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Inheritance of Tolerance to Rhizoctonia Fruit Rot of Tomato[1]

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Abstract. **The inheritance of tolerance to rhizoctonia fruit rot incited by *Rhizoctonia solani* Kuhn differed depending upon the source of the plant material of tomato (*Lycopersicon esculentum* Mill.). The tolerance in USDA 75B 846-1-1 was controlled by 1 major gene without dominance. The tolerance in USDA 75B 610-3 was polygenic with 4 major genes. Tolerance in USDA 75B 846-1-1 had a narrow-sense heritability of 71% while in USDA 75B 610-3 heritability was 30%. Fruit rot tolerance and resistance to puncture pressure were highly correlated in both families. Fruit shape and fruit rot tolerance were also highly correlated in the family with USDA 75B 846-1-1.**

A limiting factor in the production of processing tomatoes in the southern United States is the prevalence of fruit rot caused by the soil-borne fungus, *Rhizoctonia solani* (2, 3, 5, 7, 11, 12, 19). This rot can reduce yields by up to 40% if moisture and temperature are high (2, 3, 5, 12, 20). Neither chemicals nor use of mulches have provided adequate control (5, 6, 7, 8, 12, 15, 16, 19).

Many cultivars and Plant Introductions (PI) have been screened for resistance and acceptable levels of tolerance have been found in some PI lines, but the trait is probably not simply inherited (3, 4). The actual mechanism of tolerance is not clearly understood, but it appears to involve the nature of the fruit's epidermal layer (8, 9, 10). For penetration to occur, the fungus must first form an infection cushion. Hyphal pegs then penetrate the cuticle and invade the epidermal cells. Although the presence of enzymes and/or toxins have been shown to precede penetration in other hosts, this has not been demonstrated with tomato fruit (10, 13). Tolerance to infection may be related to skin thickness or toughness (18).

The objectives of this study were to determine the number of genes involved in tolerance to rhizoctonia fruit rot, the narrow-sense heritability of this tolerance, and the correlation between tolerance to fungal penetration and resistance to puncture.

Materials and Methods

Single plants from two USDA tomato breeding lines were selected for tolerance to rhizcotonia fruit rot by Dr. T. H. Barksdale at Agricultural Research Station, Beltsville, Maryland in 1975. These lines were crossed with a susceptible cultivar ('Campbell-28') to develop genetic families which included the parents, the F_1, F_2, and backcrosses to each parent. The 3 parental lines are characterized below:

P_1: a single plant selection from PI 205001 (USDA 75B 846-1-1), resistant to soil rot, small oblong fruit, indeterminate, rank vine growth.

P_2: a single plant selection from cross of PI 269140 × PI 272636 (USDA 75B 610-3), resistant to soil rot, small round fruit. indeterminate, low spreading vine growth.

P_3: a commercial cultivar ('Campbell-28'), susceptible to soil rot, large round fruit, determinate, compact vine growth.

A modified randomized complete block with 10 replications was the field design. Each replicate had 36 plots containing 2 plot entries for each parent and F_1, 3 plot entries for each backcross, and 6 plot entries for the F_2. All plots contained 5 plants and were randomly distributed within each replicate. Fifteen fruit were harvested from each plant at the green mature stage to standardize maturity, and brought to the laboratory to determine tolerance, puncture pressure, shape, and average weight.

The screening method used to evaluate tolerance to *R. solani* was modified after that of Barksdale (4). Instead of using a greenhouse soil bed, a metal flat was used to hold the soil. The strain of *R. solani* used was isolated from an infected tomato fruit collected at Jackson Springs, North Carolina. Inoculum was grown on sterilized oat grain and incubated for 2 weeks at 24°C before incorporation into the sterile soil flat (1.6 liters/m^2). The flats were placed in a dark room at 29° with a relative humidity of 95%.

Ten fruit per plant were placed on the soil surface without allowing the blossom end or the stem scar to come in contact with the soil. After 9 days, the fruit were turned to determine the percent infection. Any fruit showing a lesion of 0.5 cm was considered infected. The surface area infected was not determined but has been shown to be highly correlated to percent infection (4). Lesions were of 2 types: type 1 lesions caused by direct penetration through the cuticle, and type 2 lesions developed through the stem scar. The total infection was calculated as the sum of both types of lesions.

Puncture pressure was measured with an Instron Universal Tester. Five fruit from each plant were tested after maturing for 7 days at 24°C. Each fruit was punctured midway between the blossom and stem end by a needle moving at a constant speed of 0.5 cm/min. A recording of the force needed to puncture the initial epidermal barrier and the time taken were made. A subjective rating of fruit shape was made within the segregating populations of the A family ($P_1 \times P_3$). The oblong shape of P_1 was rated as 1.0, while the round, globed shape of P_3 was rated 5.0. The average weight of fruit from each plant was determined.

The data were anlayzed be computing the plant to plant variance of each population within the family. Methods of statistical and genetic analysis were similar to Allard (1). Heritability estimates were made using the method proposed by Warner (21). The estimate of the minimum number of factors involved was taken from Mather and Jinks (14). Estimates of the genetic parameters were calculated as follows:

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$$\text{Environmental variance (E)} = \frac{(V_{\text{susc. parent}} + V_{F_1} + V_{\text{res. parent}})}{3}$$

$$\text{Additive variance } (\tfrac{1}{2}D) = 2\,V_{F_2} - (V_{BC\text{-res.}} + V_{BC\text{-susc.}})$$

$$\text{Dominance variance (H)} = V_{F_2} - (E + \tfrac{1}{2}D)$$

$$\text{Heritability } (h^2) = \tfrac{1}{2}D/V_{F_2}$$

$$\text{Minimum no. factors (N)} = (\tfrac{1}{4})\,(d^2/\tfrac{1}{2}D);\ d = \text{diff. in parental means}$$

$$\text{Predicted gain (Gs)} = (k\ .05)\,(\sqrt{V_{F_2}})\,(h^2)$$

Results and Discussion

'Campbell-28' had the highest incidence of infection and the lowest puncture pressure, while the tolerant parents (P_1 and P_2) had the lowest levels of infection and the highest puncture pressure (Tables 1 and 2). The mean values of all populations within the A family were different for percent fruit infected except between the F_1 and F_2 (Table 1). The mean values for puncture pressure also differed except between the F_1 and the backcross to P_1. There was a slight heterosis in the F_1 for percent infection and puncture pressure.

Within the A family, the F_2 had the highest intraplot variance for percent infection and puncture pressure. The additive variance for percent infection and puncture pressure was high, reflecting a high variance in the F_2 and moderate variances within the backcross populations. The dominance component was negative for both the variables but the value was assumed to be zero.

The heritability for percent infection and puncture pressure within the A family was relatively high. The estimated number of genes controlling the inheritance was one for percent infection and puncture pressure. The predicted gain reflects the expected shift in the mean of the F_3 after selecting the top 5% of the resistant F_2s. In the A family the selection would be expected to change the mean percent infection from 48 to 32.

The percent infection and puncture pressure means within the B family. (P_2 by P_3) were different for all populations except between the F_1 and the backcross to P_3 (Table 2). The lack of difference between F_1 and the backcross of P_3 corresponded to the expression of heterosis of the F_1 toward P_3.

The intraplot variances in the B family for percent infection were higher for the F_2 followed by the backcross variances. The variances associated with puncture pressure showed a different distribution. The highest variance within puncture pressure was the tolerant parent P_2 suggesting that the line was not homozygous for this character or that the environmental influence was very strong. Correspondingly, the variance of the backcross to P_2 was also high.

The genetic parameters for percent infection in the B family showed a high environmental variance and a low additive component. The dominance component was near zero. The heritability of percent infection within the B family was relatively low and 4 gene pairs were estimated. The expected gain in the mean value of the F_3 after selection of the top 5% of the F_2s was estimated at 12%, thus the predicted F_3 mean would be 42%.

The environmental variance for puncture pressure within the B family was also high but there was a correspondingly strong additive component. This was reflected in a relatively high heritability. The dominance component for puncture pressure was considered to be zero and the estimated number of gene pairs involved was two.

A test of the 1-gene model for the A family indicated a proper fit of the F_2 and the backcross populations from the base data set of the non-segregating populations (17). The frequency distribution of this family (Fig. 1) showed only

Table 1. Means and SE, intraplot variances, and estimates of genetic parameters for percent fruit infected, puncture pressure, and shape from genetic populations of susceptible and tolerant tomato lines, C-28 and USDA 75B 846-1-1, respectively for family A.

Variable	No. of plants	Fruit infected (%)	Avg puncture pressure (g)	Shape[z]
Population means ± SE				
P_3 (C-28)	112	66.8 ± 1.6	12.05 ± .17	5.00 ± .00
BCP_3	95	57.7 ± 2.0	14.26 ± .20	4.30 ± .06
F_1	64	49.5 ± 2.5	16.22 ± .23	2.91 ± .06
F_2	219	47.8 ± 1.5	15.07 ± .18	2.54 ± .07
BCP_1	86	40.8 ± 1.8	16.17 ± .21	1.67 ± .05
P_1 (USDA 75B 846-1-1)	86	26.1 ± 1.5	17.02 ± .26	1.00 ± .00
Midparent value (MP)		46.5	14.535	3.0
Deviation of F_1 from MP		+3.0	1.685	−.09
Heterosis (%)		6.5	11.6	3.0
Intraplot variance				
P_3 (C-28)		0.027	3.13	
BCP_3		0.036	3.78	
F_1		0.040	3.48	
F_2		0.049	7.21	
BCP_1		0.027	3.65	
BCP_1 (USDA 75B 846-1-1)		0.019	5.59	
Estimates of genetic parameters				
Environmental variance	(E)	0.029	4.07	
Additive variance	(½D)	0.035	6.99	
Dominance variance	(H)	−0.015	−3.85	
Heritability	(h^2)	0.71	0.97	
Minimum no. factors	(N)	1.18	0.89	
Predicted gain	(G_s)	32.4%		

[z]Subjective rating: 1 = oblong, 5 = round.

Table 2. Means and SE, intraplot variances, and estimates of genetic parameters for percent fruit infected and puncture pressure from genetic populations of susceptible and tolerant tomato lines, C-28 and USDA 75B 610-3, respectively for family B.

Variable	No. of plants	Fruit infected (%)	Avg puncture pressure (g)
Populations means ± SE			
P_3	112	66.8 ± 1.6	12.05 ± .17
BCP_3	47	55.1 ± 2.5	13.47 ± .24
F_1	45	58.0 ± 2.5	13.42 ± .19
F_2	156	47.7 ± 1.5	14.79 ± .18
BCP_2	86	39.1 ± 1.9	15.29 ± .22
P_2 (USDA 75B 610-3)	72	24.6 ± 2.0	17.51 ± .29
Midparent value (MP)		45.2	14.8
Deviation of F_1 from MP		12.8	−1.4
Heterosis (%)		28.3	−9.2
Intraplot variances			
P_3 (C-28)		0.029	3.13
BCP_3		0.029	2.73
F_1		0.028	1.58
F_2		0.035	5.01
BCP_2		0.029	4.16
P_2 (USDA 75B 610-3)		0.016	6.17
Estimates of genetic paremeter			
Environmental variance	(E)	0.024	3.63
Additive variance	(½D)	0.010	3.14
Dominance variance	(H)	0.000	−1.75
Heritability	(h^2)	0.30	0.63
Minimum no. of factors	(N)	4.20	2.41
Predicted gain	(G_s)	11.7%	

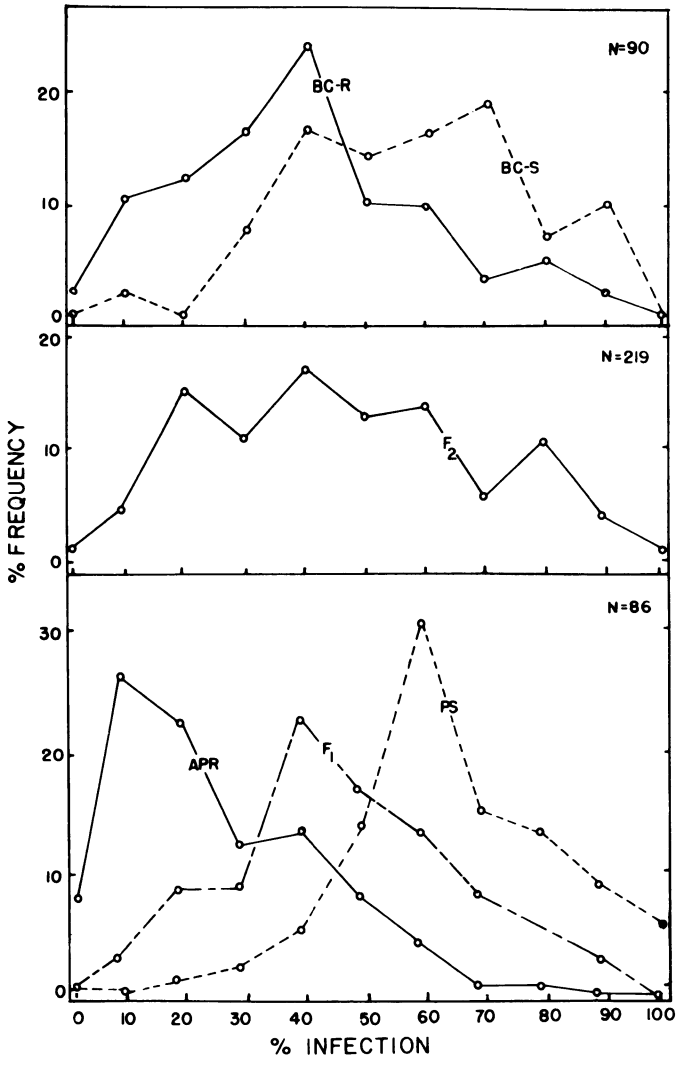

Fig. 1. Frequency for percent infection of F_1, F_2, backcross, and parental populations of A family.

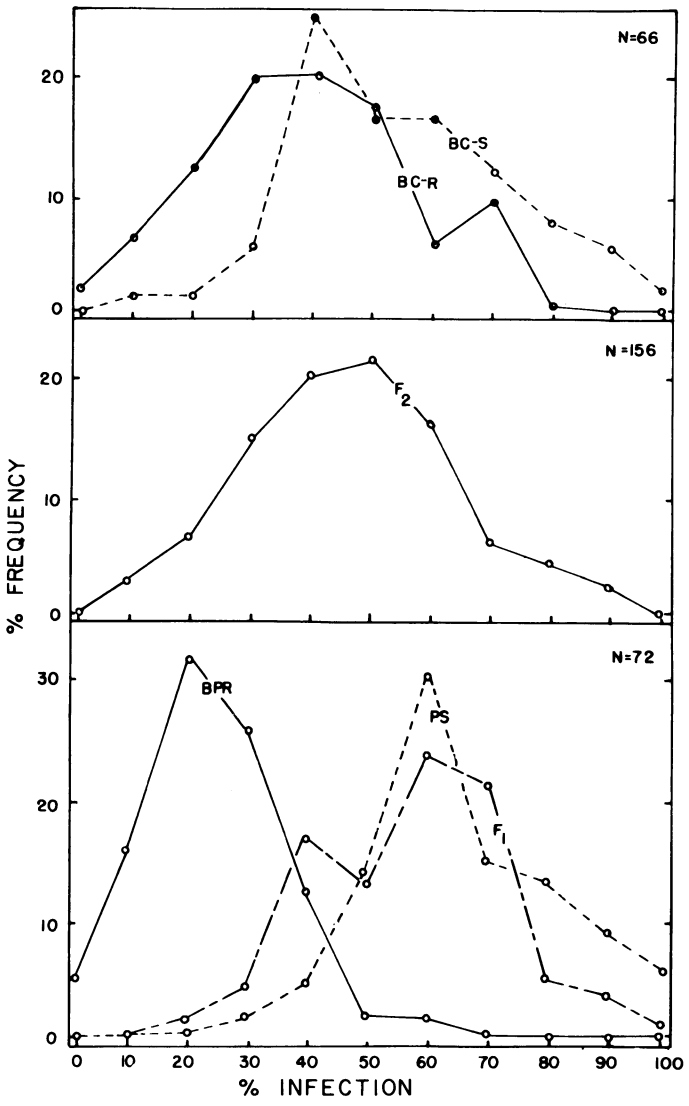

Fig. 2. Frequency for percent infection of F_1, F_2, backcross, and parental populations of B family.

indistinct segregation of the F_2 suggesting a sizable effect of the environment or a more complex inheritance model. The F_1 population fell midway between the susceptible (P_3) and the resistant (P_1) parents suggesting a lack of dominance.

The frequency distribution for the B family (Fig. 2) supported the evidence of low heritability and polygenic inheritance because the normal distribution found in the F_2 population. Such a distribution in the F_2 could be due to the relatively high environmental variance and the additive effect of the four genes. Both factors would tend to produce a normal distribution of the F_2. The F_1 showed a shift toward the susceptible parent (P_3).

Both families supported the conclusion that 1 or 2 genes control the expression of puncture pressure. This measurement may have reflected cuticle thickness, cell turgor, strength of the epidermal cell wall, or some other character of the epidermal layer (13). The original parents of this study were not selected for their resistance to puncture pressure. Thus, the strong negative correlation between incidence of infection and puncture pressure tends to substantiate the theory of a mechanical means of penetration by *R. solani* (Table 3). The r values for type 1 lesions were higher than for total infection, further supporting the importance of the epidermal layer as a barrier. The correlation between fruit shape and tolerance suggests the possibility of linkage between shape and the nature of the epidermis.

The use of USDA 75B 846-1-1 (P_1) in a breeding program would be favored since this tolerant source seems to be controlled by one gene with a high heritability while USDA 75B 610-3 (P_2) seems to be polygenic with a low heritability. The expected gain from F_2 to F_3 after selection is 32% with P_1 compared to 12% with P_2. Although P_1 has smaller fruit size and the vine habit is presently unacceptable, these traits can be easily manipulated. P_1 has the added advantage of field tolerance to tomato fruit worm (field observation).

There is a possibility of using puncture pressure or shape to facilitate the selection procedure and thus enable the breeder to handle larger populations of plants. The use of puncture pressure as a screening method must be viewed with caution since the actual mechanism of tolerance is not clearly understood. The consideration that fruit size if related to tolerance is removed since the correlation between fruit weight and resistance within P_3, which has a variable fruit weight (22 to 110 g), is near zero (r = .02). The actual mechanism of tolerance must pertain to the nature of the epidermis and its interaction with the fungus. If the mechanism of tolerance is mechanical, the precise barrier must be determined. The possibility that

Table 3. Correlation coefficients between type 1 infection, total infection, puncture pressure and shape within parents, whole family, and segregating populations of family A and family B.

Infectious type	Correlation coefficient (r^2)								
	Total infection			Puncture pressure			Shape		
	Parents	Family	Segregates	Parents	Family	Segregates	Parents	Family	Segregates
Type 1 Family A	0.87[z]	0.79	0.77	−0.67	−0.43	−0.25	0.85	0.55	0.37
Type 1 Family B	0.92	0.82	0.77	−0.69	−0.50	−0.36			
Total Family A				−0.58	−0.33	−0.19	0.75	0.42	0.22
Total Family B				−0.69	−0.47	−0.30			

[z]All values significantly different from r = 0 at 1% level.

the mechanism of tolerance is biochemical remains open. If the nature of tolerance is elucidated, then a more precise screening technique may be developed.

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