Components of Partial Resistance in Peanut Genotypes to Isolates of *Cercosporidium personatum* from the United States and Thailand

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ABSTRACT

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Leaves were detached from 14 peanut (*Arachis hypogaea*) genotypes that previously were characterized as having low, moderate, or high partial resistance to *Cercosporidium personatum*. Detached leaf cultures were inoculated with isolates of *C. personatum* from diverse locations within Thailand and the United States. Lesion numbers, the percentages of lesions that sporulated 20 (%LS20) and 30 (%LS30) days after inoculation, lesion diameters, and conidial production per sporulating lesion were determined. Thai isolates of *C. personatum* generally caused more lesions than U.S. isolates on all genotypes. Differences among isolates for other disease components were small and varied between two trials. Stability of resistance to several disease components was evaluated with regression analysis, in which low mean disease ratings, nonsignificant deviations, and slopes near zero in the regression indicated stable resistance. Significant slopes, which indicate increasing disease with increasing isolate virulence, occurred in lesion numbers, %LS20, %LS30, and conidial production for some genotypes. Few significant deviations, which indicate significantly higher disease in specific isolate × genotype combinations, were found among moderately and highly resistant genotypes. The three most resistant genotypes varied little in overall resistance but differed in stability of resistance. KUP 24D-248W, a genotype selected in Thailand, had the most stable resistance to the isolates tested. Differences within isolates and between trials were nearly as great as differences among isolates from the diverse locations.

Additional keywords: groundnut, host-pathogen interactions, leaf spot.

Cercosporidium personatum (Berk. and Curt.) Deighton causes late leaf spot of peanut (*Arachis hypogaea* L.). Chemical control is expensive and requires several sprays per season; without control, susceptible varieties worldwide suffer extensive losses.

Barriers to fertility in interspecific crosses of *Arachis* spp. have inhibited the incorporation of leaf spot resistance genes from wild species into cultivated peanuts (1,4), but large variations in partial resistance to *C. personatum* occur within *A. hypogaea*. Components of partial resistance of many genotypes have been characterized (2,12,16,19,20), and a cultivar with desirable agronomic characteristics and partial resistance to late leaf spot has been released in Florida (6).

Many investigators (2,12,16,19,20) have used detached leaves and greenhouse inoculations to characterize resistance to *C. personatum*. Leaves cultured in nutrient solution (11) or sand (2,16,19,20) live at least 2 mo if not inoculated. Genotype rankings by lesion diameter, spore production, and latent period on detached leaves are highly correlated with rankings by leaf spot ratings taken in the field (19,20).

Genotypes that have high partial resistance to *C. personatum* generally have performed similarly in tests conducted in diverse locations and growing seasons. Variation in expression of moderate resistance occurred in some genotypes in India and China (18), but interplot interference may have been partially responsible because no buffer rows were used. Qualitative resistance categories were imposed on continuously varying resistance data, which also may have exaggerated differences in genotype performance.

Greenhouse and field evaluations of peanut for partial resistance to *C. personatum* have been conducted with single isolates or with indigenous field populations of the pathogen, and little is known about variability among geographic populations of *C. personatum*. Isolates from Thailand and the United States are similar in conidial morphology and size, but on agar-coated glass

slides, Thai isolates germinate more readily at temperatures ranging from 16 to 36 C than U.S. isolates (17).

If isolates of *C. personatum* also differ in virulence on some partially resistant genotypes, progress in international cooperative projects to improve resistance to the pathogen would be impaired. The purpose of this research was to determine if isolates of *C. personatum* from Thailand or the United States exhibit pathogenic specialization on peanut genotypes with partial resistance to the pathogen.

MATERIALS AND METHODS

Leaves infected with *C. personatum* were collected in April and May 1985 from peanuts grown at each of two field locations in north, northeast, and south Thailand. Conidia on each leaf sample were collected by suction with a cyclone spore collector (ERI Machine Shop, Ames, IA) into small plastic capsules, and capsules were mailed to Raleigh, NC. Conidia also were collected from infected leaves from Texas, Alabama, Florida, and North Carolina in summer 1985. Dry conidia in capsules were stored at 4 C until they were used to establish new cultures. Potted plants of the peanut genotype NC 3033, which is susceptible to late leaf spot, were used to culture the test isolates. Conidia of each isolate (which originated from many lesions) were suspended in a mixture of 1 drop of Tween-80 per 100 ml of deionized water and were sprayed with an artist's airbrush onto plants. These stock plants were placed individually into plastic bags and incubated for 1 wk; plants inoculated with each isolate then were placed in separate isolation chambers in the greenhouse. Each isolation chamber consisted of a wooden frame covered with clear plastic and was humidified by wet burlap placed on the floor of the chamber. Maximum greenhouse temperatures ranged from 30 to 21 C; minimum temperatures ranged from 26 to 17 C.

Plants for experimental inoculations were grown in the greenhouse in 15-cm-diameter pots that contained a mixture of two parts steamed sandy loam soil and one part (v:v) steamed builder's sand. A small amount (about 20 mg) of a commercial rhizobium inoculant (cowpea group; Keel Peanut Company, Greenville, NC) was sprinkled in each pot. Eight genotypes were characterized previously in field trials in Thailand as very susceptible (Tainan 9, SK 38), moderately susceptible (KUP 24D-080 and KUP 24D-084), moderately resistant (KUP 24D-421 and KUP 24D-615), or highly resistant (KUP 24D-448 and KUP 24D-248W) to late leaf spot. Six additional genotypes were standards that represent high resistance (PI 259747, NC Ac 17133 RF), moderate resistance (GP-NC 343), and susceptibility (NC 3033, Robut 33-1, FESR 5-P2-B1) to late leaf spot in greenhouse and field trials in North Carolina.

Leaves at nodes three and four (in relation to the shoot terminal) were excised from 8- to 12-wk-old plants and their petioles were placed in 75-ml-capacity plastic beakers (2,16). Beakers were filled with steamed, washed builder's sand, and the sand was moistened with deionized water. Leaves in beakers were held in a mist chamber in the greenhouse for 1 wk before inoculation. The mist chamber had a plastic cover that prevented direct wetting of leaves by the misting nozzles. Misters operated 10 sec/10 min during the day and 15 sec/hr at night.

For experimental inoculations, conidia from large ($\geq$ 10 mm^2), sporulating lesions of each test isolate were collected from infected leaves on stock plants. These leaves were immersed in deionized water plus surfactant (1 drop of Tween-80 per 100 ml of water), and conidia were removed from lesions with gentle brushing. Conidial suspensions were diluted to 20,000 conidia/1 ml of water (2,16,20) and applied with an artist's airbrush operated at 48 kPa air pressure. About 0.3 ml of the conidial suspension was used for each leaf. Inoculated leaves were returned to mist chambers for the duration of the experiment. Maximum greenhouse temperatures ranged from 40 to 27 C; minimum temperatures ranged from 26 to 17 C.

Two replicate sets of two experiments were conducted from September to December 1985 in separate trials. In the first experiment, four leaves per replicate of each of the 14 genotypes were inoculated with each of the six Thailand, the Florida, and the North Carolina isolates of *C. personatum*. In the second experiment, three Thai isolates (one from each region) and the Alabama, North Carolina, Texas, and Florida isolates were individually inoculated onto four leaves of the 14 genotypes in each replication.

Lesions and sporulating lesions were counted 10, 20, and 30 days after inoculation. The four largest lesions in each replication of each treatment were measured, and these lesions were excised with a cork borer. Excised lesions and a micro-stir rod were placed in a small (1-ml-capacity) vial that contained 0.5 ml of deionized water plus surfactant and agitated 80 sec. Conidia in the suspension were counted with a hemacytometer.

The experimental design was a split plot with isolates as whole plots. Genotypes were randomized within isolates for each replication. Data from the four leaves in each treatment and replication were averaged before analysis. Lesion numbers were transformed by square roots, and the percentage of lesions that sporulated (%LS) was transformed by arcsine square roots before analysis of variance and other analyses. Because analysis of variance with large error variances may be relatively insensitive to isolate specificity (7,13), probability levels ≤ 0.10 were considered significant for isolate $\times$ genotype interactions.

Some components of resistance also were subjected to stability analysis (5,7,10). A simple linear regression was calculated for each genotype such that the independent variable was an index of virulence for each isolate, calculated as the mean value of a disease component on all genotypes inoculated with that isolate (10). The dependent variable was the level of a disease component present on each genotype inoculated with each isolate. Therefore, specificity in a particular isolate and genotype combination causes significant deviation from the regression (10). Slopes significantly greater

TABLE 1. Mean disease components of 14 peanut genotypes inoculated with isolates of *Cercosporidium personatum* from the United States and Thailand[a,b]

Isolate origin	%LS20	%LS30	Lesion number	Lesion diameter	Number conidia
Trial one					
Thailand					
1 North-1	23.5	39.2	7.5	2.5	3.9
2 North-2	20.2	43.4	6.4	2.6	7.9
3 South-1	32.1	35.2	11.7	2.5	5.2
4 South-2	22.9	39.4	8.9	2.3	3.7
5 NE-1	17.9	38.4	6.9	3.0	7.9
6 NE-2	11.1	39.1	6.5	2.6	4.9
United States					
8 FL	26.4	45.3	4.3	2.5	5.4
10 NC	13.3	48.5	2.8	2.7	3.8
LSD	15.3	19.2	5.2+	0.6	5.8
CV(%)	52.2	36.1	29.2	21.6	82.8
Trial two					
Thailand					
1 North-1	12.8	40.5	4.7	2.6	3.8
3 South-1	11.1	33.0	4.7	2.3	4.0
5 NE-1	17.3	33.2	4.3	2.8	3.4
United States					
7 AL	4.9	31.6	3.7	2.2	3.5
8 FL	7.0	40.6	1.1	1.9	2.3
9 TX	10.6	34.1	1.2	1.9	3.3
10 NC	8.1	35.8	1.7	2.3	3.4
LSD	7.5+	12.8	3.7	0.6*	3.8
CV(%)	134.2	39.2	36.0	23.9	87.9

[a] Disease components are arcsine $\sqrt{\%LS20}$, arcsine $\sqrt{\%LS30}$ (percent of lesions that sporulated 20 and 30 days after inoculation, respectively), $\sqrt{\text{lesion number per leaf on day 20}}$, mean diameter of four largest lesions in millimeters, and number of conidia per lesion $\times$ 1,250.

[b] Significant F-tests for isolate main effects are indicated by + ($P \leq 0.10$) or * ($P \leq 0.05$). All other tests were not significant. Least significant difference (LSD) for any nonsignificant effect is intended as a measure of experimental error only and should not be used to compare means.

than zero indicate that the genotype is sensitive to increasing isolate virulence; as more virulent isolates are used, the level of a disease component on that genotype increases (10). Genotypes possessing stable resistance for a given disease component should have less mean disease than other genotypes and stability regressions with slopes near zero and nonsignificant deviations.

TABLE 2. Contrasts of mean disease on 14 peanut genotypes inoculated with isolates of *Cercosporidium personatum* from Thailand and the United States[a,b]

Disease component	Mean of Thai isolates ($\bar{X}_T$)	Mean of U.S. isolates ($\bar{X}_U$)	$\bar{X}_T - \bar{X}_U$
Trial one			
%LS20	21.3	19.9	1.5
%LS30	39.2	46.9	−7.7+
Number of lesions	8.0	3.6	4.5**
Lesion diameter	2.6	2.6	0.0
Number of conidia	5.6	4.6	1.0
Trial two			
%LS20	13.7	7.7	6.0**
%LS30	35.7	35.5	0.2
Number of lesions	4.6	1.9	2.7**
Lesion diameter	2.6	2.1	0.5+
Number of conidia	3.7	3.1	0.6

[a] Disease components are arcsine $\sqrt{\%LS20}$, arcsine $\sqrt{\%LS30}$ (percent of lesions that sporulated 20 and 30 days after inoculation), $\sqrt{\text{lesion number per leaf on day 20}}$, mean diameter of four largest lesions in millimeters, and number of conidia per lesion × 1,250.

[b] Indicated differences ($\bar{X}_T - \bar{X}_U$) are significant at $P \leq 0.10$ (+) or $P \leq 0.01$ (**), according to linear contrasts of Thai (T) versus U.S. (U) isolates in analysis of variance.

RESULTS

Disease caused by individual isolates of *C. personatum* generally varied continuously (Table 1), and there were few significant differences in disease components among individual isolates. Isolates from Thailand on the average caused more lesions and, in the second trial, larger lesions than U.S. isolates (Table 2). A greater proportion of lesions caused by Thai isolates sporulated by 20 days after inoculation (%LS20), but by day 30, the proportion of lesions that sporulated (%LS30) for U.S. isolates equaled or exceeded %LS30 for Thai isolates (Table 2).

According to analysis of variance of data from each trial, for the isolates common to both trials, isolate × genotype interactions occurred only for %LS20 ($P < 0.01$) and lesion number ($P \leq 0.10$) in trial one. When data from the two trials were combined in a single analysis, these interactions were no longer significant at $P \leq 0.10$. Because genotype performance varied in the two trials for three of five disease components (genotype by trial significant), data are presented separately for each trial.

Apparent isolate specificity in %LS20 in trial one is illustrated in Figure 1. FESR 5-P2-B1, 084, 421, and 448 were susceptible to moderately resistant (larger %LS20) to *C. personatum* and did not discriminate among pathogen isolates (not shown). The

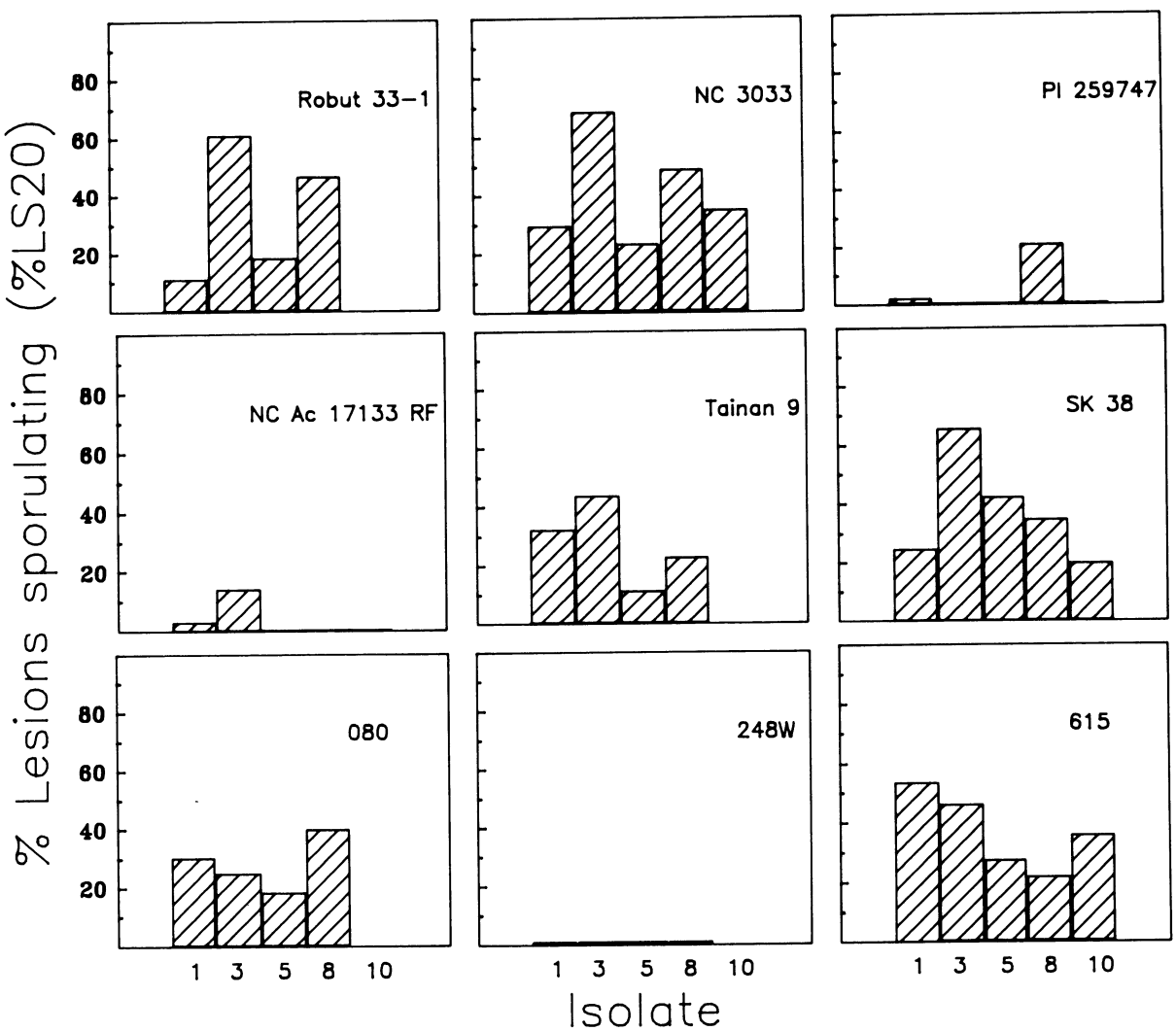

Fig. 1. Percent of lesions that sporulated 20 days after inoculation (%LS20) on selected peanut genotypes with partial resistance to *Cercosporidium personatum* in trial one. Isolates 1, 3, and 5 were from north, south, and northeast Thailand, respectively. Isolate 8 was from Florida, and isolate 10 was from North Carolina. Values are arcsine transformed means of four observations per two replications. Least significant difference = 22%.

susceptible to moderately resistant genotypes NC 3033, Robut 33-1, Tainan 9, and SK 38 had the greatest %LS20 when inoculated with isolate 3 (Thai S-1), but %LS20 was greatest on 615 when it was inoculated with isolate 1 (Thai N-1) or isolate 3 (Fig. 1). In trial one, isolate 8 (FL) had a greater %LS20 than the other isolates on 080 and also on PI 259747, which has high partial resistance (low %LS20) to *C. personatum*. No isolate differences occurred on NC Ac 17133 RF, which also has high partial resistance, and no isolate sporulated on 248W by 20 days after inoculation in this trial.

Although the isolate × genotype interaction for the percentage of lesions that sporulated was no longer significant by 30 days after inoculation, %LS was still larger on leaves of PI 259747 inoculated with isolate 8 than with other isolates in trial one. In general, isolate differences decreased as percentage of lesions that sporulated increased between 20 to 30 days after inoculation (Fig. 2).

Sporulation began later in trial two than in trial one, resulting in less discrimination among isolates by %LS20 in the second trial. None of the specific combinations of isolate and genotype that resulted in larger %LS20 in trial one were significant in trial two, and no significant specificity was detected on the three most resistant genotypes for %LS20 or %LS30 in trial two.

Stability analysis. Mean lesion number per leaf (transformed) for all host genotypes and isolates was 6.9 in trial one and 3.0 in trial two. Four genotypes, 080, FESR 5-P2-B1, 421, and GP-NC 343, had low mean lesion numbers in both trials (Table 3). SK 38, 448, and NC Ac 17133 RF had few lesions in only one of the two trials. None of the deviations from regressions in the stability analyses of lesion number were significant for any genotype, but all slopes of the regressions of lesion number caused by each isolate versus the virulence index for each isolate (mean lesion number on all genotypes inoculated with that isolate) were significantly greater than zero (Table 3).

In trial one, an average of 21% of lesions on all genotypes sporulated on day 20. In trial two, 10% of lesions sporulated by day 20. The genotypes 248W, NC Ac 17133 RF, PI 259747, and 084 had relatively few lesions sporulating by 20 days after inoculation in trial one, but in trial two, 084 was not resistant (Table 4). Regression of %LS20 for each isolate on the virulence index for each isolate (mean %LS20 on all genotypes inoculated with that isolate) had a highly significant deviation mean square for 084 in trial two because isolate 9 (from Texas) was more virulent than average on this genotype (Fig. 3). In trial two (but not in trial one), GP-NC 343, 080, and 421 had low mean %LS20. The trial-to-trial differences in performance of GP-NC 343, 080, and 421 were not related to deviations from regressions of %LS20 on virulence index for these moderately resistant genotypes; all deviations were nonsignificant. Slopes of the regressions were greater than zero, however (Table 4), indicating that %LS20 on these genotypes was sensitive to differences in virulence among isolates.

Only PI 259747 satisfied the criteria for stable resistance to %LS20 (low mean %LS20, nonsignificant slopes and deviations in stability regressions) in both trials. Genotype 248W satisfied the criteria in trial one, and NC Ac 17133 RF failed to meet the criteria in both trials. Slopes of the regressions involving NC Ac 17133 RF were significantly greater than zero in both trials (Table 4). The small ($b = 0.1$) slope for 248W also was greater than zero in trial

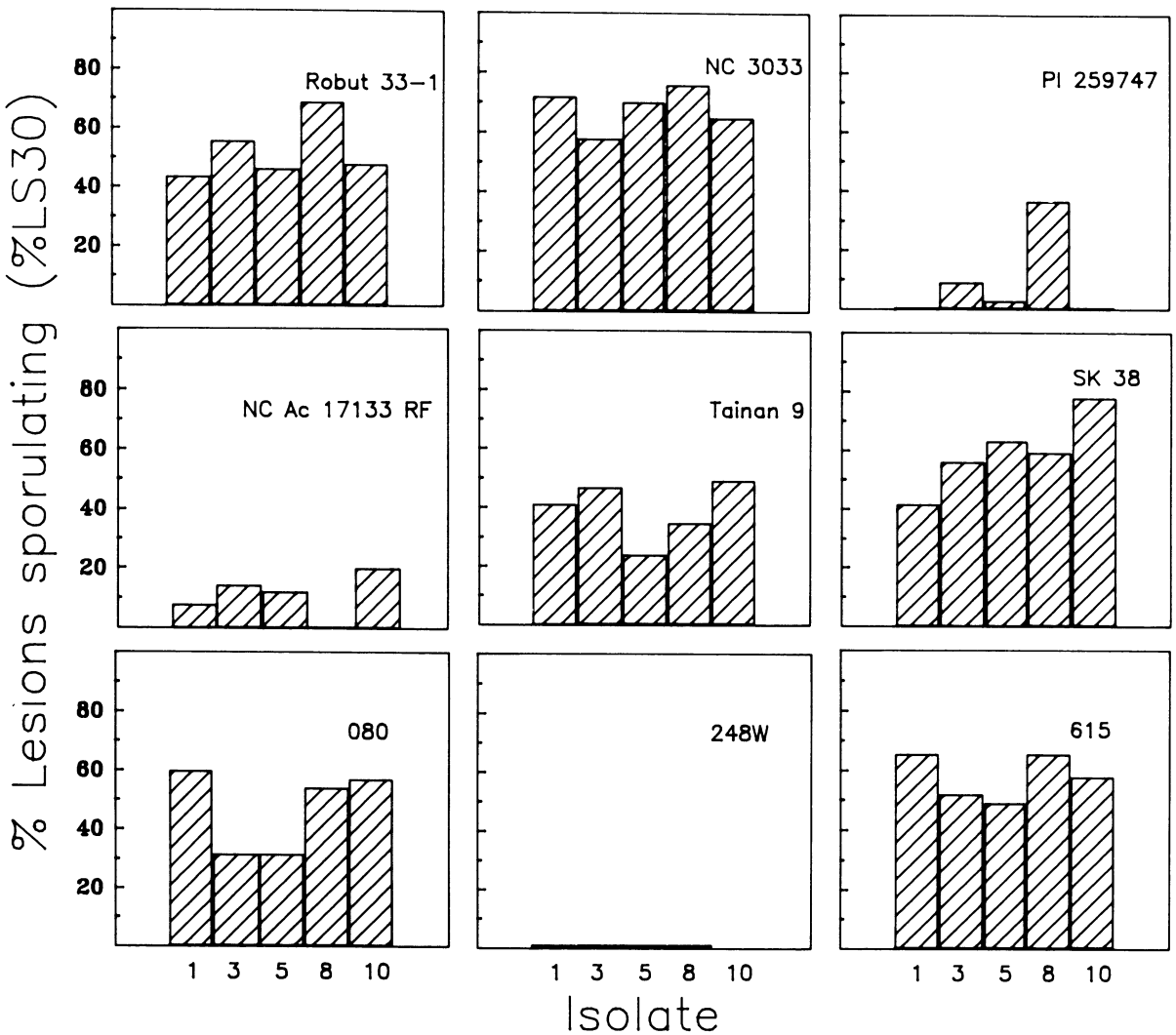

Fig. 2. Percent of lesions that sporulated 30 days after inoculation (%LS30) on selected peanut genotypes with partial resistance to *Cercosporidium personatum* in trial one. Isolates 1, 3, and 5 were from north, south, and northeast Thailand, respectively. Isolate 8 was from Florida, and isolate 10 was from North Carolina. Values are arcsine transformed means of four observations per two replications. Least significant difference = 27%.

two ($P \leq 0.10$, Table 4). On both genotypes, %LS20 increased with increasing isolate virulence.

Although deviations from regression of %LS20 on the virulence index were nonsignificant for the three genotypes with the lowest %LS20 in both trials (Table 4), deviations were relatively large for PI 259747 in trial one. Plots of %LS20 versus the virulence index for this genotype showed that isolate 8 caused the large deviation from regression, as previously seen with isolate × genotype means (Fig. 1). In the second trial, however, isolate 8 had no specificity for PI 259747.

GP-NC 343 and 084 continued to express moderate resistance to lesion sporulation 30 days after inoculation. PI 259747, NC Ac 17133 RF, and 248W continued to express high resistance relative to the average %LS30 of 41% in trial one and 36% in trial two.

TABLE 3. Stability analysis of mean lesion number on selected peanut genotypes inoculated with *Cercosporidium personatum*[a]

Genotype	Mean	Deviation mean square	Slope of regression
Trial one			
080	5.0	1.2	0.9**
FESR 5-P2-B1	5.4	2.9	1.1**
421	5.8	0.5	1.0**
GP-NC 343	5.9	0.6	0.8**
SK 38	5.3	5.6	0.8**
448	5.9	2.9	1.1**
NC Ac 17133 RF	9.8	10.4	1.0**
Error mean square (111 df) = 8.0			
Trial two			
080	2.5	0.2	0.8**
FESR 5-P2-B1	2.8	0.4	1.1**
421	2.7	1.0	0.8**
GP-NC 343	2.2	0.4	0.9**
SK 38	3.8	0.8	1.0**
448	3.2	1.3	1.0**
NC Ac 17133 RF	2.5	0.5	1.0**
Error mean square (97 df) = 3.1			

[a] Lesion numbers for each genotype were transformed by square roots and regressed on the average transformed lesion number produced by each isolate. Deviation from regression had 6 df in trial one and 5 df in trial two. All slopes were significantly different from zero at $P \leq 0.01$ (**).

TABLE 4. Stability analysis of mean percentage of lesions that sporulated on day 20 (%LS20) on selected peanut genotypes inoculated with *Cercosporidium personatum*[a]

Genotype	Mean	Deviation mean square	Slope of regression
Trial one			
248W	0.0	...	...
NC Ac 17133 RF	2.0	25.7	0.5**
PI 259747	4.0	187.7	0.4
084	7.7	53.0	0.1
GP-NC 343	17.3	79.2	−0.1
080	24.1	237.5	1.3**
421	23.1	74.8	1.2*
Error mean square (111 df) = 148.8			
Trial two			
248W	0.2	0.4	0.1+
NC Ac 17133 RF	1.9	25.0	0.9+
PI 259747	0.0	...	...
084	14.0	573.0*	0.9
GP-NC 343	4.6	18.7	1.5*
080	6.5	51.4	1.5*
421	2.7	7.3	0.8*
Error mean square (97 df) = 185.5			

[a] %LS20 for each genotype was transformed by arcsine $\sqrt{\%LS20}$ and regressed on the average transformed %LS20 for each isolate. Deviations from regression had 6 df in trial one and 5 df in trial two. Deviations and slopes were significantly different from zero at $P \leq 0.10$ (+), $P \leq 0.05$ (*), and $P \leq 0.01$ (**).

Regressions of %LS30 on the virulence index for this component had no significant deviations for any of the moderately or highly resistant genotypes in either trial, and the only regression for these genotypes with a slope significantly greater than zero was for 084 in trial one ($P \leq 0.05$, Table 5).

Mean number of conidia per lesion for all host genotypes and isolates was 5.3 in trial one and 3.4 in trial two. Four genotypes met the criteria for stable resistance to conidial production, expressed as conidia per lesion. Lesions on PI 259747, NC Ac 17133 RF, 248W, and 080 produced few conidia, and regression of conidia per lesion versus virulence index for these genotypes had no significant deviations or slopes (H_0:$b = 0$, Table 6). Two genotypes (GP-NC 343 and 421) appeared resistant in trial two but not in trial one. Slopes of the regressions of number of conidia on virulence index for these genotypes in trial one were significantly greater than zero, indicating a strong relationship between increasing virulence (particularly in isolates 2 and 5) and conidial production on these genotypes in trial one (Fig. 4).

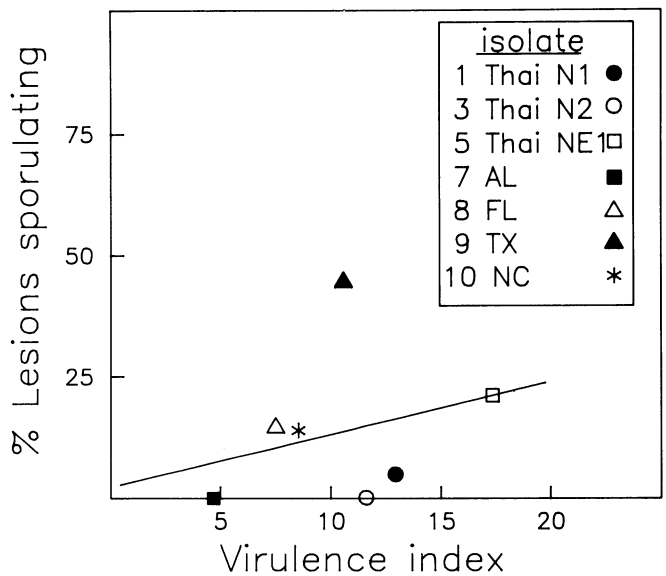

Fig. 3. Regression of percent of lesions that sporulated 20 days after inoculation (%LS20) with each of seven isolates of *Cercosporidium personatum* on virulence index (mean %LS20 for each isolate across all genotypes tested) for peanut genotype KUP 24D-084 in trial two. The deviation of isolate 9 was significant at $P < 0.05$.

TABLE 5. Stability analysis of mean percentage of lesions that sporulated on day 30 (%LS30) on selected peanut genotypes inoculated with *Cercosporidium personatum*[a]

Genotype	Mean	Deviation mean square	Slope of regression
Trial one			
248W	0.4	2.3	0.0
NC Ac 17133 RF	9.6	134.4	−0.1
PI 259747	6.6	351.1	0.7
084	32.1	70.9	2.5*
GP-NC 343	35.1	144.4	0.6
Error mean square (111 df) = 263.9			
Trial two			
248W	6.7	211.1	−0.2
NC Ac 17133 RF	13.2	92.1	−1.0
PI 259747	3.9	25.5	−0.5
084	33.1	104.9	0.1
GP-NC 343	29.2	181.9	1.5
Error mean square (97 df) = 206.0			

[a] %LS30 for each genotype was transformed by arcsine $\sqrt{\%LS30}$ and regressed on the average transformed %LS30 for each isolate. Deviation from regression had 6 df in trial one and 5 df in trial two. Slopes were significantly different from zero at $P \leq 0.05$ (*).

DISCUSSION

Several investigators have shown that significance of isolate × genotype interactions in analysis of variance is a poor indicator of specificity between parasite and host (3,8,13). Stability analyses of components of late leaf spot severity on peanut were more sensitive than analysis of variance and more precisely identified causes of interaction. For example, analysis of variance showed no significant isolate specificity for lesion number in trial two and significance at only $P \leq 0.10$ in trial one. Plots of lesion numbers and rankings of genotypes by lesion numbers in trial one did not clearly identify the source of the interaction. Stability analysis showed that no specific isolate and genotype combination caused significant deviations but that all genotypes in both trials had more lesions when they were inoculated with more virulent isolates of *C. personatum*.

For all disease components and genotypes where instability was identified in both trials, the cause was a significant relationship between increasing isolate virulence and increasing disease. An ideal partially resistant genotype would express nearly equal levels of resistance across all pathogen genotypes; if there are large differences, resistant hosts may increase selection pressure for the most virulent pathogen genotypes.

As originally proposed, deviations from regression in stability analyses indicated specificity among isolates and host genotypes (7,10). In model host-parasite systems where some genes acted additively and some acted interactively (gene-for-gene), however, increasing slopes in stability analysis regressions were correlated with increasing numbers of genes for specific (gene-for-gene) virulence and resistance, and deviations from regression were not correlated with increasing specificity (8). Gene-for-gene specificity in partially resistant host-parasite systems does not necessarily cause instability, however. Some variation in gene-for-gene systems is masked by nonmatching virulence and resistance genes, whereas all genes are expressed in an additive system (13). Size of deviations from regressions might be more highly correlated with the relative effects of resistance and virulence genes than with specificity per se.

We detected significant deviations only twice: for %LS20 by isolate 8 on PI 259747 in trial one and for %LS20 by isolate 9 on 084 in trial two. Isolate 9 was used only in trial two. However, isolate 8 was used in both trials, and no specificity was found on PI 259747 in trial two. Because results of stability analyses are relative depending on the isolates and genotypes used (7), we reanalyzed the relevant data using only isolates common to both trials and obtained similar results. Environmental variations may have allowed expression of specificity by isolate 8 for %LS20 on PI 259747 in trial one, but not in trial two. Specificity in certain isolate and genotype combinations apparently is sensitive to subtle changes in environment in some pathosystems (9,15). Our experiments were concluded within 4 mo, but environment varied slightly between trials. Cooler temperatures probably accounted for the overall slower development of disease in the second trial. Relatively small differences in temperature and humidity can affect development of *C. personatum* on peanut (16) and might alter isolate and genotype specificities.

Isolate variability also could account for different results of stability analyses for PI 259747. All isolates originated from mass spore collections and therefore were genetically heterogeneous. Perhaps a component of isolate 8 that attacked PI 259747 was not present or was present at a much reduced frequency in trial two. A genotype with high specific virulence for PI 259747 might have been less fit relative to other component genotypes on the very susceptible NC 3033 stock plants used to culture isolates.

Latent periods were not explicitly measured in these experiments because of the large number of observations required. Our results show, however, that a single final reading of the percentage of lesions that sporulated (%LS30) could miss differences among isolates and genotypes. Isolate differences and instability of resistance were more easily detected by 20 days after inoculation than by 30 days. These differences could have significant epidemiological impact after several disease cycles.

Resistance to rapid lesion maturation and high rates of lesion sporulation (as measured by %LS20, %LS30, and conidia per lesion) shows more promise for increasing overall resistance to *C. personatum* than does resistance to infection (expressed as lesion number per unit of inoculum) (2,16,20). The instability of resistance to infection in all genotypes we tested also indicates that this component is of little value in varietal improvement.

PI 259747, NC Ac 17133 RF, and KUP 24D-248W consistently had high resistance to %LS20, %LS30, and conidial production, but results of regression analyses for these genotypes detected some instability in expression of resistance. Resistance in 248W appeared most stable, but the slope of the stability regression for %LS20 was significant at $P = 0.06$ in trial two. In the case of 248W, the joint requirements for small deviations in the stability regression and for slope not significantly different from zero may have been too restrictive because small deviations increase the likelihood that even small slopes will be declared significantly different from zero. Although significant, the slope of 0.1 was the

TABLE 6. Stability analysis of mean number of conidia produced per lesion on selected peanut genotypes inoculated with *Cercosporidium personatum*[a]

Genotype	Mean	Deviation mean square	Slope of regression
Trial one			
248W	0.1	0.1	0.0
PI 259747	0.8	1.2	0.2
NC Ac 17133 RF	1.6	3.5	0.2
080	3.3	2.9	0.4
GP-NC 343	4.5	4.8	1.1*
421	7.3	8.5	2.5*
Error mean square (111 df) = 10.4			
Trial two			
248W	0.6	3.3	−0.2
PI 259747	0.5	0.7	0.8
NC Ac 17133 RF	0.5	0.6	0.5
080	2.2	1.1	−0.9
GP-NC 343	1.2	2.0	−0.2
421	2.1	5.3	−0.5
Error mean square (97 df) = 10.4			

[a] Means were regressed on the average number of conidia per lesion for each isolate. Deviations from regression had 6 df in trial one and 5 df in trial two. Slopes were significantly different from zero at $P \leq 0.05$ (*).

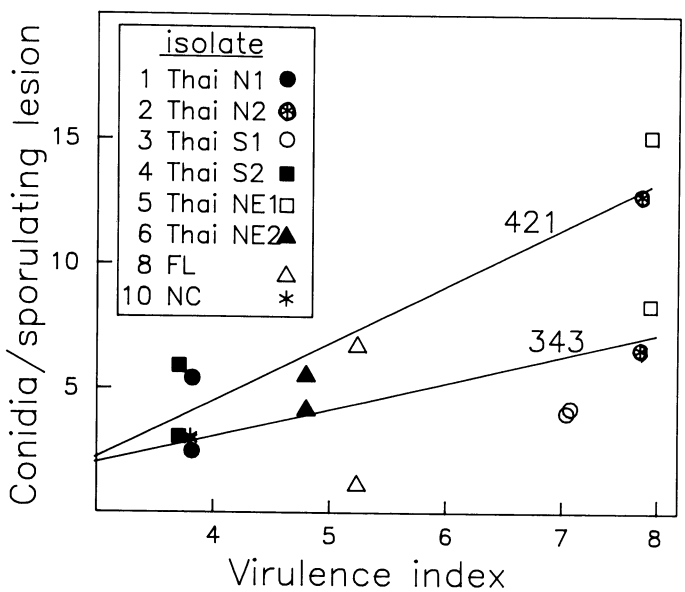

Fig. 4. Regression of number of conidia produced per sporulating lesion (× 1,250) of each of eight isolates of *Cercosporidium personatum* on virulence index (mean conidia per lesion for each isolate across all genotypes tested) for peanut genotypes KUP 24D-421 and GP-NC 343 in trial one.

smallest measured in trial two. The slight tendency for larger %LS20 on 248W inoculated with virulent isolates is probably less important than the greater responses to increasing isolate virulence in NC Ac 17133 RF and the significant deviation found for %LS20 on PI 259747.

The resistant genotypes 248W, NC Ac 17133 RF, and PI 259747 have common genetic backgrounds. NC Ac 17133 RF and PI 259747 are accessions from Tarapoto (18), and 248W is a selection from a cross of Dht 200 × Tarapoto. Genotypes with the Tarapoto background have shown resistance to *C. personatum* over a wide range of locations and environments (2,14,16,18–20); of the Tarapoto genotypes we tested, 248W had the most stable resistance to the isolates used in our trials.

The genotypes of peanut used in these evaluations represent a wide range of response to *C. personatum*, whereas the mean differences among isolates were small relative to the experimental error for most disease components. The only clear difference among Thai and U.S. isolates was in the number of lesions they caused. In previous studies, Thai isolates germinated better than U.S. isolates over a wide range of temperatures (17), which could account for higher rates of infection by Thai isolates. Once infection was established, however, variation within isolate collections and trial-to-trial variation was more important than variation among isolates from different geographic areas. Differences were not related to specific interactions of genotypes with isolates of different geographic origin but most consistently were related to increasing isolate virulence on all genotypes within a given trial. Given this response to increasing virulence, the most virulent isolate available should be used in resistance screening. In practice, differences in isolate virulence in different trials and among different resistance components make identification of a single most virulent isolate difficult. Genetically heterogeneous isolates with overall high virulence for most components of resistance may be the most useful in screening for resistance to *C. personatum* in peanut.

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