations of 282 F_2 seedlings, 40–50 seedlings of each of the two backcrosses, 30–40 seedlings of both 189-5 and the F_1 populations, and 10 each of VR-4, ECW-10R, and ECW seedlings were transplanted to pots and grown as described above.

Both parents were homozygous for the *Bs*1 gene, and, therefore, all progeny were homozygous for resistance to race 2. As a result, race 2 was not used to determine the quantitatively assessed resistance. The three leaves preceding the first fork of the stem were each infiltrated with a suspension of approximately 2×10^3 cfu/ml each of race 1 (XV 82-8) and race 3 (XV 77-3A). Overlapping of areas infiltrated with each race was avoided. These leaves were harvested 14 days later, sealed in plastic bags, and refrigerated (4 C). Over the next several days, the numbers of discrete lesions per 2 cm^2 of leaf and diameters of the lesions were determined as described above. Mean values for lesion numbers and diameters were obtained for each strain on each plant.

Statistical treatment of data. The quantitative data were evaluated by analysis of generation means weighted by their variances (2,14,23). No prior assumptions on the relative importance of dominance is required in this analysis. The matrix specification of Fisher (12) was used with parameters: m = general theoretical population mean at F_∞, a = value of additivity, and d = value of dominance deviation. The analysis applies a X^2 goodness of fit test to computed parameters for a simple, single-gene model. In the event of poor fit, the analysis proceeds to fit a digenic model with interactions (2). Specific comparisons of means were performed by the Student's t test.

RESULTS AND DISCUSSION

Slow hypersensitive resistance in 189-5. Hypersensitive resistance in plants of 189-5 and ECW-10R to strains of race 2 was detected by infiltration of high inoculum (5×10^8 cells/ml) into leaves. Electrolyte leakage from such leaves was complete by 9 hr after inoculation (Fig. 1). The curves of electrolyte leakage from leaves of 189-5 after infiltrations with strains of races 1 and 3 at the high inoculum levels approximated that obtained by inoculation of susceptible leaves (ECW) with the same inoculum, however. Therefore, detection of resistance in plants to races 1 and 3 was not possible by use of concentrated inoculum. In contrast, after inoculation with a low concentration (2×10^3 cells/ml) of bacteria, lesions in leaves of 189-5 were few in number and were of small diameter for all the strains tested (Table 1). Many lesions of relatively large diameter developed in leaves of ECW with strains of all three races and in leaves of ECW-10R with the strains of races 1 and 3 when inoculated with the low concentration. The lesions became visible in leaves 5 to 6 days after inoculation and continued to increase in size. A few small necrotic hypersensitive flecks developed in leaves of ECW-10R inoculated with the strain of race 2 used at the low concentration.

The cell populations of all three strains increased to between 10^7 and 10^8 cfu/cm in leaves of the susceptible cultivar, ECW, when inoculum of $1-3 \times 10^3$ was used (Fig. 2). With the same inoculum level, cell populations of all strains in leaves of 189-5 and the strain of race 2 in ECW-10R plateaued between 2 and 5 days after inoculation and at levels of $10^3 - 10^5$ cfu/cm^2.

The curves for increase of populations of strains of the three races of *X. c.* pv. *vesicatoria* in leaves of 189-5 were similar to the strain of race 2 in ECW-10R. The bacterial increases in leaves of ECW-10R plants inoculated with the strain of race 2 were also very similar to those previously published for plants that contained the *Bs*1 gene and given as evidence that race 2 strains cause an HR in pepper leaves with the *Bs*1 gene (30). By analogy then, the resistance in 189-5 to strains of races 1 and 3 may also be conditioned by an HR. If so, the HR to strains of races 1 and 3 in 189-5 fails to produce the rapid necrotic response as that to strains of race 2 in leaves of ECW-10R. As a result of the slow HR some small lesions develop in leaves of 189-5 inoculated with low concentrations of strains of races 1 and 3 and HR to those strains

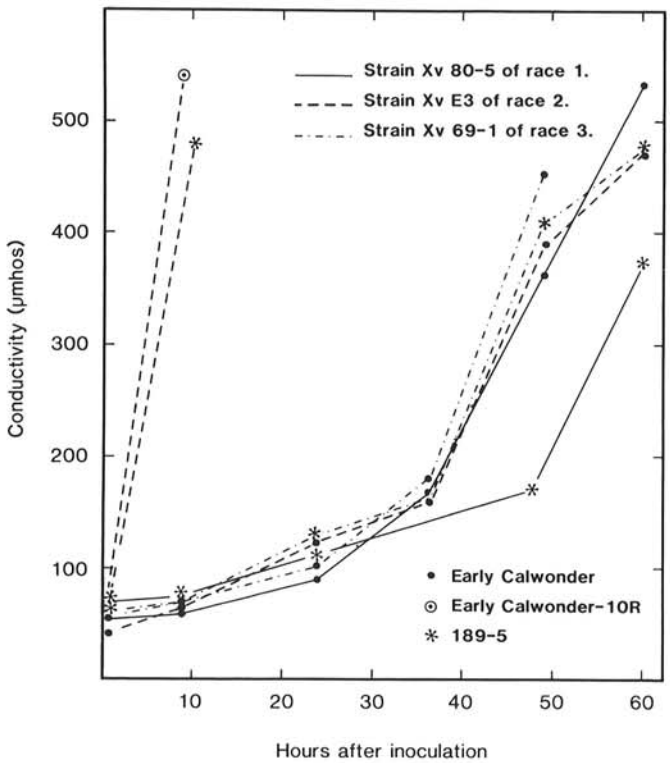

Fig. 1. Electrolyte leakage from leaves of 189-5 and Early Calwonder pepper plants that were inoculated with strains of races 1, 2, and 3 of *Xanthomonas campestris* pv. *vesicatoria*. Early Calwonder-10R was inoculated with a strain of race 2 as a hypersensitive control. Points represent means of three replicates.

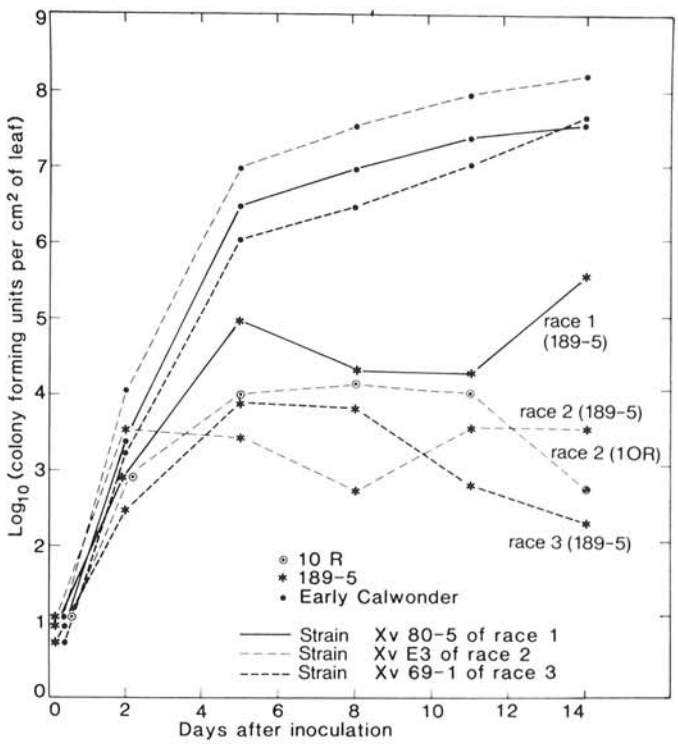

Fig. 2. Bacterial populations in leaves of Early Calwonder and 189-5 after inoculation with strains of races 1, 2, and 3 of *Xanthomonas campestris* pv. *vesicatoria*. Early Calwonder-10R was inoculated with a strain of race 2 as a hypersensitive control. Points represent means of six replicates.

TABLE 1. Numbers and diameters of lesions in leaves of pepper lines 15 days after inoculation with races 1, 2, or 3 of *Xanthomonas campestris* pv. *vesicatoria*, using an inoculum concentration of approximately 1.5×10^3 cfu/ml

Host	XV 80-5 (race 1)		XV E3 (race 2)		XV 69-1 (race 3)	
	Lesions (no.)	Lesion diameter[a]	Lesions (no.)	Lesion diameter[a]	Lesions (no.)	Lesion diameter[a]
189-5[b]	0.9 ± 0.54[c]	1.7 ± 0.41	0.4 ± 0.36	2.0 ± 1.41	0.2 ± 0.24	1.0 ± 0.0
ECW[c]	22.3 ± 1.27	6.9 ± 0.88	28.0 ± 1.67	8.2 ± 1.07	15.4 ± 1.71	7.5 ± 1.19
ECW-10R[d]	24.7 ± 2.08	4.9 ± 0.33	5.7 ± 0.94	1.7 ± 0.29	11.1 ± 1.95	5.4 ± 0.82

[a] Lesion diameter in mm × 10.
[b] 189-5 = Resistant selection from PI 163189.
[c] ECW = Early Calwonder (susceptible).
[d] ECW-10R carried *Bs*1 gene for resistance to race 2.
[e] Mean ± standard error of mean.

can be detected by comparing the quantitative measurements of lesion numbers and lesion diameters.

Quantitative assessment of resistance. Differences occurred among the plant generations for numbers of lesions per inoculated area and mean diameter per lesion in leaves infiltrated with the strains of both races 1 and 3 at low concentrations of inoculum. The frequency distributions of plants in each generation based on categories of lesion numbers and lesion diameters for race 1 and for race 3 are in Tables 2 and 3. The distributions of plants inoculated with strain XV 82-8 of race 1 were consistently skewed towards greater numbers and diameters than those inoculated with strain XV 77-3A of race 3. Also, generation means of lesion numbers and of lesion diameters were correlated for each strain, XV 82-8 and XV 77-3A ($r = 0.75$, $P < 0.01$, and $r = 0.94$, $p < 0.01$, respectively).

Single-gene hypothesis. After inoculation with strain XV 82-8 of race 1, plants in the F_1 and backcross to VR-4 generations were skewed toward susceptibility for both components of resistance, but wide variation in both components occurred in the F_2 and backcross to 189-5 generations (Tables 2 and 3). The frequency distributions varied continuously and did not seem to fit into any segregation pattern or ratio. However, in the F_2 generation, the distributions were intermediate between the parents, which could mean an additive genetic effect.

The inheritance patterns with the same plants inoculated with the strain XV 77-3A of race 3 was somewhat different. The frequency of plants in the F_1, F_2, and backcross to 189-5 generations was skewed toward the resistant parent for both resistance components. This type of distribution could mean that resistance was dominant for the race 3 strain. For lesion number, two distinct groups of plants occurred in equal numbers in the population from the backcross to VR-4 (Table 4), i.e., the distribution appeared bimodal. One group was classified as resistant and had mean values of both components of resistance not significantly different from the F_1 population means. The remaining plants of the backcross to VR-4 were classified as susceptible. The mean values of both components of resistance in this group were much greater ($P < 0.01$) than those of the resistant group in this population, but were slightly lower ($P < 0.05$) than for plants of the susceptible parent, VR-4. This could mean that a single dominant gene contributed much to resistance to this strain of race 3.

Despite continuous distribution of plants in the generations based on both resistance components, plants were classified into categories of either resistant or susceptible to strain XV 77-3A of race 3. The cutoff point for classification was 8.7 lesions per 2 cm^2 combined with 0.30-mm lesion diameter. This classification resulted in 203 resistant and 76 susceptible plants in the F_2 population (Table 4). Difficulty was found in classifying 13 (4.7%) of those plants. However, this ratio is consistent with a single dominant gene for resistance in 189-5 ($X^2 = 0.75$, $P = 0.5$–0.3). The mean values for both components did not differ significantly ($P < 0.05$) among resistant plants in F_1, F_2, and backcross to VR-4 populations. These means also were marginally but significantly greater ($P < 0.05$) than means for resistant plants in the 189-5 and backcross ($F_1 \times$ 189-5) populations.

The correlation of resistance (or susceptibility) in plants inoculated with both races at the same time gave strong support to the hypothesis that the same gene in 189-5 confers resistance to

TABLE 2. Frequency distributions for lesion number in populations of pepper plants derived from the cross VR-4 (susceptible) × 189-5 (resistant) after leaf inoculation (1.3×10^3 cfu/ml) with strain SV82-8 of race 1 and WV 77-3A of race 3 of *Xanthomonas campestris* pv. *vesicatoria*

Plant	*Xcv*	Upper class limits for number of lesions per 2 cm^2											Population mean S.E.
populations	race	3	6	9	12	15	18	21	24	27	30	>30	of mean
VR-4	1							2	2	1	2		23 ± 1.55
	3					2		3	3				18 ± 1.23
189-5	1	16	7	4	2								3.5 ± 0.47
	3	30											0.1 ± 0.55
F_1	1			1	3	8	9	3	6	5	3	1	18.6 ± 0.94
	3	27	10	2									2.1 ± 0.32
F_2	1	8	11	13	24	37	51	49	39	26	12	8	17.4 ± 0.40
	3	152	31	21	16	24	15	7	10	1		2	5.8 ± 0.42
BC (VR-4)	1				1	3	2	12	13	8	4	1	21.2 ± 0.64
	3	17	4	2	4	5	6	2	1				9.3 ± 1.23
	1	6	3	7	11	3	5	2	4	4	2		12.7 ± 1.20
BC (189-5)	3	44	2	1									0.8 ± 0.18

TABLE 3. Frequency distributions for lesion diameter in populations of pepper plants derived from the cross VR-4 (susceptible) × 189-5 (resistant) after leaf inoculation (1.3×10^3 cfu/ml) with strain XV 82-8 of race 1 and XV 77-3A of race 3 of *Xanthomonas campestris* pv. *vesicatoria*

Plant	*Xcv*	Upper class limits for lesion diameter (Mean of 5 lesions: mm × 10)									Population mean ± S.E.
populations	race	2	4	6	8	10	12	14	16	18	of mean
VR-4	1					1	2	2	2		14.6 ± 0.32
	3		2	3	2	1					5.1 ± 0.67
189-5	1	19	8	2							1.7 ± 0.10
	3	30									0.2 ± 0.08
F_1	1		2	14	14	7	2				6.5 ± 0.29
	3	11	20	7	1						2.4 ± 0.26
F_2	1	5	49	77	61	39	19	9	7	14	6.5 ± 0.27
	3	89	146	39	4	1					2.5 ± 0.09
BC (VR-4)	1			1	5	11	13	5	4	5	11.7 ± 0.77
	3	3	29	10	2						3.2 ± 0.18
BC (189-5)	1	9	21	8	4	1	2		1	1	4.6 ± 0.59
	3	36	10	1							1.2 ± 0.17

TABLE 4. Number of lesions per 2 cm^2 of leaf and diameter per lesion from resistant and susceptible plants in parental, F_1, F_2, both backcrosses, and check lines inoculated with 1.5×10^3 cfu/ml of race 3 strain XV 77-3A of *Xanthomonas campestris* pv. *vesicatoria*

Generation of cultivar	Resistant plants			Susceptible plants		
	Number	Lesions per 2 cm^2	Diameter[a] per lesion (mm × 10)	Number	Lesions per 2 cm^2	Diameter[a] per lesion (mm × 10)
VR-4	...	...	...	8	18.5 ± 1.23[b]	5.1 ± 0.72
189-5	30	0.1 ± 0.05	1.2 ± 0.21	...	...	...
(VR-4 189-5) F_1	39	2.1 ± 0.32	2.9 ± 0.22	...	...	...
F_2	203	2.1 ± 0.16	2.3 ± 0.07	76	16.0 ± 0.06	3.8 ± 0.13
(F_1 × VR-4)	22	2.1 ± 0.31	2.7 ± 0.23	22	16.6 ± 0.98	3.8 ± 0.21
(F_1 × 189-5)	47	0.9 ± 0.18	1.8 ± 0.16	...	...	...
Controls						
10R	...	...	...	10	12.4 ± 1.64	3.7 ± 0.83
Early Calwonder	...	...	...	9	13.7 ± 0.92	3.6 ± 0.24

[a] Calculated from those plants with visible lesions.
[b] Mean ± standard error of mean.

TABLE 5. Generation means analysis of lesions per 2 cm^2 of leaf and lesion diameter from VR-4 × 189-5 pepper progenies inoculated with strain XV 82-8 of race 1 and strain XV 77-3A of race 3 of *Xanthomonas campestris* pv. *vesicatoria*

Bacterial strain	Parameter[a] computer	Parameter[b] estimate	Parameter[b] estimate
XV 82-8 (race 1)	*m*	13.7 ± 0.56	6.9 ± 0.69
	a	9.9 ± 0.56	6.7 ± 0.49
	d	6.3 ± 1.12	−0.3 ± 0.86
	aa	N.A.[c]	1.6 ± 0.86
	X^{2d}	6.11 ($P<0.95$)	5.64 ($P<0.95$)
XV 77-3A (race 3)	*m*	9.4 ± 0.43	2.6 ± 0.15
	a	9.3 ± 0.43	1.6 ± 0.15
	d	−7.7 ±0.46	0.3 ± 0.26
	X^{2} [d]	3.64 ($P<0.95$)	5.48 ($P<0.95$)

[a] Parameters: *m* = estimated mean at F; *a* = additivity; *d* = dominance deviation; *aa* = additive epistasis.
[b] Estimated value ± standard error of estimate.
[c] Not applicable.
[d] X^2 goodness of fit test value and *P* level.

races 1 and 3. All F_2 plants listed as susceptible to strain XV 77-3A of race 3 were, without exception, listed as susceptible to strain XV 82-8 of race 1. Resistance to strain XV 82-8 occurred only among F_2 plants that were resistant also to strain XV 77-3A. All plants of the backcross to VR-4 were susceptible to strain XV 82-8 irrespective of reaction (resistant or susceptible) to strain XV 77-3A. The lesion diameters caused by the two strains in F_2 plants were moderately correlated ($r = 0.61$, $P < 0.05$), whereas numbers of lesions were only weakly correlated ($r = 0.33$).

Generation means analysis. Resistance to strain XV 77-3A appeared to be dominant and under monogenic control, but quantitative and correlated with quantitative resistance to strain XV 82-8. The lesion number component of resistance to strain XV 82-8 appeared to be recessive. To investigate further the number of genes involved in resistance, analyses were made of the weighted generation means of both components of resistance. As expected, single-gene models fit the data for lesion numbers with both strains of the pathogen (Table 5). Dominance of high lesion numbers with strain XV 82-8 and of low numbers with strain XV 77-3A was incomplete. A single-gene model adequately fitted generation means of lesion diameter with strain XV 77-3A, but marginally failed to fit with strain XV 82-8. A digenic model with additive epistasis fit the lesion diameter means with strain XV 82-8. Dominance for lesion diameter was negligible with both strains, and in both, additivity was significant and the parameter of major importance. Statistical deviation from a single gene model for lesion diameter with strain XV 82-8 was probably a result of sampling error, because means were based on few measurements

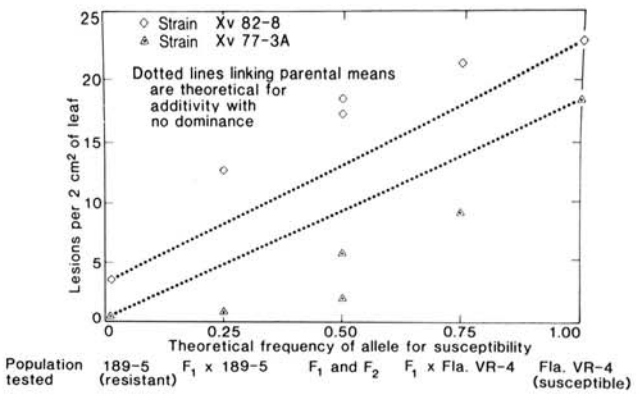

Fig. 3. Variation in lesion numbers related to theoretical frequency of the allele for susceptibility in pepper generations inoculated with strains of races 1 and 3 of *Xanthomonas campestris* pv. *vesicatoria*.

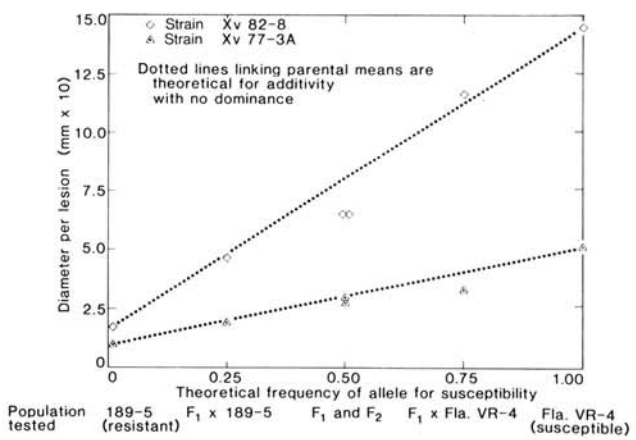

Fig. 4. Variation in lesion diameters related to theoretical frequency of the allele for susceptibility in pepper generations inoculated with strains of races 1 and 3 of *Xanthomonas campestris* pv. *vesicatoria*.

from the parent, VR-4.

Additive inheritance is based on a linear increase in means with increased frequency of the allele(s) and dominance is assessed as deviation from linearity (10). These relationships are illustrated in Figure 3 for lesion numbers and Figure 4 for lesion diameters. The theoretical frequency of one allele of a single gene in F_1 and F_2 progenies is 0.5 and is 0.25 and 0.75 in respective backcrosses. When generation means were plotted against these allele frequencies, lesion diameters with both strains increased linearly, but lesion numbers increased nonlinearly in accordance with dominance.

Conclusions and practical applications. The determinations of inheritance of the resistance to races 1 and 3 of *X. c.* pv. *vesicatoria* in leaves of 189-5 is complicated by the necessity of accumulation of quantitative data. The quantitative data are also subject to variation caused by environment and measurement errors. Such data would appear superficially to support polygenic inheritance of resistance. Upon statistical analysis, however, the quantitatively assessed resistance in 189-5 to races 1 and 3 is inherited as a single gene.

Additive gene action basically controlled lesion diameter with both bacterial strains in leaves of 189-5. The rate of lesion expansion was reduced in linear proportion to the frequency of the resistance allele. On the other hand, control of low lesion numbers seems to be dominant with XV 77-3A and recessive with XV 82-8. Lesion numbers may not be a good indicator of inheritance, because bacterial multiplication may be so restricted in homozygous resistant plants that very few necrotic areas become visible lesions. More necrotic areas grow to become visible lesions when resistant plants are inoculated with an aggressive strain rather than with a weak strain. In addition, the expansion rate of lesions in heterozygotes appeared to be insufficient to prevent all potential lesions of an aggressive strain from becoming visible but was sufficient to prevent most necroses of the weak strain from becoming visible. Consequently, reversal of apparent dominance of gene action controlling lesion numbers would occur in proportion to the aggressiveness of the bacterial strains. Studies of population dynamics in heterozygotes and homozygotes should clarify this phenomenon. Nevertheless, the continuous variability observed from both lesion numbers and lesion diameters among populations of F_2 progenies would support the hypothesis that some sort of additive gene action is involved with slow-forming hypersensitivity in PI 163189.

Practical use of additive resistance in breeding is important. Resistance in heterozygotes varied with the aggressiveness of the strain. It should be assumed that naturally occurring strains of *X. c.* pv. *vesicatoria* are aggressive. In this event, each cycle of selection for a high degree of resistance in recurrent backcrosses to bell pepper will require inbred progenies. This finding is consistent with previous work in selecting resistant segregates from self-pollinated progenies (1,3). Alternatively, susceptible but heterozygous parents may be identified by progeny-testing in single-generation cycles of recurrent backcrossing (13). A third alternative involves identifying heterozygous backcross plants by inoculating with a weakly aggressive strain.

Many recessive resistances to bacterial plant pathogens, including pepper, have been reported in the literature (4,5,11,16,18,25,29). In some, the resistance was incompletely recessive in heterozygotes (16). Sidhu and Khush (26) reported that reversal of dominance in heterozygotes may occur with aging of plants. These reports may, in fact, reflect additive gene action controlling reduced rates of multiplication of the pathogens.

LITERATURE CITED

1. Adamson, W. C., and Sowell, G. 1983. Inheritance of bacterial spot resistance in pepper. HortScience 18:905-906.
2. Basford, K. E., and DeLacy, I. H. 1979. The use of matrix specifications in defining gene action in genotypic value models and generation mean analysis. Theor. Appl. Genet. 55:225-229.
3. Borchers, E. A. 1985. Breeding bacterial leaf spot resistant bell peppers. Vegetable Growers News 19 (7), Virginia Truck and Ornamentals Res. St., Norfolk, VA.
4. Brinkerhoff, L. A., Verhalen, L. M., Johnson, W. M., Essenberg, M., and Richardson, P. E. 1984. Development of immunity to bacterial blight of cotton and its implications for other diseases. Plant Dis. 68:168-173.
5. Chand, J. N., and Walker, J. C. 1964. Inheritance of resistance to angular leaf spot of cucumber. Phytopathology 54:51-53.
6. Cook, A. A. 1984. 'Florida VR-4' bell pepper. HortScience 19:456.
7. Cook, A. A., and Guevara, Y. G. 1984. Hypersensitivity in *Capsicum chacoense* to race 1 of the bacterial spot pathogen of pepper. Plant Dis. 68:329-330.
8. Cook, A. A., and Stall, R. E. 1963. Inheritance of resistance in pepper to bacterial spot. Phytopathology 53:1060-1062.
9. Cook, A. A., and Stall, R. E. 1969. Differentiation of pathotypes among isolates of *Xanthomonas vesicatoria*. Plant Dis. Rep. 53:617-619.
10. Falconer, D. S. 1960. Introduction to Quantitative Genetics. Oliver and Boyd, Edinburgh, United Kingdom. 365 pp.
11. Fallik, E., Bashan, Y., Okon, Y., and Kadar, N. 1984. Genetics of resistance to bacterial speck of tomato caused by *Pseudomonas syringae* pv. *tomato*. Ann. Appl. Biol. 104:321-325.
12. Fisher, R. A. 1918. The correlation between relatives on the supposition of Mendelian inheritance. Trans. R. Soc. Edinburgh 52:399-433.
13. Hanson, W. D. 1959. Minimum family sizes for the planning of genetic experiments. Agron. J. 51:711-715.
14. Hayman, B. I. 1958. The separation of epistatic from additive and dominance variation in generation means. Heredity 12:371-390.
15. Hibberd, A. M. 1985. The inheritance of components of resistance to bacterial leaf spot of pepper (*Capsicum annuum* L.) Ph.D. dissertation. University of Florida, Gainesville. 217 pp.
16. Hibberd, A.M., and Gillespie, D. 1982. Heritability of field resistance to bacterial leaf spot in pepper (*Capsicum annuum* L.). Sci. Hortic. 17:301-309.
17. Hibberd, A. M., Herrington, M. E., and Gillespie, D. 1979. Sweet pepper disease resistance breeding in Queensland, Australia. Veg. Hot Humid Tropics 4:63-64.
18. Innes, N. L., Conway, J., and Taylor, J. D. 1984. Resistance to halo blight in the Cambridge accessions V4604 and V4058 of *Phaseolus* beans. Ann. Appl. Biol. 104:307-314.
19. Jones, J. B., Pohronezny, K. L., Stall, R. E., and Jones, J. P. 1986. Survival of *Xanthomonas campestris* pv. *vesicatoria* in Florida on tomato crop residues, weeds, seeds, and volunteer tomato plants. Phytopathology 76:430-434.
20. Kim, B-S., and Hartmann, R. W. 1985. Inheritance of a gene (Bs3) conferring hypersensitive resistance to *Xanthomonas campestris* pv. *vesicatoria* in pepper (*Capsicum annuum*). Plant Dis. 69:233-235.
21. Klement, Z., Farkas, G. L., and Lovrekovitch, L. 1964. Hypersensitive reaction induced by phytopathogenic bacteria in the tobacco leaf. Phytopathology 54:474-477.
22. Marco, G. M., and Stall, R. E. 1983. Control of bacterial spot of pepper initiated by strains of *Xanthomonas campestris* pv. *vesicatoria* which differ in sensitivity to copper. Plant Dis. 67:779-781.
23. Mather, K., and Jinks, J. L. 1971. Biometrical Genetics. The Study of Continuous Variation. Cornell University Press, Ithaca, NY. 382 pp.
24. Parlevliet, J. E. 1983. Durable resistance in self-fertilizing annuals. Pages 347-381 in: Durable Resistance in Crops. R. Lamberti, J. M. Waller, and N. A. Van der Graff, eds. NATO ASI Series. Series A: Life Sciences. Vol. 55. Plenum Press, New York. 454 pp.
25. Patel, P. N. 1982. Genetics of host reactions to three races of the bacterial pustule pathogen in cowpea. Euphytica 31:805-814.
26. Sidhu, G. S., and Khush, G. S. 1978. Dominance reversal of a bacterial blight resistance gene in some rice cultivars. Phytopathology 68:461-463.
27. Sowell, G. 1960. Bacterial spot resistance of introduced peppers. Plant Dis. Rep. 44:587-590.
28. Sowell, G., and Dempsey, A. H. 1977. Additional sources of resistance to bacterial leaf spot of pepper. Plant Dis. Rep. 61:684-686.
29. Stall, R. E. 1981. Selection for components of horizontal resistance to bacterial spot of pepper. Pages 511-517 in: Proc. of the Fifth Intern. Conf. on Plant Pathogenic Bacteria. J. C. Lozano, ed. CIAT, Cali, Columbia. 640 pp.
30. Stall, R. E., and Cook, A. A. 1966. Multiplication of *Xanthomonas vesicatoria* and lesion development in resistant and susceptible pepper. Phytopathology 56:1152-1154.
31. Stall, R. E., and Thayer, P. L. 1962. Streptomycin resistance of the bacterial spot pathogen and control with streptomycin. Plant Dis. Rep. 456:389-392.
32. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 206 pp.