Effects of Temperature and Relative Humidity on Expression of Resistance to *Cercosporidium personatum* in Peanut

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ABSTRACT

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Detached peanut leaves were inoculated with the leaf spot pathogen *Cercosporidium personatum* and exposed to 20, 24, 28, or 32 C for 6 days. Maximum infection occurred at 20 C, provided leaves also were exposed to at least 12 hr/day high relative humidity (RH > 93%). Infection of genotypes with high (PI 259747, NC Ac 17133), moderate (GP-NC 343), and low (NC 3033, Robut 33-1) partial resistance decreased with increasing temperature. Few infections occurred at 28 and 32 C regardless of duration of the high relative humidity period. Daily periods of high relative humidity shorter than 12 hr also reduced the number of infections on all genotypes regardless of temperature. Ranking of genotypes by lesion numbers was similar at all temperature and relative humidity periods tested. Lesions on leaves of PI 259747, NC Ac 17133, GP-NC 343, NC 3033, and Robut 33-1 were largest, developed most rapidly, and sporulated most profusely at 24 C. Lesions on FESR 5-P2-B1, a genotype reported to have high combining ability for leaf spot resistance, developed fastest at 28 C. Postinfection development of *C. personatum* was completely inhibited at 28 C on the highly resistant genotypes, and at 32 C on all genotypes.

Additional key words: *Arachis hypogaea*, groundnut.

Leaf spots caused by *Cercosporidium personatum* (Berk. & Curt) Deighton and *Cercospora arachidicola* Hori are the most important diseases of peanut (*Arachis hypogaea* L.) worldwide, and both occur wherever the crop is grown. Local climate, cultural practices, and host genotypes probably determine which leaf spot is more abundant. *C. personatum* predominates in the tropics and subtropics, and since the late 1970s has become the major peanut leaf spot in the southernmost United States (16).

Partial resistance to late leaf spot has been identified in several peanut genotypes (18) and is inherited quantitatively (2,19). Long latent periods, small lesions, small amounts of leaf area damaged, and low rates of sporulation contribute to high partial resistance to late leaf spot (9,20). The importance of reduced infection efficiency (lesions per unit of inoculum) in partial resistance is less clear, and expression of this component, as well as the others, may depend on environment (11,20).

Temperatures of 16–20 C are very favorable for germination of *C. personatum* conidia (17). Germination declines gradually up to 28 or 30 C, and then is sharply inhibited at higher temperatures (17). No information about effects of temperature on further development of the pathogen is available from controlled experiments, but Jensen and Boyle (5) developed a leaf spot forecasting model from correlations between disease progress on susceptible varieties in the field and temperature, relative humidity, and rainfall. The model predicts increasing leaf spot hazard with increasing temperature above 21 C (5). It is unclear what stages of development of both or either leaf spot pathogen are favored by the conditions the Jensen and Boyle model describes.

In greenhouse inoculations, lesions develop on leaves exposed to 24 hr of continuous misting (14). Longer mist periods up to 8 days increase lesion numbers (14), and many investigators expose plants or leaves to several days of continuous high relative humidity after inoculation with *C. personatum* (2,20). Such long periods of uninterrupted leaf wetness rarely occur in the field, so natural infections often must develop during discontinuous periods of high relative humidity. The length of the high relative humidity period necessary for infection also could depend on the level of partial resistance in the host.

Success in efforts to develop high yielding cultivars with better resistance to late leaf spot depends on identification of partial resistance that is expressed even when environment favors rapid disease increase. The objectives of this research were: to determine the temperatures and durations of high humidity that favor infection of peanut by *C. personatum*, to determine the temperatures favorable for postinfection development of *C. personatum*, and to determine if temperature or relative humidity affect expression of resistance to the late leaf spot pathogen in peanut.

MATERIALS AND METHODS

Peanut leaves infected with *C. personatum* were collected at the Peanut Belt Research Station near Lewiston, NC. Conidia were removed from lesions with a cyclone spore collector (ERI Machine Shop, Ames, IA) and were stored in glass test tubes at 4 C. These conidia were inoculated onto potted plants of NC 3033 peanut, which is susceptible to late leaf spot, and infected plants were placed in growth chambers or in benchtop humidity chambers in the greenhouse. Cultures of the pathogen were maintained by periodic inoculation of additional plants. Conidia used in experimental inoculations were collected from infected leaves of these plants.

Five peanut genotypes that represent various levels of partial resistance to the late leaf spot pathogen were used in all experiments. PI 259747 and NC Ac 17133 (RF) have high partial

resistance to *C. personatum* (18), GP-NC 343 has moderate resistance (6), and NC 3033 (6) and Robut 33-1 (18) have very low resistance to the late leaf spot pathogen. Plants of each genotype were grown in the greenhouse in 15-cm-diameter pots that contained a 2:1 mixture (v:v) of pasteurized sandy loam soil and greenhouse potting mix (W. R. Grace and Co., Cambridge, MA). Commercial Rhizobium inoculant (cowpea group; Keel Peanut Co., Greenville, NC) was included in the soil mixture.

All experiments were performed on leaves detached from 8–12-wk-old plants (7). Previous experiments have shown that results of detached leaf inoculations agree closely with field evaluations of resistance (2,18,20). Leaves at nodes one to three (in relation to the branch terminals) were excised and each petiole was placed in a plastic 75-ml capacity beaker. Steamed builders' sand and water were added to beakers, which were then sealed with a layer of plastic film. The plastic film prevented evaporation from the moist sand surface. Deionized water was added to sand by syringe as needed. Uninoculated detached leaves have remained in excellent condition for experiments lasting 80–90 days.

Conidial suspensions were prepared from dry conidia of *C. personatum*. A solution of one drop of Tween 80 per 100 ml of deionized water was added to dry conidia, and the resulting suspension was poured through a sieve (246-μm opening) to remove leaf tissue and conidiophores. Conidial concentration was determined by hemacytometer counts and was adjusted to 40,000 conidia per milliliter. Suspensions were applied to runoff on leaves by an artist's airbrush operated at 48 kpa air pressure.

Humidity chambers for experiments were built from 6-mm-thick clear acrylic and had dimensions of 30.5 × 30.5 × 16 cm. The chambers were built with an inner platform that was 7 cm high, and the chamber volume below the platform formed a 2.85-L reservoir. A grid of 36 2-cm-diameter holes in the platform allowed vapor exchange between the reservoir and the air above the platform. Detached leaves in beakers were placed on the platform inside humidity chambers. Reservoirs were filled with deionized water for high humidity treatments; reservoirs in low humidity treatments were filled with a saturated NaCl solution. Humidity chambers had a 5-cm-diameter port in one side. A plastic tube that led to a cool-air humidifier was attached to the port in high humidity treatments. The port in low humidity chambers was attached to a small pump that bubbled air through a saturated sodium chloride solution and then into the chambers. Relative humidities in chambers were monitored with a Vaisala Humicap humidity meter (Vaisala, Woburn, MA), and measured humidities ranged from 65 to 75% in low humidity chambers and from 93 to 99% in high humidity chambers.

Infection experiment. A low humidity chamber and a high humidity chamber were placed in each of four 0.91- × 1.22- × 1.22-m growth chambers. Fluorescent lights in growth chambers provided 292 hlx illuminance between 6 a.m. and 8 p.m. daily. Growth chamber temperature was lowered when lights were on so that temperature inside humidity chambers was maintained at constant 20, 24, 28, or 32 C as measured by no. 24 type T thermocouples (4). Leaves were inoculated between 4 and 5 p.m. and were allowed to dry. At 8 p.m., all leaves were misted with deionized water and placed in either high humidity or low humidity chambers in each growth chamber. Leaves in low humidity chambers dried within 3 hr and stayed dry for the remaining 21 hr of each day. Additional leaves were transferred from high to low humidity chambers after 12 and 18 hr. The remaining leaves were kept in high humidity boxes 24 hr. The cycle was repeated for a 6-day infection period, and then leaves were transferred to a greenhouse chamber for further incubation. Greenhouse chambers were enclosures covered with thin plastic. Chambers were placed under misting nozzles that operated for 7 sec every 10 min during daylight and for 7 sec every hour during the night. Leaves in chambers were exposed to constant high relative humidity but were not directly wetted by the mist. Average daily maximum and minimum temperatures in the greenhouse were 35 and 25 C in experiment one and 31 and 20 C in experiment 2. Lesions were counted 21 days after inoculation.

The five genotypes, four temperatures, and four humidity periods were arranged in a factorial treatment design; the experimental design was a split-split-plot with temperatures as whole plots, humidity periods as subplots, and genotypes as subsubplots. Because the number of available growth chambers was limited to four, one replicate of the complete set of treatments was run at one time, and four replicate sets of the treatments were completed over time. The entire experiment was then repeated in a similar fashion with three replications.

Postinfection experiment. Inoculated detached leaves were placed in high humidity chambers that were maintained at 20 C in growth chambers. Leaves were swabbed with 70% aqueous ethanol to stop further infections after a 4-day infection period at 20 C. Leaves were then transferred to high humidity chambers that were maintained at 20, 24, 28, or 32 C in growth chambers. Leaves were checked daily for sporulation and once sporulation was detected, lesions and sporulating lesions were counted every 2 to 3 days until 32 days after inoculation. At each counting date, stroma development on each leaf was also recorded on a 0–3 scale. After 32 days of incubation, the lengths and widths of the three largest lesions on each leaf were measured to the nearest 0.5 mm. Conidia were then removed from all lesions on each leaf with a cyclone spore collector. These conidia were suspended in 0.2–1 ml of deionized water plus surfactant, and drops from the suspension were counted with a hemacytometer.

The five genotypes used in infection studies and a genotype that was reported to consistently produce F_1 progeny with partial resistance to late leaf spot, FESR 5-P2-B1 (2), were exposed to each of four temperature treatments in the factorial treatment design. The experimental design was a split-plot, with temperatures as whole plots and genotypes as subplots. One replicate set of treatments was run at a time with three replications in all. The entire experiment was then repeated with two replications.

Data analysis. Analysis of data from the two runs of each experiment yielded similar results and comparable error mean squares. Experimental runs were therefore combined in further analyses to give a total of five postinfection or seven infection replications. Data were transformed by square roots (lesion number), by arcsine-square roots (proportion of lesions sporulating), or by natural logs (conidia per leaf) to reduce heterogeneity of variances among treatments (including days from inoculation). Areas under curves of percent lesions sporulating versus days from inoculation (AUSC) were calculated (13), and all data were analyzed by analysis of variance. Response to quantitative variables (temperature and time in days from inoculation) was characterized with linear and quadratic regression models. Fit of various regression models was evaluated by *F*-tests for significant models and for significant lack-of-fit to models, by visual inspection of residual plots, by size of standard errors associated with the estimated regression parameters, and by R^2 (8).

RESULTS

Infection experiment. Constant exposure to 20 or 24 C was very favorable for infection of peanut by *C. personatum* if leaves also were exposed to high relative humidity for at least 12 hr/day in a six-day infection period (Fig. 1). In contrast, leaves from all peanut genotypes had few lesions when they were exposed to 28 or 32 C (Fig. 2) or to <3 hr/day high relative humidity after inoculation (Fig 3). Temperature and relative humidity effects were highly significant ($P < 0.01$) but mutually dependent. Both favorable temperatures and sufficient durations of high relative humidity were necessary for large numbers of infections ($P < 0.01$, Fig. 1). Changes in lesion numbers in response to temperatures and humidity periods tested were abrupt and could not be described by linear or continuous curvilinear (polynomial) models. The most lesions developed on leaves exposed to 20 C after inoculation, and the greatest decrease in infection with increasing temperature occurred between 24 and 28 C (Fig. 2). Infection increased most sharply as humidity period increased to 12 hr and continued to increase gradually with longer exposure to high relative humidity (Fig. 3). Temperature and relative humidity period had similar

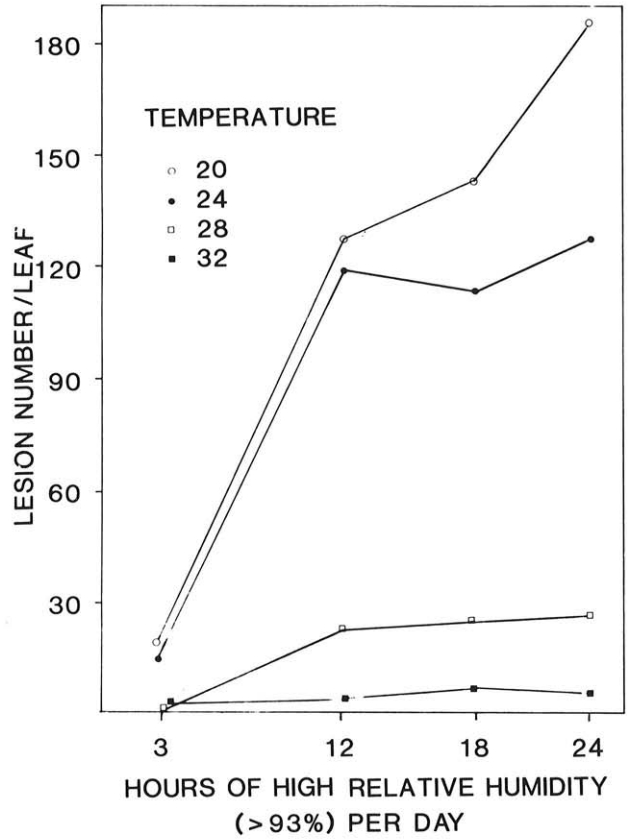

Fig 1. Influence of constant temperature and length of daily exposure to high relative humidity (RH > 93%) on infection of peanut leaves by *Cercosporidium personatum*. Points represent mean lesions per leaf on seven replications of five peanut genotypes.

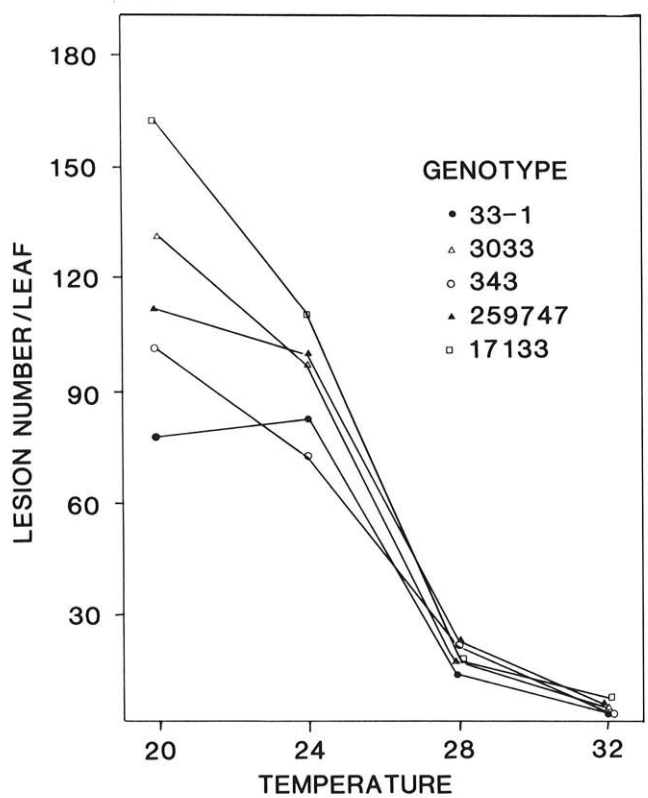

Fig. 2. Infection of five peanut genotypes by *Cercosporidium personatum* at four constant temperatures. Values represent mean lesions per leaf on seven replications of four relative humidity treatments.

effects on infection of all genotypes (*F*-tests for temperature × genotype, relative humidity × genotype, and temperature × relative humidity × genotype were nonsignificant at $P = 0.05$), although different numbers of lesions occurred on the genotypes tested ($P < 0.01$, Table 1). Fewest lesions developed on Robut 33-1, whereas NC Ac 17133 (RF) had the most lesions in all temperature and humidity treatments.

Postinfection experiment. The relationship between temperature and lesion size was nonlinear and was not well described by linear or polynomial regressions for any genotype. By 32 days after inoculation, lesions on five of six genotypes were larger on leaves that were incubated at 24 C than on leaves that were incubated at 20 or 28 C (Fig. 4). Lesion size on one genotype, FESR 5-P2-B1, was less sensitive to temperature than on the other genotypes. Lesions on FESR 5-P2-B1 were largest at 20 C and smaller as temperature increased from 20 to 28 C (Fig. 4). The differential response of genotypes to increasing temperature caused a highly significant genotype × temperature interaction for lesion size. For all genotypes, lesions on leaves incubated at 32 C were less than 1 mm^2 (Fig. 4).

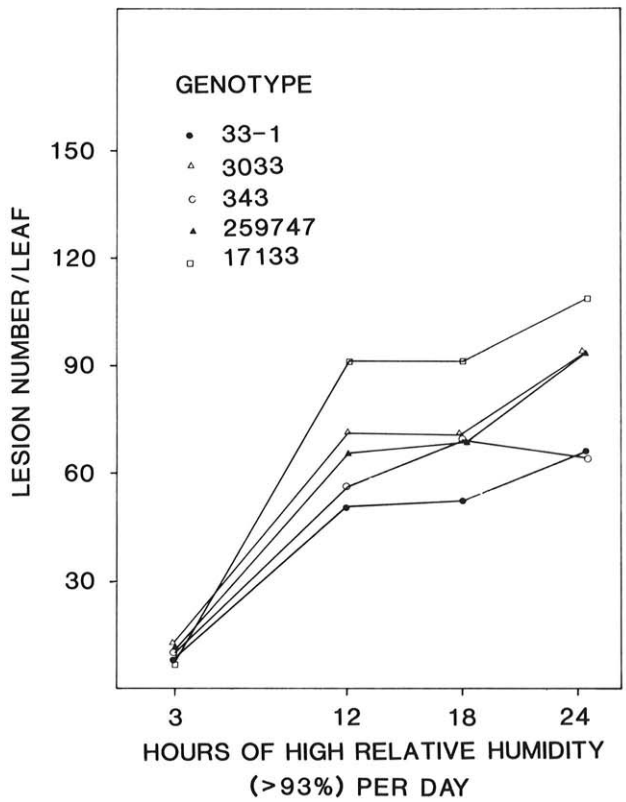

Fig. 3. Infection of five peanut genotypes by *Cercosporidium personatum* after exposure to four daily relative humidity regimes. Values represent mean lesions per leaf on seven replications of four temperature treatments.

TABLE 1. Number of leaf spot lesions on leaves of five peanut genotypes inoculated with *Cercosporidium personatum*[a]

Genotype	Lesions per leaf
Robut 33-1	4.44[b]
NC 3033	5.41
GP-NC 343	4.98
PI 259747	5.55
NC Ac 17133 (RF)	5.71
LSD ($P = 0.05$)	0.64

[a] Leaves were incubated 6 days at 20, 24, 28, or 32 C and at <3, 12, 18, or 24 hr high relative humidity (>93%) day, and then in the greenhouse for 15 days.

[b] Mean of 16 temperature × relative humidity treatments and seven replications after square root transformation of data.

Latent period, expressed as time in days to first observed sporulation, was dependent on temperature for the genotypes examined ($P < 0.01$) because lesions on PI 259747 and NC Ac 17133 failed to sporulate by 32 days after inoculation (Table 2). Lesions on these genotypes were small (< 1 mm^2) and exhibited little or no stromatal development at 28 C. In contrast, sporulation on the other genotypes at 28 C began by at least 23 days after inoculation (Table 2). Sporulation began soonest at 24 C for all genotypes except FESR 5-P2-B1 (Table 2).

For most treatments, a large proportion of lesions on a given leaf were sporulating once sporulation was first observed (Table 2), but many leaves in a treatment had no sporulating lesions until 21 days after inoculation. Therefore, only data from days 21 through 32 were included in regressions of percent lesions sporulating versus time (in days after inoculation) to reduce heterogeneity of errors. There was no sporulation at 32 C on any genotype, and these observations also were dropped before percent lesions sporulating was analyzed.

Analysis of variance of the remaining data showed a highly significant ($P<0.01$) interaction among time (days after inoculation), genotype, and temperature because lesions on PI 259747 and on NC Ac 17133 did not sporulate at 28 C. The percent lesions that sporulated on the other genotypes at 28 C was at least equal to the rates at 24 C for these genotypes (Fig. 5). The percent lesions sporulating data were reanalyzed without the PI 259747 and NC Ac 17133 observations, and the time × genotype × temperature interaction was no longer significant ($P > 0.05$). The main effect of genotypes was still significant ($P < 0.01$), and an interaction of time and temperature ($P < 0.01$) indicated that different models were necessary to describe the increase in percent lesions sporulating with time at 20, 24, and 28 C (Fig. 5). A linear model fitted to the data at 20 C predicted 11.6% of lesions sporulating on day 21, and increasing sporulation at a rate of 6.3% per day. Time to 50% of lesions sporulating, averaged for all genotypes at 20 C, was calculated from regression as 27.1 days. Sporulation increased as a quadratic function of time at 24 and 28 C. Models predicted 34.5% of lesions sporulating on day 21 at 24 C and 37.8% of lesions sporulating on day 21 at 28 C. Both models predicted increasing sporulation up to 32 days after inoculation, with maximum sporulation at time ≥ 32 days. Average time to 50% lesions sporulating was calculated as 23.7 days at 24 C and as 22.4 days at 28 C.

Genotypes differed ($P < 0.01$) in accumulated rates of percent lesions sporulating, according to AUSC (Fig. 6). Rankings of genotypes by AUSC were similar at 20, 24, and 28 C ($P > 0.05$);

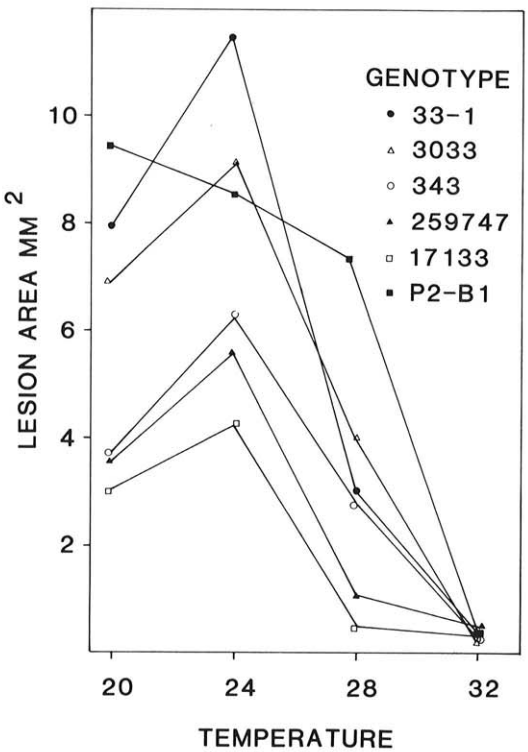

Fig. 4. Mean size of three largest lesions on leaves of six peanut genotypes that were inoculated with *Cercosporidium personatum* and incubated at four temperatures. Values represent means of five replications.

TABLE 2. Effect of temperature on initiation of sporulation by *Cercosporidium personatum* on six peanut genotypes

	Days to first sporulation[a]			Lesions sporulating (%)[b]		
Genotype	20	24	28	20	24	28
Robut 33-1	23.4	20.0	20.4	11	10	17
NC 3033	21.6	17.8	23.2	22	14	18
GP-NC 343	25.2	20.4	21.3	7	8	21
PI 259747	28.4	25.2	...[c]	8	9	...
NC Ac 17133	27.6	23.8	...	23	19	...
FESR 5-P2-B1	21.6	19.2	18.0	17	25	16
LSD ($P=0.05$)			3.9			ns

[a] Means of five replications. LSD is for comparisons within columns (temperatures).
[b] Percent of lesions sporulating on the day sporulation was first observed.
[c] No sporulation was observed by 32 days after inoculation.

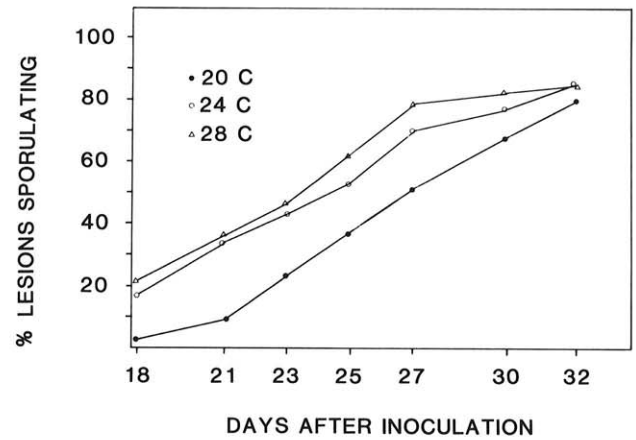

Fig. 5. Relationship of percent lesions sporulating and days after inoculation for peanut leaves incubated at three temperatures after infection by *Cercosporidium personatum*. Data were transformed by arcsine-square roots and points represent means of five replications and six genotypes at 20 and 24 C. Only genotypes that sporulated at 28 C were included in the graph; NC Ac 17133 and PI 259747 did not sporulate at 28 C.

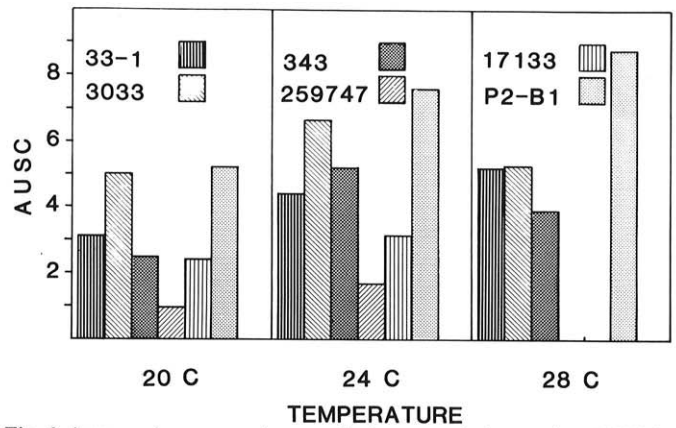

Fig. 6. Areas under curves of percent lesions sporulating vs. time (AUSC) for six peanut genotypes infected with *Cercosporidium personatum* and incubated at 20, 24, or 28 C. Areas are means of five replications. Main effects of temperature and genotypes were significant at $P = 0.01$; the interaction was not significant.

FESR 5-P2-B1 and NC 3033 had the greatest areas and PI 259747 the least at all temperatures. Rankings of genotypes by the number of conidia collected per leaf (log transformed) were the same as rankings by AUSC (Fig. 7). The number of conidia collected differed significantly among genotypes ($P<0.01$) and temperatures ($P<0.01$), but the response to temperatures was independent of genotype ($P>0.05$). Lesions on leaves of a given genotype generally produced slightly more conidia at 24 than at 20 C, and fewer conidia at 28 C (Fig. 7). No conidia were collected from leaves incubated at 32 C for any genotype.

DISCUSSION

Numbers of lesions resulting from inoculations in greenhouse screenings have been an unreliable measure of partial resistance to peanut leaf spots (2,11,19,20). In single infection cycles, genotypes with high partial resistance such as PI 259747 and NC Ac 17133 often have many more lesions than less resistant genotypes. Furthermore, although average lesion number often differs among genotypes within trials, rankings of genotypes by lesion number can change dramatically between trials (11). Environmental variation has been suggested as a cause of shifts in rankings of genotypes (2,9,18,20), but genotypes in our infection experiments maintained similar rankings by lesion number in different temperature and relative humidity treatments. One or more of the other environmental parameters that were held constant in growth chambers could partially account for the consistency in lesion numbers that we observed. In our experiments, temperature and duration of relative humidity had a greater impact on lesion numbers than did genotype.

Jensen and Boyle believed their leaf spot forecasting model predicted periods favorable for infection of peanut by leaf spot pathogens. Their model predicts high leaf spot hazard when leaves are wet for at least 10 hr/day on two consecutive days at favorable temperatures. We likewise found 12 hr/day of high relative humidity adequate for high rates of infection of all genotypes tested. The relationship of increasing infection with decreasing temperature corresponded well with published conidial germination data (17) but not with the Jensen and Boyle model (5). For example, the model predicts high leaf spot hazard at 28 C for any relative humidity period longer than 7 hr, whereas few lesions developed at 28 C in any humidity treatment on any genotype in our experiment. Our observation of high infection rates at 20 C was also in marked contrast to the leaf spot model. The model predicts only low to moderate disease hazard at 20 C with 20 hr/day of high relative humidity, and no hazard at 20 C for relative humidity periods shorter than 14 hr/day. In spite of these discrepancies, the model has been the basis for a spray advisory that has been used successfully in Virginia (10) and North Carolina (3) for several years. The most important peanut leaf spot in these states is caused by *C. arachidicola*, which may have different temperature optima than *C. personatum*. Also, the model may be effective because it may predict periods that are favorable for sporulation (1) or lesion development rather than infection.

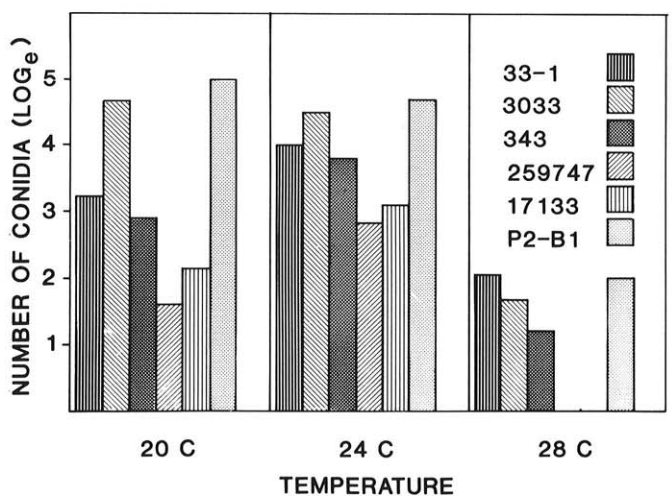

Fig. 7. Influence of temperature and peanut genotype on production of conidia by *Cercosporidium personatum*. Raw counts (shown log transformed on graph) were multiplied by 222 to obtain number of conidia collected per leaf. Means represent five replications. Main effects of temperature and genotypes were significant at $P=0.01$; the interaction was not significant.

Jensen and Boyle were aware that postinfection development might proceed more rapily at warmer temperatures, thus altering the accuracy of their model (5). The higher rates of postinfection development (as measured by latent period, lesion size, and spore production) that we observed at 24 and 28 C could be more important in a polycyclic epidemic on susceptible and moderately resistant genotypes than the lower rates of infection observed at these temperatures.

Leaf spot severity can be expected to be highest when temperatures near 24 C occur during long leaf-wetness periods. Infections probably occur at night or in early morning when leaves are wet and temperatures are cool. Postinfection development probably proceeds rapidly at warm temperatures, slows during the hottest part of the day, and resumes in the evening as temperatures decrease.

The nearly complete inhibition of leaf spot infection and development on all genotypes at 32 C was unexpected because plants in the field are nearly always exposed to daily maximum temperatures of at least 32 C. Conidia must survive exposures to high temperature and low relative humidity that occur between deposition and favorable infection periods. Leaves of NC 3033 became infected if transferred to 20 C after 4, but not 6, days of exposure to 32 C (15). The minimum period of exposure to high temperature that irreversibly inhibits postinfection development of leaf spot is not known, but lesions on leaves that were transferred from 32 C to lower temperatures at the end of our experiments (after 28 days exposure to 32 C) did not resume development within 2 wk of transfer.

The two genotypes with highest partial resistance to late leaf spot were more sensitive to high temperature than the other genotypes, but this sensitivity did not affect genotype rankings by the components of resistance examined. NC Ac 17133 and PI 259747 were always the most resistant genotypes over the range of temperatures tested, according to lesion size, latent period, AUSC, and spore production. Lesion number did not correspond well with the other measures of resistance because expression of resistance in these genotypes was associated with the appearance of many small, nonsporulating lesions.

Resistance in PI 259747 and NC Ac 17133 appears to be recessive and may be controlled by similar genetic systems (19). Evidence from evaluation of progeny from F_1 crosses involving GP-NC 343 suggests that moderate partial resistance in this genotype is inherited differently from the resistance in NC Ac 17133 (2). Differential sensitivity to temperature of the highly (PI 259747 and NC Ac 17133) and moderately (GP-NC 343) resistant genotypes also suggests that resistance is controlled differently in these plants. Still other resistance genes may be present in FESR 5-P2-B1. This genotype appeared highly susceptible in our trials but is thought to convey partial resistance to its progeny (2).

Latent period of *C. personatum* was difficult to describe for statistical comparisons of individual genotypes because curves of percent lesions sporulating usually did not follow the S-shaped models appropriate for rusts and mildews (12). On susceptible to moderately resistant genotypes, sporulation began abruptly, without the typical lag phase seen with biotrophic pathogens. Percent sporulating lesions on these genotypes remained high and relatively constant for the length of the experiment and may remain high even after infected leaves abscise (11). This pattern accounts for poor fit of linear and polynomial models to sporulation curves. On genotypes with high partial resistance, fewer than half the lesions may sporulate even at favorable temperatures, which hampers calculation of a latent period for these genotypes, where latent period is defined as time to

sporulation of 50% of lesions. AUSC describes the entire sporulation process and could be useful when more specific models are not appropriate. An alternate definition of latent period can be time to first observed sporulation, but this measurement is sensitive to large changes in lesion number (11). We reduced this variation by studying postinfection development separately from infection, using a uniform infection period for all treatments.

The abrupt decrease in all measures of infection and development as temperature increased suggests that uneven temperature variation within experiments has the potential to cause confusing results and increase experimental errors in leaf spot resistance screening. In the field, however, we expect expression of partial resistance from the sources we examined to be consistent and stable regardless of climate.

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