PHYTOPATHOLOGICAL NOTES

Additional Genes for Resistance to Pseudomonas solanacearum in Solanum phureja

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ABSTRACT

Certain clones of Solanum phureja have high levels of resistance to Pseudomonas solanacearum in growth chamber tests. Ten hybrid families were tested under growth chamber conditions by inoculation with a race 1 isolate of P. solanacearum (S-123). The genetic hypothesis that best fits the observed resistant-susceptible ratios requires that three dominant and independent genes provide resistance. One of these genes is required to give resistance to another race 1 isolate, K-60. Resistance apparently is controlled by relatively few genes in a system that is partially interrelated yet specific for certain strains of the bacterium.

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Clones of the cultivated diploid potato, Solanum phureja Juz. & Buk., differ in their reaction to inoculation with different isolates of Pseudomonas solanacearum E. F. Sm. (3). Clones which are resistant to one isolate may be susceptible to isolates from other geographical areas. Resistance to a race-1 isolate of P. solanacearum (K-60), under growth chamber conditions, is controlled by dominant genes at three independent loci (2). To obtain additional evidence on the inheritance of resistance, tests were conducted with hybrid clones of S. phureja inoculated with a more virulent isolate of the bacterium.

The parental S. phureja clones originated from open-pollinated seed provided from the Coleccion Central Colombiana by H. David Thurston. Based on the reaction of these clones to isolates of P. solanacearum (3), crosses were made to produce resistant (R) X susceptible (S), R X R, and S X S combinations. Usually, 100 plants of each cross were grown from true seed in a growth room at 21 C and a 14-hr photoperiod from a combination of Sylvania

Gro-Lux and cool-white fluorescent and incandescent lamps that provided 2,000 ft-c. At approximately the prebud stage, the individual plants were inoculated by the stem-inoculation procedure (3) with a cell suspension (108 cells/ml) of a race-1 isolate (S-123) from Eupatorium odoratum L. prepared from the 48-hr growth at 30 C on a tetrazolium medium (1). After inoculation, the plants were transferred to a growth chamber held at $28 \, \text{C}$, $70 \pm 3\%$ relative humidity, and 2,000 ft-c on a 14-hr photoperiod. After 15 days, the plants were scored according to the disease index scale (ranging from 1.0 = no infection to 5.0 = complete wilting) described previously (3). Those plants, rated 1.0 to 2.5, were classified as resistant. The methods have been described in detail previously (2, 3).

Genotypes were assigned to the parents based on the ratios of resistant to susceptible plants that were obtained with each family (Table 1). As in the case of the K-60 isolate, a hypothesis involving dominant genes for resistance at three independent loci appeared to provide the best explanation for the observed ratios. This model gave good agreement between the observed and the expected ratios for the majority of the crosses tested. It was also noted that in two of the three progenies inoculated with both K-60 and S-123, a higher proportion of the individual clones was resistant to S-123 than to K-60. This suggested that resistance to these two isolates was inherited in an independent rather than an additive manner.

Since completely independent inheritance systems for resistance to individual strains of the bacterium would not be expected, the results from the K-60 and S-123 studies were analyzed to determine how they related to each other. If a minor change in one of the genotypes proposed earlier (2) for resistance to K-60 (from RrAaBB to RRAaBb for clone 1386.26) is made, it can be demonstrated that the two models have one gene in common. For the purpose of the present working hypothesis, this common gene is designated as R. Although the data now available do not indicate that one gene has a greater effect than the others, it seems likely that the R gene is the "basic" gene for resistance, and that the other genes provide a certain degree of specificity.

If the genotype R A B provides resistance to K-60 and R D E to S-123, it should be possible to detect individual clones in a progeny that are resistant to one isolate but not to the other. To explore this possibility, a population from the cross 1386.26 X 1339.28 was inoculated in the small seedling stage with S-123. The seed was distributed in rows in flats (35 X 50 cm) containing Jiffy-Mix (Jiffy Pot Co., Chicago, Ill.), and seedlings were grown for 3 weeks before they were inoculated by flooding the soil with a suspension (106 cells/ml) of isolate S-123. The roots were wounded by cutting along the row with a knife immediately after flooding. Two weeks later, the survivors were transplanted to individual pots and allowed to grow to the prebud stage before they were stem-inoculated with either isolate S-123 or K-60. The fact that 33 of the 36 plants that survived root

TABLE 1. Observed and expected reaction of hybrid progenies of Solanum phureja to Pseudomonas solanacearum (isolate S-123) and proposed genotypes of parental clones

Cross	Proposed genotype	R:Sa Ratio	
		Expected	Observed
1386.12 (R) × 1339.28 (S)	Rr Dd Ee × rr Dd Ee	14:36	21:29
× 5536.7 (S)	X rr dd ee	12:88	11:89
× 1386.22 (R)	× RR Dd EE	75:25	85:15
1386.15 (R) × 1339.28 (S)	RR Dd Ee x rr Dd Ee	56:44	58:42
× 1386.22 (R)	× RR Dd EE	75:25	68:32
× 1386.26 (R)	× RR Dd Ee	75:25	65:35
1386.22 (R) × 1339.28 (S)	RR Dd EE X rr Dd Ee	75:25	70:30
× 1386.26 (R)	X RR Dd Ee	75:25	76:24
1386.26 (R) × 1339.28 (S)	RR Dd Ee X rr Dd Ee	56:44	58:42
1339.28 (S) × 5536.7 (S)	rr Dd Ee X rr dd ee	0:100	0:100

a R = resistant; S = susceptible.

TABLE 2. Resistance to *Pseudomonas solanacearum* isolates K-60 and S-123 in potato plants of the cross 1386.26 (R) \times 1339.28 (S) that survived seedling root inoculation with S-123

No. plants tested		R:Sa Ratio	
	Isolate	Expected	Observed
27	K-60	6:21	11:16
36	S-123	36:00	33:03

a R = resistant; S = susceptible.

inoculation with S-123 as seedlings were resistant later to stem inoculation with the same isolate (Table 2) indicates that the seedling test was effective in eliminating most of the susceptible progeny. Since 16 out of 27 survivors were susceptible to K-60 by stem inoculation, the hypothesis of an independent rather than an additive mode of inheritance appears plausible.

In summarizing the results from the investigations on the inheritance of resistance to *P. solanacearum* in *S. phureja*, it is evident that relatively few genes are involved in a system that is at least partially interrelated yet apparently specific for certain strains of the bacterium. Studies with more pathogenic isolates may uncover new genes for resistance, but it is logical to anticipate that related groups of isolates will respond only to certain genes for resistance.

The results provide some guidance for efforts to

breed potato cultivars with resistance to P. solana cearum. The relatively simple inheritance patterns that have been found in S. phureja are encouraging because it should be reasonably simple to transfer this resistance into breeding stocks. However, in view of the highly variable nature of the pathogen, field evaluation and selection may have to be conducted at several geographical locations, and breeding will have to be done for specific areas. Any attempts to screen populations for resistance under artificial conditions probably should involve a mixture of isolates of the bacterium containing at least the dominant strain in the area of concern. Considering the field observations on the performance of potato clones with resistance derived from S. phureja that have been reported to us, the chances for control of bacterial wilt in the subtropical areas by means of resistant cultivars seem very good.

LITERATURE CITED

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