Effect of Temperature on Stability of Components of Resistance to Cercospora arachidicola in Peanut

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ABSTRACT


Expression of resistance to early leaf spot disease of peanut, caused by Cercospora arachidicola, varies across diverse geographic locations. Environment is known to influence expression of partial resistance in some pathosystems and could affect stability of resistance to early leaf spot. Multiple components of resistance were studied at controlled temperatures on seven peanut genotypes selected at North Carolina State University and on six genotypes selected at ICRISAT in West Africa. The genotypes were inoculated with a North Carolina field isolate of C. arachidicola and incubated under day/night temperature regimes of 24/24, 26/20, 32/26, 38/26, and 38/38 C (the high-temperature regimes simulate the conditions in Niger, West Africa, and the cooler regimes simulate the conditions in North Carolina). Numbers of lesions were inversely related to temperature. Days after inoculation significantly influenced numbers of lesions and infection frequency. Regression of lesion numbers or infection frequencies on time and temperature accounted for 90% or more of experimental variation for 12 of 13 genotypes. Values for most resistance components examined (number of lesions, lesion frequency, incubation period, lesion diameter, and necrotic area diameter) were dependent on both temperature and genotype. Several peanut genotypes were identified that expressed stable levels of resistance to C. arachidicola across temperature regimes. The North Carolina line 91 PA 150, derived from the wild diploid species Arachis cardenensis, consistently was ranked as resistant for all components in all temperature regimes. Other genotypes that ranked high in partial resistance to C. arachidicola included NC Ac 17894, PI 274194, NC Ac 18045, and 91 PA 131. Another group of genotypes, including GP-NC 343, NC 6, and N92069L, were moderately resistant. PI 476033 and NC 7 were highly susceptible at all temperatures, and N92064L varied in ranking for components.

Additional keywords: Arachis hypogaea, Arachis spp., groundnut, partial resistance.

Early leaf spot disease, caused by Cercospora arachidicola S. Horii, is a major constraint on production of peanut (Arachis hypogaea L.) throughout the world. Yield losses range from 10 to 50%, depending on the agroecological zone (6,11,14,25,27). Although effective chemical control methods are available, their utilization is limited in many production areas because of high costs and because fungicide-tolerant strains of the pathogen exist (13). Improving levels of resistance to C. arachidicola in locally adapted cultivars would substantially increase peanut yields in developing countries. Elsewhere, reduced fungicide use would lessen environmental impacts and increase profitability. Genotypes with partial resistance to C. arachidicola have been reported from different locations (1,4,5,7,10,12,15,22,26,27,29), and introgression of resistance into adapted peanut genotypes has resulted in advanced breeding lines with moderate to high partial resistance but low yield potential (22).

Waliyar et al. (27,29) found that expression of resistance to C. arachidicola in some genotypes depended on the geographic location where the lines were tested. For example, genotypes previously identified as highly resistant in North Carolina were susceptible in India and Niger, West Africa. Isolate specificity was identified as a possible source of this variability in collaborative research conducted by the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) and Centre de Cooperation Internationale en Recherche Agronomique pour le Developpement (CIRAD). In those studies, isolates of C. arachidicola collected from around the world produced differential responses on some peanut genotypes (22,24).

Studies on late leaf spot of peanut, caused by Cercosporidium personatum, have indicated that resistance expression can be influenced by environmental conditions present during the infection or postinfection period (18). Thus, genotype × environment interactions could cause the variable expression of resistance observed in diverse locations. Similar interactions also are possible for early leaf spot disease because development is highly dependent on environment (2,3,9). C. arachidicola infects peanut during extended periods of leaf wetness, which generally occur when temperatures are at their daily minimum. Temperatures between 20 and 24 C are highly favorable for infection (9), whereas high temperatures may slow or stop the infection process (B. B. Shew, unpublished). Daily minimum temperatures during the growing season (1 June to 30 September) average 19 C in Lewiston, North Carolina, compared with 24 C in Sadore, Niger. Mean maximum temperatures are 31 C in North Carolina and 35 C in Niger. In 1993, cooperators from ICRISAT in West Africa and the departments of Crop Science and Plant Pathology at North Carolina State University (NCSU) initiated a project to evaluate stability of resistance to early leaf spot disease in selected peanut
genotypes. Multiple components of resistance to infection were studied using a North Carolina field isolate of *C. arachidica*. In particular, stability of various host-resistance components in recently developed genotypes was investigated under different day/night temperature regimes. Temperatures used in the experiments were selected to approximate the range found in North Carolina and Niger.

**MATERIALS AND METHODS**

Seven genotypes from NCSU (8, 22) and six genotypes from ICRISAT, with various levels of resistance to *C. arachidica*, were included in the experiment (Table 1). The susceptible cultivar NC 7 and the NCSU genotypes were Virginia-type peanut (*Arachis hypogaea* subsp. hypogaea var. hypogaea). Two of the NCSU lines (91 PA 131 and 91 PA 150) were derived from a cross between the leaf spot–resistant species *A. cardenasi* Kraptop. & Greg. (2n=20) and *A. hypogaea* PI 261942-3. A tetraploid line recovered from this cross (GP-NC WS4) was hybridized in 1982 and 1983 with *A. hypogaea* PI 270806 (22), which previously was identified as having resistance to *C. arachidica* (5, 21). Finally, progeny from these crosses were hybridized to NC 5 or NC 6, and single plant progenies were selected in 1989. The lines represent the second-generation progenies of these single plant selections. Four of the ICRISAT genotypes were derived from mutants of NC 4 and two were exotic plant introductions (Table 1). Seeds from each genotype were planted in 15-cm-diameter pots, one per pot, and grown in the greenhouse. Pots contained a 2:1 mixture (v/v) of pasteurized sandy loam soil and greenhouse potting mix (Metromix 220, Grace Sierra Company, Milpitas, CA). A commercial Bradyrhizobium inoculant (cowpea group, Keel Peanut Company, Greenville, NC) was added to the soil mixture.

A detached leaf technique was used in all experiments (15). The second or third fully expanded leaves were excised from the main stems of 8- to 12-wk-old plants. Petioles of the tetrafidolate detached leaves were placed in glass 75-ml beakers, and steamed sand and water were added to beakers. Beakers containing leaves were placed in a greenhouse moist chamber for 24 h. Uninoculated detached leaves remain viable for at least 6 wk under these conditions; inoculated detached leaves provide resistance data predictive of field performance (4, 5, 10).

All experiments were conducted in growth chambers at the Phytootron facilities of the Southeastern Plant Environment Laboratory at NCSU. Enclosed Plexiglas boxes of 30.5 × 30.5 × 16 cm were used in the growth chambers to maintain high humidity during the experiments (18). Boxes were built with a 7-cm-high inner platform, which held detached leaves in beakers. The chamber volume below the platform formed a 2.85-L reservoir filled with deionized water. A grid of 36 2-cm-diameter holes in the platform allowed vapor exchange between the reservoir and the air above the platform. A plastic tube attached to a port at the side of each box led to a flask containing deionized water, and humid air was constantly passed through the boxes by bubbling incoming air through H₂O in the flask. In addition, leaves in

![Fig. 1. Number of lesions per leaf at 12, 15, 18, and 21 days after inoculation with a North Carolina isolate of *Cercospora arachidica* for 13 peanut genotypes incubated at different day/night temperature regimes: A, 26/20 C day/night, B, 32/26 C day/night, C, 38/28 C day/night. Entries: 1 = N92064L, 2 = N92069L, 3 = 91 PA 131, 4 = 91 PA 150, 5 = NC 7, 6 = GP-NC 343, 7 = NC 6, 8 = PI 476033, 9 = NC Ac 10811A, 10 = NC Ac 18045, 11 = NC Ac 18091, 12 = PI 274914, and 13 = NC Ac 17894.](image-url)

TABLE 1. Identities of genotypes used to evaluate temperature effects on expression of resistance to *Cercospora arachidica*.

<table>
<thead>
<tr>
<th>Entry</th>
<th>Identity</th>
<th>Synonym</th>
<th>Pedigree*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>N92064L</td>
<td>...</td>
<td>Florigian/GP-NC 343, F₄ derived line</td>
</tr>
<tr>
<td>2</td>
<td>N92069L</td>
<td>...</td>
<td>GP-NC 343/NC 5, F₄ derived line</td>
</tr>
<tr>
<td>3</td>
<td>91 PA 131</td>
<td>...</td>
<td>NC 6 × [PI 270806 × (GP-NC WS4)]</td>
</tr>
<tr>
<td>4</td>
<td>91 PA 150</td>
<td>...</td>
<td>NC 5 × [PI 270806 × (GP-NC WS4)]</td>
</tr>
<tr>
<td>5</td>
<td>NC 7</td>
<td>...</td>
<td>NC 5/3F93</td>
</tr>
<tr>
<td>6</td>
<td>GP-NC 343</td>
<td>...</td>
<td>NC Bunch/PI 121067</td>
</tr>
<tr>
<td>7</td>
<td>NC 6</td>
<td>...</td>
<td>GP-NC 343/VA 61R</td>
</tr>
<tr>
<td>8</td>
<td>PI 476033</td>
<td>ICG 10900</td>
<td>Valeneia line, collected in Peru in 1980 by Simpson, Pietrarelli, and Arriola</td>
</tr>
<tr>
<td>9</td>
<td>NC Ac 10811A</td>
<td>ICG 7878</td>
<td>Selection from cross of two mutants from irradiated NC 4</td>
</tr>
<tr>
<td>10</td>
<td>NC Ac 18045</td>
<td>ICG 8298</td>
<td>Selection from cross of NC Ac 17894 and F₄ #7</td>
</tr>
<tr>
<td>11</td>
<td>NC Ac 18091</td>
<td>ICG 8339</td>
<td>&quot;Recurved&quot; mutant selected from irradiated NC 4</td>
</tr>
<tr>
<td>12</td>
<td>PI 274914</td>
<td>ICG 6284</td>
<td>Bullion line obtained from Manfredi (RCM 387)</td>
</tr>
<tr>
<td>13</td>
<td>NC Ac 17894</td>
<td>ICG 6902</td>
<td>&quot;Recurved&quot; mutant selected from irradiated NC 4</td>
</tr>
</tbody>
</table>

*Information on pedigrees of entries 5-7 was extracted from Isleib and Wynne (8). GP-NC WS4 was derived from *Arachis hypogaea* (PI 261942-3) × *A. cardenasi* (PI 262141) (22).
boxes were misted manually two times each daytime period. Leaves were first placed in growth chambers set at moderate temperatures (24/24 and 26/20 C day/night), and randomly selected beakers were moved progressively to higher temperatures each 24 h to allow acclimation to high-temperature treatments before inoculation. Growth chambers were adjusted to give five day/night temperature regimes (T1-T5) within the boxes: T1 = 24/24 C day/night, T2 = 25/20 C day/night, T3 = 32/26 C day/night, T4 = 38/26 C day/night, and T5 = 38/32 C day/night. A 12-h photoperiod was obtained with fluorescent lights; intensity was about 350–400 m W m⁻² PFDD.

A mass isolate of C. arachidicola conidia was used in all tests. Conidia were obtained from infected leaves that had been collected at the Peanut Belt Research Station, Lewiston, North Carolina, and stored at 4°C until use. Conidia were suspended in a solution containing one drop of Tween 80 per 100 ml of deionized water, and the conidial concentration was adjusted to 50,000 ml⁻¹. The conidial suspension was sprayed by an artist’s airbrush at about 50 kPa air pressure. Leaves were sprayed individually with a uniform amount of inoculum; about 0.5 ml of the suspension was used per leaf inoculated. After inoculation, leaves were allowed to dry in order to avoid runoff of conidia from the leaf surface. One beaker per treatment then was placed in Plexiglas boxes. The experimental design was a split plot with temperatures as main plots and genotypes as subplots. Because the number of growth chambers in the Phytotron was limited to five (one for each temperature), one replicate of the complete set of treatments was run at one time. The entire experiment was repeated five times at weekly intervals.

Seven days after inoculation, all leaves were removed from chambers and were incubated in a greenhouse mist chamber for an additional 14 days (18). The greenhouse chamber was covered with thin plastic and contained misting nozzles that operated 10 s/1 h during the day and 10 s/2 h during the night. Relative humidity varied from 94 to 98%. Temperature in the greenhouse varied from 25 to 30 C during the day and from 20 to 25 C at night. Numbers of lesions were counted at 3-day intervals until 21 days after inoculation. No increase in lesion number or infection frequency was noted in observations made after 21 days. At that time, lesion diameters and diameters of the lesion plus the surrounding necrotic areas were measured to the nearest millimeter with a clear ruler. Lesions also were observed under a stereo-microscope at 70× magnification and sporulation was rated on a scale of 1 = no sporulation to 5 = profuse sporulation. Incubation periods were calculated as the time for appearance of 50% of the total lesion number per genotype. Infection frequency was the number of lesions divided by leaf area, as measured with a leaf area meter.

Analysis of variance did not show significant differences (P > 0.05) between repeated tests and indicated that magnitude and significance of treatment effects and mean square were similar among tests. Therefore, all data were pooled before further analysis. All data were subjected to analysis of variance. Data for lesion number and infection frequency were transformed by square roots to reduce heterogeneity of variances among treatments. Lesion number data for each genotype were subjected to regression analysis to measure the effects of time (linear and quadratic), day and night temperatures (linear and quadratic), and interaction of the linear component of temperature with the linear components of day and night temperatures. Fit of regression models was evaluated by the F test, coefficient of determination (R²), and examination of residuals. Genotypes were ranked from most resistant = 1 to most susceptible = 13 for individual resistance components at each temperature regime, and means and standard deviations of these ranks were calculated.

**RESULTS**

The main effects of temperature regimes and genotypes were highly significant (P ≤ 0.0001) for all components of resistance examined. The temperature regime × genotype interaction also was highly significant for all components of resistance except spore production.

Lesion numbers increased with time in days after inoculation (P ≤ 0.0001), but the rates of increase depended on both temperature and genotype (P ≤ 0.0001; Fig. 1A-C). Regression analysis of both independent variables accounted for 90% or more of the variation in lesion number for 12 of the 13 genotypes. Regression accounted for only 74% of the variation exhibited by entry 4 (91 PA 150), a highly resistant line derived from A. cardenasi. Time effects (T regression coefficient in Table 2) were positive for 12 entries and varied from −0.15 for entry 1 (N92064L) to 1.00 for entry 3 (91 PA 131). Nonlinear effects of time (T²) were significant only for the NCSU entries 1 (N92064L) and 2 (N92069L); all other genotypes exhibited only linear responses within the test period (Table 2). The most lesions were observed at 21 days after inoculation at the two coolest temperature regimes (24/24 and 26/20 C), with the greatest numbers consistently produced at 26/20 C. Entry 5 (NC 7) was the most susceptible and had 68 lesions at 26/20 (Fig. 1A). The main effects of day and night temperatures were highly significant but relatively small (Table 2). For example, the largest predicted change in lesion number ascribable to either day or night temperature was about four lesions per leaf. Rate of increase in lesion number depended on night temperature (T × Nᵢ significant) for all entries except 1 and 2 (Table 2). As a result, entries 1 and 2 appeared resistant at cooler temperature regimes but had relatively more lesions than other genotypes at warmer temperature regimes (Fig. 1A and C). Lesion development was much faster at lower temperatures, particularly in susceptible lines, which had many lesions by 12 days after inoculation and continued to develop additional lesions more rapidly than resistant lines (Fig. 1A-C).
Infection frequencies (lesions per square centimeter) were highly correlated with lesion numbers \( (r = 0.96, P \leq 0.0001) \). The magnitude of the increase in infection frequency with time again depended on temperature and genotype \( (data \ not \ shown) \). Infection frequency was greatest in the 26/20 C regime and decreased at the higher temperatures for most genotypes. As a result, infection frequencies on some entries, for example, 12 (PI 274194) and 13 (NC Ac 17894), were very low at the two highest temperature regimes. In contrast, infection frequency on entries 1 (NL92064L) and 2 (NL92069L) was relatively unaffected by temperature.

Incubation periods also were greatly influenced by temperatures and genotypes. Overall, incubation periods were shorter at lower temperatures (Fig. 2). Incubation periods were shortest on entries 1 (NL92064L) and 5 (NC 7) in the 26/20 and 24/24 C regimes. Incubation periods consistently were long on entry 4 (91 PA 150) and were relatively unaffected by temperature on entries 2 (NL92069L), 9 (NC Ac 10811A), and 11 (NC Ac 18091). Rankings of some genotypes varied with temperature regime. For example, incubation period on entry 6 (GP-NC 343) was relatively short at 38/26 C (12.6 days) and 32/26 C but was intermediate at the two coolest temperature regimes. Incubation period on entry 1 was shortest among all genotypes at low temperatures but was among the longest at high temperatures (Fig. 2).

Although ANOVA indicated that temperature significantly affected sporulation rating, most of the effect occurred at the highest temperature regime. Only the mean rating of 2.4 (on a scale of 1-5) at 38/32 C differed significantly from the other ratings, which ranged from 2.7 to 2.9. Significant differences in sporulation were observed among genotypes, but rankings of genotypes by sporulation potential were independent of temperature. Sporulation ratings were lowest on entries 4 (91 PA 150), 10 (NC Ac 18045), and 6 (GP-NC 343), whereas entries 5 (NC 7) and 8 (PI 476033) supported the most sporulation (Fig. 3).

Temperatures, genotypes, and their interaction influenced necrotic area diameter (Fig. 4) and lesion diameter. Necrotic area diameter and lesion diameter were highly correlated \( (r = 0.84, P \leq 0.0001) \), indicating that genotypes with large necrotic areas also had large areas. Entries 10 (NC Ac 18045), 4 (91 PA 150), and 2 (NL92069L) had small lesions and small necrotic areas at 26/20 and 24/24 C; entries 5 (NC 7) and 8 (PI 476033) had the largest lesions and necrotic areas. In general, lesion diameter and necrotic area diameter were less at higher temperatures. Temperature effects were especially pronounced on susceptible entries 5 and 8 and on more resistant entries 2, 11 (NC Ac 18091), and 12 (PI 27194) (Fig. 4). In contrast, lesion size and necrotic areas for entries 3 (91 PA 131), 4, 9 (NC Ac 10811A), and 10 did not respond significantly to temperature treatment.

Although the temperature \( \times \) genotype interaction was highly significant for most components of resistance, genotype rankings generally were similar under different temperature regimes (Figs. 1-4). Interactions usually were attributed to differences in the magnitude of genotype response to temperature rather than to changes in genotype ranking. Variability across temperature regimes was greatest at intermediate rankings; standard deviations were very small for highly susceptible and highly resistant entries.
Entry 4 (91 PA 150) consistently was very resistant for all components tested (Table 3). Entries 10 (NC Ac 18045), 13 (NC Ac 17894), 12 (PI 274194), and 3 (91 PA 131) were resistant overall to *C. arachidica*. Ranks of these entries across temperature regimes tended to be more variable than for highly resistant entry 4. Another group of genotypes, including entries 7 (NC 6), 6 (GP-NC 343), and 2 (N92069L), were moderately resistant overall. Entries 8 (PI 476033) and 5 (NC 7) were highly susceptible for all components in all temperature regimes. Ranking of entry 1 (N92064L) was intermediate overall but was highly variable (had large standard deviation), depending on temperature. Ranks across temperature treatments were most variable for incubation period and least variable for spore production.

**DISCUSSION**

Temperature and relative humidity are known to affect the epidemiology of early leaf spot (2,3,9). Disease forecasting is based primarily on occurrence of moderate temperatures during periods of prolonged high relative humidity, which presumably favors infection (9). Temperatures in the range of 20-24°C are optimal for germination of *C. arachidica* conidia (2). Germination decreases with increasing temperature, but germ tube elongation may continue at temperatures inhibitory to germination (2). In tests performed concurrently with the experiments we report here (data not shown), a lower percentage of conidia germinated after 48 h at constant 24°C than at 26/20°C. These treatments did not differ after 72 h of incubation. The lag in conidial germination at 24/24°C may account for the smaller number of lesions observed than at 24/20°C.

Differences among temperature treatments in lesion number, incubation period, infection efficiency, and lesion size were attributed to the direct effects of temperature on infection and initial colonization. Temperature effects on sporulation were indirect because sporulation occurred after the treatments ended. Differences in sporulation probably reflected different rates of coloniza-

![Fig. 4. Effect of day/night temperature on diameter of lesions and surrounding necrotic areas caused by *Cercospora arachidica* on 13 peanut genotypes. Entries: 1 = N92064L, 2 = N92069L, 3 = 91 PA 131, 4 = 91 PA 150, 5 = NC 6, 6 = GP-NC 343, 7 = NC 7, 8 = PI 476033, 9 = NC Ac 10811A, 10 = NC Ac 18045, 11 = NC Ac 18091, 12 = PI 274194, and 13 = NC Ac 17894. LSDs are shown for comparisons of genotypes within a temperature treatment.](image)

**TABLE 3. Means and standard deviations of genotype ranks across five temperature regimes for selected components of resistance to *Cercospora arachidica* in peanut**

<table>
<thead>
<tr>
<th>Entry</th>
<th>Spore production</th>
<th>Incubation period</th>
<th>Infection frequency</th>
<th>Necrotic area</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entry</td>
<td>Mean ± SD</td>
<td>Entry</td>
<td>Mean ± SD</td>
<td>Entry</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>4</td>
<td>1.0 ± 0.00</td>
<td>4</td>
<td>1.8 ± 0.84</td>
<td>4</td>
<td>1.4 ± 0.89</td>
</tr>
<tr>
<td>10</td>
<td>2.2 ± 0.45</td>
<td>12</td>
<td>5.6 ± 1.82</td>
<td>12</td>
<td>3.4 ± 1.67</td>
</tr>
<tr>
<td>6</td>
<td>3.4 ± 0.89</td>
<td>3</td>
<td>5.6 ± 3.05</td>
<td>13</td>
<td>4.0 ± 1.87</td>
</tr>
<tr>
<td>13</td>
<td>5.6 ± 2.07</td>
<td>10</td>
<td>6.0 ± 2.35</td>
<td>7</td>
<td>4.4 ± 1.52</td>
</tr>
<tr>
<td>1</td>
<td>6.6 ± 3.13</td>
<td>1</td>
<td>6.6 ± 5.60</td>
<td>3</td>
<td>5.8 ± 4.60</td>
</tr>
<tr>
<td>7</td>
<td>6.6 ± 2.70</td>
<td>11</td>
<td>6.8 ± 3.83</td>
<td>11</td>
<td>8.2 ± 2.17</td>
</tr>
<tr>
<td>11</td>
<td>7.4 ± 2.70</td>
<td>4</td>
<td>7.0 ± 4.18</td>
<td>11</td>
<td>8.2 ± 3.56</td>
</tr>
<tr>
<td>3</td>
<td>7.6 ± 2.05</td>
<td>9</td>
<td>7.0 ± 3.32</td>
<td>10</td>
<td>9.2 ± 1.64</td>
</tr>
<tr>
<td>12</td>
<td>8.0 ± 2.45</td>
<td>13</td>
<td>7.2 ± 3.77</td>
<td>6</td>
<td>9.4 ± 0.55</td>
</tr>
<tr>
<td>2</td>
<td>8.4 ± 2.30</td>
<td>8</td>
<td>7.8 ± 2.39</td>
<td>8</td>
<td>9.4 ± 2.61</td>
</tr>
<tr>
<td>9</td>
<td>9.2 ± 1.79</td>
<td>6</td>
<td>11.2 ± 1.79</td>
<td>2</td>
<td>10.0 ± 1.87</td>
</tr>
<tr>
<td>8</td>
<td>12.4 ± 0.55</td>
<td>5</td>
<td>11.8 ± 1.64</td>
<td>5</td>
<td>12.8 ± 0.45</td>
</tr>
</tbody>
</table>

*Genotypes were ranked from 1 = most resistant to 13 = most susceptible at each of five temperature regimes, and the ranks were averaged.

*Ranks were based on data from day 21.

See Table 1 for key to entry numbers.
tion during the treatment period or the effects of temperature on lesion number. Spore production begins earlier and is more profuse on leaves with large numbers of lesions.

In the current tests, temperature regimes including or exceeding 32°C inhibited development of early leaf spot, although lesions were observed in all treatments. During the growing season, minimum temperatures very rarely exceed 26°C in North Carolina, but in West Africa, they regularly fit into the nonconducive range observed in this study. Nevertheless, early leaf spot causes extensive damage to peanut in West Africa, with more than 90% leaf area damage reported (28). Isolates of C. arachidicola from West Africa may be less sensitive to higher temperatures than the isolate used in the current study. High-temperature adaptation has been reported for conidial germination of Cercosporidium personatum (20). It also is possible that West African isolates are more aggressive or germinate more quickly, allowing them to infect during even brief cool periods.

These studies demonstrate that certain genotypes do not possess stable resistance in diverse environments because some lines reacted differently to C. arachidicola under different temperature regimes. In a study of stability of resistance to Cercosporidium personatum Shew et al. (19) also reported that performance of two peanut lines was more sensitive to high temperatures than other genotypes in their study. Interactions among time, temperature, and genotype are of great importance, especially for regional and international breeding programs. Breeders may need to evaluate their lines in diverse environments to ensure that expression of leaf spot resistance is stable.

Several components of resistance to peanut leaf spots contribute to the reduction in the rate of epidemic progress (4, 10, 16, 29). In the current study, genotypes differed for all components of resistance measured under a range of temperatures. Most genotypes that were classified as resistant to C. arachidicola had fewer lesions and lower infection frequencies than susceptible genotypes; lesions on resistant genotypes exhibited longer incubation periods and lower intensities of sporulation and were smaller than those on susceptible genotypes. In general, most components (numbers of lesions, infection frequency, incubation period, etc.) of disease were reduced at temperatures of more than 32°C, but even with reduced disease, highly significant differences among genotypes were observed. Some genotypes changed in rankings for specific components of resistance under high temperatures. For example, entry 11 (NC Ac 18091), which was reported resistant to C. arachidicola in West Africa (26, 29), was ranked among susceptible lines based on lesion number in the two lower temperature regimes (24/24 and 26/20°C) in this study. In higher temperature regimes, however, it was among the genotypes with a low lesion number. Entry 11 also had variable reactions for other components of resistance in different temperature regimes. In contrast, the North Carolina entries 1 (N92064L) and 2 (N92069L) had fewer lesions relative to other genotypes at lower temperatures than at higher temperatures. Other differences were attributed to isolate source rather than to temperature. Entry 8 (PI 476033) was reported resistant to C. arachidicola in West Africa (26) but was susceptible with the North Carolina isolate at all temperatures. This reversal in the resistance characterization of some lines when tested elsewhere (using local pathogen isolates) previously had been reported by Waliyar et al. (28). They observed that PI 350680 (ICG 6340) and NC 5 (ICG 2711), which are moderately resistant to early leaf spot in the United States (5, 10, 12), were susceptible (PI 350680) or moderately susceptible (NC 5) according to most components of resistance when tested with a C. arachidicola isolate from India. Several peanut lines expressed stable levels of resistance to C. arachidicola in this study. Among the North Carolina genotypes, entry 491 (PA 150) was highly resistant in all temperature regimes. The resistance in this line is believed to come mostly from the wild diploid species A. cardenasi and to a lesser extent from PI 260806. Although resistance to C. arachidicola in these lines is multigenic (17), the results indicate that multiple resistance factors can be combined into a single genotype. Among the lines from West Africa, NC Ac 17894, PI 274194, NC Ac 18045, and NC Ac 10811A were resistant to the North Carolina isolate of C. arachidicola.

This study confirms, and adds additional information to, previous reports of variability in expression of resistance to peanut to C. arachidicola. This variability is important to international peanut breeding efforts and may help explain the failure of some breeding lines to express resistance to early leaf spot in diverse environments.

LITERATURE CITED


