PEANUT SCIENCE

VOLUME 35 JANUARY–JUNE 2008 NUMBER 1

Disease Progress of Early Leaf Spot and Components of Resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in Runner-Type Peanut Cultivars

E.G. Cantonwine[1]*, A.K. Culbreath[2], C.C. Holbrook[3], and D.W. Gorbet[4]

ABSTRACT

This study assessed components of resistance for three runner-type peanut cultivars to infection by *Cercospora arachidicola* (*Ca*) and *Cercosporidium personatum* (*Cp*), the causal organisms of early leaf spot and late leaf spot, respectively. Resistance components were compared to disease resistance observed in the field. A field study monitored the progression of leaf spot incidence and severity in peanut cultivars Georgia Green, Georganic, and DP-1. Time of disease onset (TDO) and temporal epidemic rate (rate) were estimated for incidence with the logistic model, and for severity with the linear model. Early leaf spot was the predominant disease in the field. Estimates of TDO were 9 d later for DP-1 than for Georgia Green, based on incidence models, and 6 and 7 d later for Georganic and DP-1 than for Georgia Green, respectively, based on severity models. Incidence progression rate was highest for Georganic in 2002 and Georgia Green in 2003, while severity progression rate was highest for Georgia Green across years. A detached leaf assay was used to determine components of resistance for these genotypes to infections by *Ca* and *Cp*. Infection frequency 30 d after inoculation, lesion diameter, and percent necrotic area were greatest for Georgia Green for both pathogens. Besides a 2-d longer latent period for resistant genotypes, no *Ca* reproduction differences were detected. For *Cp*, latent period was shorter for Georganic than DP-1, and sporulation per unit lesion area was greatest for Georganic. Enhanced field resistance to early and late leaf spots reported for DP-1 and Georganic is in part due to lower infection frequencies, smaller lesions, and for DP-1, longer latent periods.

Key Words: C11-2-39, partial resistance, field resistance, modeling.

Early leaf spot, caused by *Cercospora arachidicola* S. Hori, (teleomorph = *Mycosphaerella arachidis* Deighton) (*Ca*) and late leaf spot, caused by *Cercosporidium personatum* (Berk. & M. A. Curtis) Deighton, (teleomorph = *Mycosphaerella berkeleyi* Jenk.) (*Cp*) are the most important foliar diseases affecting peanut (*Arachis hypogaea* L.) throughout the world (Shokes and Culbreath, 1997). The diseases often occur together or one disease may be more predominant in a given location or year. In Georgia, late leaf spot was more common in the 1980s and early 1990s, while early leaf spot became predominant in much of Georgia during the late 1990s. Late leaf spot is still predominant in Florida and lately has again increased its prevalence in Georgia. For this reason it is critical to evaluate the resistance of runner-type peanut genotypes, the type most widely grown throughout Georgia, Alabama, and Florida, to

[1]Department of Biology, Valdosta State University, Valdosta GA 31698 (egcantonwine@valdosta.edu).

[2]Department of Plant Pathology, University of Georgia, Tifton, GA 31793-0748.

[3]USDA-ARS, Crop Genetics and Breeding Research Unit, Tifton, GA 31793-0748.

[4]Department of Agronomy, University of Florida NFREC, Marianna, FL 32446-7906.

*Corresponding author

combat the menace of both *Ca* and *Cp*. Fields tests can be used for evaluation of resistance to the prevalent pathogen, but greenhouse or growth chamber evaluations should be utilized when field evaluations to both pathogens are not feasible.

There has been no complete or single-gene resistance to *Ca* or *Cp* reported in cultivated peanut. Resistance is partial and rate-reducing (Abdou *et al.,* 1974). Partial resistance is typically a function of multiple components of resistance that contribute additively to a reduction in the rate of epidemic progress (Parlevliet, 1979). Components of resistance to early leaf spot (Foster *et al.,* 1980; Green and Wynne, 1986; Melouk and Banks, 1984; Ricker *et al.,* 1985) and/or late leaf spot (Chiteka *et al.,* 1988a; Cook, 1981; Subrahmanyam *et al.,* 1982; Walls *et al.,* 1985; Watson *et al.,* 1998) were described for many peanut genotypes under field and greenhouse conditions in the 1980s. However, relatively little work has been published for peanut cultivars and breeding lines developed within the past 20 years (Anderson *et al.,* 1993; Aquino *et al.,* 1995; Chiteka *et al.,* 1997; Chiyembekeza *et al.,* 1993), despite significant advances in breeding for resistance to leaf spot diseases during this time. Very little work has been accomplished to investigate the resistance of genotypes to both leaf spot pathogens (Walls *et al.,* 1985), even though the inheritance of resistance to each pathogen appears to be independent (Wynne *et al.,* 1991).

Variation exists for nearly all resistance components investigated for early and late leaf spots. These include infection frequency, incubation period (time from inoculation to symptom appearance), latent period (time from inoculation to first sporulating lesion), lesion size, percent necrotic leaf area, percent lesions with sporulation, spore production, and time to defoliation. Latent period, lesion size and spore production have been the components most commonly associated with genetic resistance (Chiteka *et al.,* 1988a; Chiteka *et al.,* 1988b; Chiyembekeza *et al.,* 1993; Walls *et al.,* 1985). Infection frequency is highly dependent upon temperature and relative humidity (Shew *et al.,* 1988; Waliyar *et al.,* 1994) and has been suggested to be an unreliable measure of resistance (Ricker *et al.,* 1985; Waliyar *et al.,* 1993). Examinations of multiple resistance components within resistance genotypes have shown that components are often correlated. For example, Jogloy *et al.* (1987) and Chiteka *et al.* (1988b) reported a positive genetic relationship between late leaf spot lesion size and the quantity of secondary spores produced, both of which were negatively correlated with latent period. However, no single component has been identified as a primary or consistent predictor of resistance in the field. The stability of resistance components to *Ca* can vary across growing regions (Chiteka *et al.,* 1997; Waliyar *et al.,* 1993) due to environmental interactions (Shew *et al.,* 1988; Waliyar *et al.,* 1994), pathogen populations (Waliyar *et al.,* 1993) or both (Chiteka *et al.,* 1997). However, Shew *et al.* (1989) reported stable response of peanut genotypes to *Cp* isolates from the U.S. and Thailand.

The cultivar Georgia Green (Branch, 1996) is currently the predominant runner-type cultivar grown in the southeastern U.S. (Smith, 2003). The levels of resistance to early and late leaf spots of Georgia Green are slightly better than that of Florunner (Norden *et al.*, 1969; Branch and Culbreath, 1995), which was used as a susceptible check in studies of components of resistance of runner-type genotypes conducted in the 1980s (Aquino *et al.*, 1995; Chiteka *et al.*, 1988a). In a recent field study (Cantonwine *et al.*, 2006), the field resistance to early leaf spot was assessed for Georgia Green and seven newly released cultivars or advanced breeding lines (C-99R, Hull, DP-1, GA-01R, C11-2-39, C28-305 and C34-24), all of which were developed for resistance to early and leaf spots and/or spotted wilt, caused by *Tomato spotted wilt virus*. Cultivar DP-1, released by the University of Florida peanut breeding program, had the best field resistance to early leaf spot among the genotypes tested (Cantonwine *et al.,* 2006), and reportedly has better resistance to late leaf spot than any cultivar currently available in the U.S. (Gorbet, 2003). The cultivar Georganic (C.C. Holbrook, unpubl. data, 2006), previously tested as breeding line C11-2-39, had an intermediate level of resistance relative to Georgia Green and DP-1 (Cantonwine *et al.,* 2006), and has been shown to have good field resistance to late leaf spot (Cantonwine *et al.,* 2003). This study was conducted to describe the temporal progression of leaf spot diseases in the field and some of the components of host resistance to *Ca* and *Cp* expressed in peanut cultivars Georgia Green, DP-1, and Georganic.

Materials and Methods

Evaluation of leaf spot epidemic progress in the field

A field experiment was carried out in 2002 and 2003 at the Coastal Plain Experiment Station Ridgon Farm in Tifton, GA to monitor temporal progress of naturally occurring leaf spot epidemics in plots planted with Georgia Green, DP-1, and Georganic peanuts. The experimental design was a randomized complete block with three replications of each genotype per year. Peanut seed were planted in 91-cm spaced single rows in convention-

ally tilled plots (1.8 × 6 m) on 17 May 2002 and 20 May 2003. No fungicides were applied. Herbicides, insecticides, and fertilizers were applied following recommendations of the University of Georgia Extension Service.

Disease incidence (the percentage of leaves with one or more lesions or defoliated leaflets) was assessed on leaves of 10 lateral branches (secondary branches 1-6) arbitrarily collected from each plot. Assessments began 78 d after planting (DAP) in 2002 and 59 DAP in 2003, and continued weekly for 6 wk. Disease severity was monitored over the season using the Florida 1 to 10 scale system, where 1 = no leaf spot; 2 = very few lesions on the leaves, none on the upper canopy; 3 = few lesions on the leaves, very few on the upper canopy; 4 = some lesions with more on the upper canopy, 5% defoliation; 5 = lesions noticeable even on upper canopy, 20% defoliation; 6 = lesions numerous and very evident on upper canopy, 50% defoliation; 7 = lesions numerous on upper canopy, 75% defoliation; 8 = upper canopy covered with lesions, 90% defoliation; 9 = very few leaves remaining and those covered with lesions, 98% defoliation; and 10 = plants completely defoliated and killed by leaf spot (Chiteka *et al.,* 1988a). Severity assessments were made at 7 to 22 d intervals four to five times beginning 89 DAP in 2002, and nine to ten times beginning 59 DAP in 2003. One less severity assessment was taken for Georgia Green plots because Georgia Green matures earlier than DP-1 and Georganic, and the plants in these plots were inverted 11 to 13 d earlier than in the DP-1 and Georganic plots. Genotype means of disease incidence and severity were plotted individually by assessment date of each year.

Disease incidence and severity data were analyzed separately. For each plot, area under the disease progress curve (AUDPC) was computed (Shaner and Finney, 1977). Disease assessments were converted to proportions [proportion of incidence = percent incidence/100; proportion of severity assessment = (Florida rating - 1)/9], and linearized forms of the Gompertz [-ln(-ln y)], logistic [ln(y/1-y)] and monomolecular [ln(1/1-y)] models were fit using linear regression of transformed disease intensity proportions on time (DAP), as described by Cantonwine et al., 2007. Time of disease onset (TDO) was estimated by calculating the time (DAP) when the model predicted 5% incidence or 1.5 on the Florida scale. Effects of genotype on AUDPC, TDO and the epidemic rate parameter (r) estimates were determined using the Proc MIXED procedure (SAS Version 8.3, SAS Institute, Cary, NC). When significant year by genotype interactions occurred, years were analyzed separately. Otherwise, analyses were conducted across years. Differences among genotype levels were determined using Fisher's LSD values using standard error and t-values of adjusted degrees of freedom when the main effect was significant ($P < 0.05$).

Evaluation of components of resistance to leaf spot pathogens

A growth chamber experiment was carried out to assess various components of resistance of the cultivars Georgia Green, DP-1, and Georganic to infection by Ca and Cp under a controlled environment. The experimental design was a randomized complete block, with 12 replications of each of the three genotype treatments. The experiment was conducted twice beginning 29 Nov. 2003 and 21 Apr. 2004.

Plants were grown in 15-cm pots with Promix potting soil and maintained in a greenhouse for 9 wk. Three young first or second fully expanded leaves were cut at the base of the petiole from lateral branches on each of 12 plants of each genotype. The cut ends were dipped in a dry formulation of napthaleneacetamide and thiram (Rootone, Security Products Co., Atlanta, GA) and placed individually in sterile saturated sand in 100-ml beakers (Waliyar et al., 1995).

For inoculations, conidia of Cp were acquired by agitating leaf discs of sporulating late leaf spot lesions from Georgia Green leaves that were collected in Tifton, GA and stored at 10°C for 3 to 9 mo in a solution of 0.005% Tween 20. Conidia of Ca were acquired by rinsing conidia from single-conidium cultures isolated from infected Georgia Green peanut leaves collected in Tifton, GA the previous growing season using a technique modified from Lu et al. (2003) and described by Cantonwine et al. (2007) using a 0.005% Tween 20 rinse. Conidial suspensions were standardized to a concentration of 1.0×10^4 conidia/ml and each leaf was inoculated by spraying for 1 sec with an aerosol spray bottle. A solution of 0.005% Tween 20 without conidia was the control. Viability of conidia from inoculation suspensions was verified by measuring spore germination on 2% water agar plates after 24 hr incubation at 22 C under incandescent light.

Leaves in beakers were randomly positioned on trays and placed within a transparent enclosure in a growth chamber at 24 C, 90% RH, and 12-hr photoperiod to provide optimal conditions for Ca germination and infection (Waliyar et al., 1995). The enclosure (1.3 × 0.7 × 0.6 m) was constructed using PVC pipe for the frame, clear plastic for the sides, and overlapping sheets of glass for the top. The enclosure did not affect air temperature. Relative humidity was supplemented by maintain-

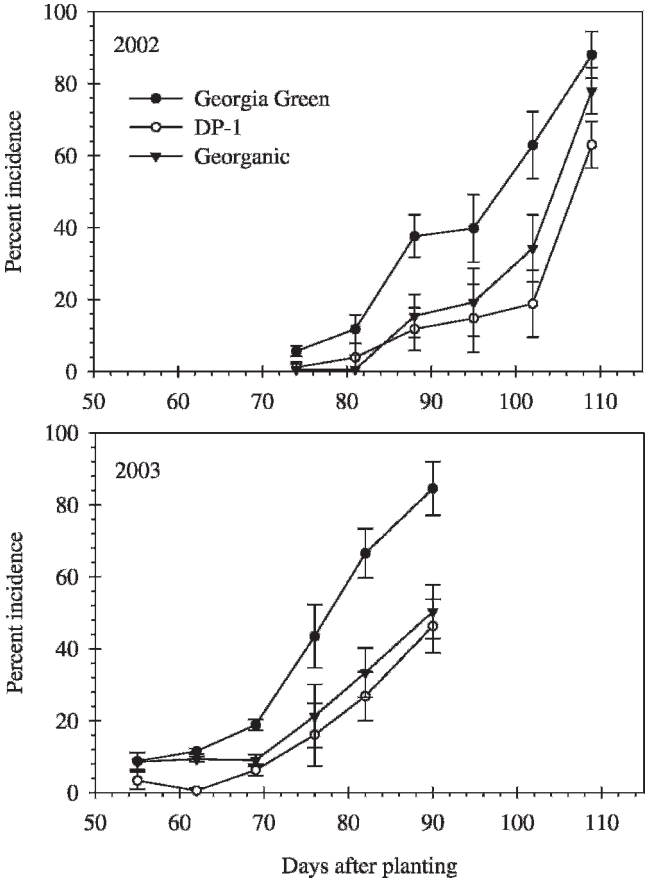

Fig. 1. Mean percent incidence of early leaf spot (diseased leaves of 10 lateral branches) in plots planted to three peanut genotypes at each assessment date in 2002 and 2003.

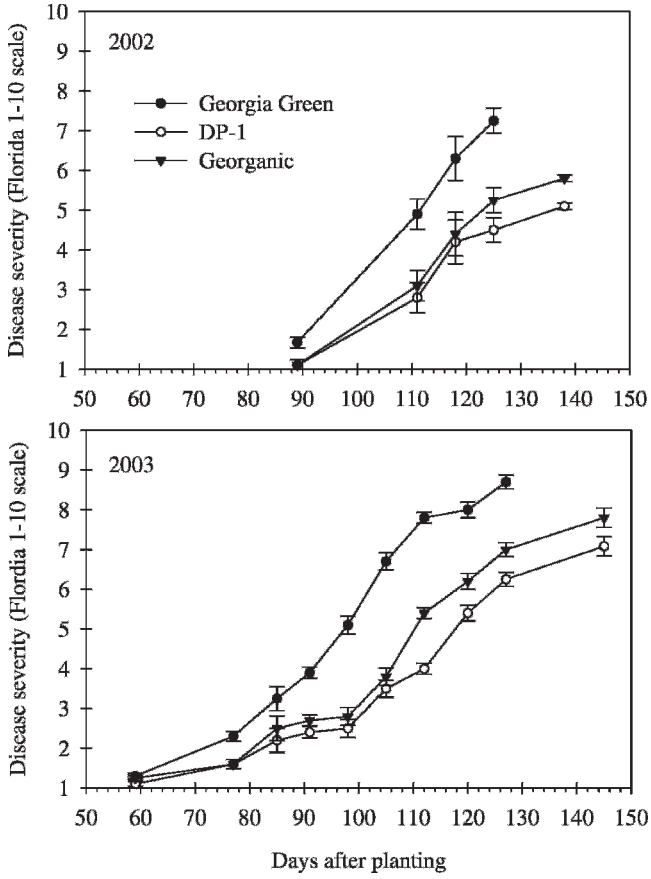

Fig. 2. Early leaf spot severity based on Florida 1 to 10 ratings at each assessment date for three peanut genotypes across replications in 2002 and 2003.

ing standing water in trays and with two humidifiers (PersonalMist ultrasonic humidifier, Kaz, Inc, Hudson, NY) evenly spaced within the enclosure. Water was added to beakers and trays as needed. Severely wilted leaves were excluded from measurements.

Lesion numbers per leaf were counted at 17 and 30 d after inoculation (DAI). The percent incubation period 17 DAI was computed by dividing the number of lesions from the 17 DAI count by the 30 DAI count and multiplying by 100. Latent period, defined as DAI until one lesion sporulated, was determined by inspecting spots daily with a 20X magnification lens beginning 17 DAI. After sporulation was observed on one lesion per leaf, or at 30 DAI if no sporulation was apparent, leaves were removed from the growth chamber and placed in a light box at 100% RH chamber for 72 hr, after which the percentage of spots with sporulation was determined. Latent period was not recorded for leaves if sporulation was not apparent 33 DAI. Three sporulating lesions per leaf were randomly selected, excised with a razor blade and placed in a 1-ml tube with 0.1 ml of 0.005% Tween 20 for spore quantification. The necrotic area of each selected lesion was measured. Number of spores per lesion, per unit sporulating lesion area, and per unit leaf area were determined. In cases where no sporulation occurred, the number of stroma was assessed. Percent necrotic area per leaf was estimated for a sub-sample of 10 to 50 randomly selected lesions per leaf. Infection frequency at 17 and 30 DAI was determined by dividing the number of lesions at each DAI by leaf area.

Effects of genotype on resistance variables and square root transformations of variables were tested using the Proc MIXED procedure. Due to unbalanced data, whenever the standard errors were reasonably similar, the largest standard error was used for means comparisons rather than computing a weighted standard error. Fisher's LSD values were computed using standard errors and t-values of adjusted degrees of freedom when the main effect was significant ($P < 0.05$). Comparisons of untransformed means of transformed variables were determined based on analysis of transformed variables, since variances cannot be transformed (Steel and Torrie, 1980).

Table 1. Parameters of models across replications that best described the progression of early leaf spot epidemics over time in the field for three peanut genotypes in 2002 and 2003.

Year	Incidence (logistic model)					Severity (linear model)				
	Intercept		Slope		Recalc. R^2	Intercept		Slope		Recalc.R^2
Genotype	Mean	SE	Mean	SE	Mean	Mean	SE	Mean	SE	Mean
2002										
Georgia Green	−15.19	2.74	0.158	0.030	0.76	−1.48	0.17	0.017	0.002	0.93
DP-1	−14.70	1.56	0.137	0.017	0.84	−0.83	0.10	0.010	0.001	0.90
Georganic	−21.98	2.37	0.214	0.026	0.89	−0.99	0.14	0.011	0.001	0.87
2003										
Georgia Green	−9.94	0.90	0.129	0.012	0.90	−0.84	0.06	0.014	0.001	0.95
DP-1	−12.06	1.96	0.132	0.027	0.81	−0.59	0.06	0.009	0.001	0.91
Georganic	−6.85	0.89	0.073	0.012	0.79	−0.66	0.06	0.010	0.001	0.92

All models significantly fit curves ($P \leq 0.03$).

Results

Evaluation of leaf spot epidemic progress in the field

Early leaf spot was the predominant disease in the field study. Late leaf spot occurred late in the season (>100 DAP) in both years, but the disease never exceeded 10% of the leaf spots combined. For this reason, the field epidemics will be referred to as early leaf spot.

Early leaf spot epidemics began later and were less severe in 2002 than in 2003 (Fig. 1–2). Disease progress curves for early leaf spot incidence varied by genotype (Fig. 1) and were best described by the logistic model ($P \leq 0.03$) (Table 1). Disease progress curves for severity also varied by genotype (Fig. 2) and were best described by the linear model ($P \leq 0.01$) (Table 1).

Across years, early leaf spot epidemics, measured as AUDPC based on incidence (AUDPC-I) and severity (AUDPC-S), were more severe for Georgia Green, than DP-1 and Georganic (Table 2). AUDPC-I was comparable between the two resistant genotypes, while AUDPC-S was significantly lower for DP-1 than Georganic. There was a significant interaction between year and genotype for incidence based estimates of TDO (TDO-I) and epidemic rate (rate-I). Estimates of TDO-I were 9 d later for DP-1 than Georgia Green both years, but this difference was only statistically significant in 2003 (Table 2). In 2002, TDO-I of Georganic was more similar to TDO-I of DP-1 than Georgia Green, while in 2003, TDO-I of Georganic was more similar to TDO-I of Georgia Green than DP-1. Estimates of rate-I were lower for Georganic than Georgia Green and DP-1 in 2002, and lower for DP-1 and Georganic than Georgia Green in 2003. Across years, severity based estimates of TDO were statistically later for DP-1 and Georganic than Georgia Green, and severity

Table 2. Effect of peanut genotype on early leaf spot disease progress variable assessed as incidence or severity, 2002–2003.

	Incidence[a] (%)					Severity[b]		
	Across years	2002		2003		Across years		
Genotype	AUDPC[c]	TDO[d]	Rate[d]	TDO[d]	Rate[d]	AUDPC[e]	TDO[f]	Rate[f]
Georgia Green	1361 A	77 A	0.158 A	54 A	0.129 A	4.6 A	77 A	0.016 A
DP-1	554 B	86 A	0.137 A	63 B	0.095 B	3.5 C	84 B	0.009 C
Georganic	746 B	89 A	0.214 B	52 A	0.079 B	3.9 B	83 B	0.011 B
Standard error	64.2	13.2	0.1063	8.8	0.0257	0.06	2.5	0.0004
P-value	< 0.01	0.13	< 0.01	0.05	0.01	< 0.01	< 0.01	< 0.01

[a]Number of leaves with leaf spot symptoms per total number of leaves averaged from 10 lateral branches arbitrarily sampled per plot.

[b]Florida 1 to 10 leaf spot severity scale.

[c]Least square means from Proc MIXED of area under the disease progress curve (AUDPC) assessed at a 7-d interval 74 to 109 d after planting (DAP) in 2002, and 55 to 90 DAP in 2003. Means within a column with the same letter do not differ at P=0.05.

[d]Estimated using linear regression of logit transformed data over time.

[e]Area under the disease progress curve was standardized by dividing by the number of days from the first to last rating assessed at a 7-d interval 89 to 138 DAP in 2002, and 59 to 145 DAP in 2003.

[f]Estimated using linear regression of severity over time.

Table 3. Components of symptom expression for detached peanut leaves inoculated with conidia of *Cercospora arachidicola*.

Genotype	Infection frequency 17-d[a]	Infection frequency 30-d[b]	Percent incubation 17-d[c]	Lesion diameter (mm) [d]	Percent necrotic leaf area[e]
Georgia Green	1.4 A	2.9 A	46.2 A	1.9 A	8.3 A
DP-1	1.2 A	2.1 B	58.1 A	1.6 B	4.4 B
Georganic	1.0 A	1.9 B	50.1 A	1.5 B	3.3 B
Standard error	0.22	0.33	6.97	0.10	1.03
P-value	0.24[f]	0.02[f]	0.47[g]	0.01[g]	< 0.01[g]

[a]Least square means from Proc MIXED of number of lesions per leaf area (cm^2), 17 d after inoculation (DAI). Means within a column with the same letter do not differ at P=0.05.
[b]Number of lesions per leaf area (cm^2), 30 DAI.
[c]Percent of lesions 30 DAI apparent 17 DAI.
[d]Based on sub-sample of 10 to 50 randomly selected lesions per leaf.
[e]Estimated as [(lesion number 30-DAI * $\pi(d / 2)^2$) / leaf area].
[f]From analysis of square root transformed data. Standard error shown is from the untransformed data and may not reflect the transformed analysis.
[g]From analysis of untransformed data.

based epidemic rates were highest for Georgia Green and lowest for DP-1 (Table 2).

Evaluation of components of resistance to leaf spot pathogens.

In the growth chamber experiment, leaf spot symptoms were observed on all inoculated leaves and none were observed on control leaves. After 24 hr, germination of *Ca* and *Cp* conidia of the inoculum suspensions were approximately 87 and 65%, respectively, in both trials. Across trials and pathogen exposures, five to seven leaves of each genotype were excluded from analyses due to severe wilting that appeared to impede lesion development. The pattern of wilting appeared to be random and the cause was not evident. Infection frequency of *Ca* did not differ among genotypes 17 DAI, but was higher for Georgia Green 30 DAI (Table 3). Percent incubation period 17 DAI did not differ among genotypes (Table 3). Lesion diameter was 16–21% greater for Georgia Green than for the other genotypes, while percent necrotic area was 47 and 60% greater for Georgia Green than for DP-1 and Georganic, respectively (Table 3). The latent period was 2 d longer for DP-1 and Georganic compared to Georgia Green, but no other differences in early leaf spot reproductive patterns were detected among genotypes (Table 4).

Infection frequency of *Cp* was significantly lower for DP-1 at 17 DAI, and for DP-1 and Georganic at 30 DAI compared to Georgia Green (Table 5). Percent incubation period at 17 DAI was lower for DP-1 than Georganic (Table 5). Lesion diameter for Georgia Green was 15% greater than Georganic and DP-1, while percent necrotic area was approximately 46% greater for Georgia Green than the other genotypes (Table 5). The latent period was more than 2 d shorter and percent sporulating lesions was greater for Georganic than DP-1 (Table 6). Spores per sporulating lesion area were significantly higher for Georganic than the other genotypes, but spores per leaf area were similar for all genotypes (Table 6).

Discussion

The field study provided a suitable setting to characterize field resistance of Georgia Green, DP-

Table 4. Components of *Cercospora arachidicola* reproductive patterns on detached peanut leaves.

Genotype	Latent period (d)[a]	Percent spots with sporulation[b]	Percent spots with stroma[b]	Stroma per lesion area (cm^2)[b]	Stroma per leaf area (cm^2)[b]
Georgia Green	22.1 B	48.1 A	34.5 A	1120 A	6.2 A
DP-1	24.4 A	32.6 A	40.4 A	980 A	3.0 A
Georganic	24.4 A	30.2 A	31.5 A	1120 A	4.1 A
Standard error	0.42	57	8.58	225	1.41
P-value[c]	< 0.01	0.14	0.76	0.87	0.27

[a]Least square means from Proc MIXED of days after inoculation until first lesion sporulating. Means within a column with the same letter do not differ at P=0.05 based on Fisher's LSD values.
[b]Data from one trial.
[c]From analysis of untransformed data.

Table 5. Components of symptom expression for detached peanut leaves inoculated with conidia of *Cercosporidium personatum*.

Genotype	Infection frequency 17-d[a]	Infection frequency 30-d[b]	Percent incubation 17-d[c]	Lesion diameter (mm)[d]	Percent necrotic leaf area[e]
Georgia Green	2.5 A	5.4 A	43.5 AB	1.3 A	7.1 A
DP-1	1.3 B	3.9 B	35.0 B	1.1 B	3.7 B
Georganic	2.6 A	3.8 B	52.1 A	1.1 B	3.9 B
Standard error	0.16	0.47	3.65	0.38	0.60
P-value	0.05[f]	< 0.01[f]	< 0.01[g]	< 0.01[g]	< 0.01[g]

[a]Least square means from Proc MIXED of number of lesions per leaf area (cm^2), 17 d after inoculation (DAI). Means within a column with the same letter do not differ at P=0.05 on Fisher's LSD values.
[b]Number of lesions per leaf area (cm^2), 30 DAI.
[c]Percent of lesions 30 DAI apparent 17 DAI.
[d]Based on sub-sample of 10 to 50 randomly selected lesions per leaf.
[e]Estimated as [(lesion number 30-DAI * $\pi(d / 2)^2$) / leaf area].
[f]From analysis of square root transformed data. Standard error shown is from the untransformed data and may not reflect the transformed analysis.
[g]From analysis of untransformed data.

1 and Georganic to *Ca* but not to *Cp*, since early leaf spot was the predominant disease both years. Differences in the onset and severity of early leaf spot epidemics by year can be attributed in part to 60% less rainfall during the growing season in Tifton, GA in 2002 than in 2003. Genotype ranking of field resistance to early leaf spot was Georgia Green most susceptible, Georganic moderately resistant, and DP-1 most resistant. These results corroborate those observed in previous field trials (Cantonwine *et al.,* 2003, 2006). The observed delay of TDO for DP-1 and Georganic compared to Georgia Green was not always consistent, but it suggests that fewer initial infections may have occurred for these genotypes. Although it is likely that the TDO differences detected for the selected disease intensities (5% incidence and 1.5 on Florida scale) were confounded by differences in the rate of disease increase, data from the growth chamber studies suggest there might be a true delay in TDO for DP-1 and Georganic since infection frequency was shown to be reduced by these genotypes. A preliminary experiment to evaluate germination rates of *Ca* and *Cp* on leaf surfaces of these genotypes suggest that germination rates of *Ca* may be suppressed on DP-1 leaves compared to leaves of Georgia Green or Georganic (Cantonwine, 2005).

Reduced infection frequencies likely also contributed to the reduced rates of epidemic progress that were observed for Georganic and DP-1. Genotype ranking by the epidemic rate parameter estimate corresponded more closely to the ranking observed by AUDPC of incidence and severity than did the ranking by disease onset. This is not surprising since the length of time that resistance factors affect disease intensity is greater for measures of rate of increase and AUDPC than disease onset. This result also supports the concept that partial resistance primarily suppresses epidemics by causing a reduction to the rate of disease increase (Parlevliet, 1979).

Infection frequency, reported as an unreliable resistance component for *Ca* (Ricker *et al.,* 1985;

Table 6. Components of *Cercosporidium personatum* reproductive patterns on detached peanut leaves.

Genotype	Latent period (d)[a]	Percent spots sporulating	Spores per sporulating lesion area (cm^2)[b]	Spores per leaf area (cm^2)[b]
Georgia Green	24.7 BC	24.4 AB	18400 B	330 A
DP-1	26.0 AB	19.4 B	23700 B	190 A
Georganic	23.4 C	32.7 A	50300 A	570 A
Standard error	0.56	4.09	7250	115
P-value	< 0.01[c]	0.04[d]	< 0.01[c]	0.08[c]

[a]Least square means from Proc MIXED of days after inoculation until first lesion sporulating. Means within a column with the same letter do not differ at P=0.05 based on Fisher's LSD values.
[b]Spore quantification estimated from three randomly selected sporulating lesions.
[c]From analysis of untransformed data.
[d]From analysis of square root transformed data. Standard error shown is from the untransformed data and may not reflect the transformed analysis.

Waliyar *et al.,* 1993), appeared to be an important component of resistance for comparison of genotypes in this study. It is possible that the environmental conditions selected for the growth chamber study, 24 C and high RH, were more suitable to measure differences of infection frequency than a more variable greenhouse environment, or different regime. The frequency and variation of conidial germination rates and infections was found to be greater for *Ca* under 24/24 C and 26/20 C day/night regimes, than 32/26, 38/26, and 38/32 C regimes (Waliyar *et al.,* 1994, 1995), and for *Cp* at 20, 24 and 28 C, compared to 32 C (Shew *et al.,* 1988). In addition to lower infection frequencies, components of resistance to *Ca* measured for DP-1 and Georganic were smaller lesion diameters, reduced percent necrotic areas, and prolonged latent periods. Inconsistencies in measurement techniques throughout the literature and lack of a consistent reference cultivar make comparisons of these results to others difficult. However, our results appear to corroborate those reported previously. The mean size of early leaf spot lesions in this study was similar to the range of diameters of *Ca* lesions plus necrotic areas surrounding lesions (halo) (approximately 1.7 to 1.8 mm) for the most resistant virginia-type peanut genotypes observed by Waliyar *et al.* (1994) under the same temperature regime. The means of early leaf spot latent period for DP-1 and Georganic were equal to the T_2 latent period (days until two *Ca* lesions sporulated) observed for NC 3033, the virginia-type genotype with the longest T_2 latent period of the genotypes investigated by Ricker *et al.* (1985). In this study, it was common to find 2 or more lesions with sporulation per leaf on the first day that sporulation was noticed. Measurements of components affecting secondary reproduction of *Ca* did not differ among genotypes. However, various obstacles to the collection of these data may have inhibited our ability to detect differences for these variables. Sporulation did not occur in trial 1. Instead lesions with stroma and total number of stroma per lesion were counted. Stroma formation is a precursor to sporulation of *Ca* and *Cp* lesions (Abdou *et al.,* 1974). Abdou *et al.* (1974) noticed less defined stroma development within lesions from moderately susceptible *Arachis* species than highly susceptible species. Estimates of stroma per leaf area did not differ among genotypes, even though the means were 50 and 33% greater for Georgia Green than for DP-1 and Georganic, respectively. In trial 2, sporulation was common for early leaf spots, but spore concentrations were not measured due to contamination of spore quantification vials that resulted in degradation of spores.

Although not assessed in this study, Georganic and DP-1 have been shown to have more field resistance to late leaf spot than Georgia Green (A.K. Culbreath, unpubl. data, 2001; C.C. Holbrook, unpubl. data, 2006). The growth chamber study suggests their resistance is due to reduced infection frequency, smaller lesion size and less percent necrotic area (Tables 5,6). The mean *Cp* lesion size for all genotypes in this study was smaller than for the most resistant genotypes reported by Chiteka *et al.* (1988a) or Aquino *et al.* (1995). Since neither of these earlier studies included Georgia Green, it is unclear whether genetic variation is responsible for the lesion diameter discrepancy observed between the genotypes tested in our study and those tested by Chiteka *et al.* (1988a) and Aquino *et al.* (1995), since factors other than genetics have been shown to influence lesion size. Temperature does not appear to be the cause of the lesion diameter discrepancy since the temperatures in Chiteka's greenhouse trials (19.8 to 30.8 C in one trial, mean daily temperatures 27 to 34 C in a second trial) may have been less favorable for lesion expansion than 24 C in this study (Shew *et al.,* 1988). It is possible that the high mean *Cp*-induced lesions per leaf in this study, which ranged from 55.5 to 101.2 per leaf, limited the expansion potential of *Cp* lesions. This type of negative interaction between lesion number and lesion size interaction was reported for *Ca* (Johnson *et al.,* 1986). The mean number of *Cp* lesions per leaf in Chiteka's study ranged from 8.6 to 13.0, but was not reported by Aquino *et al.* (1995). The latent periods for the genotypes in this study fell within the range observed by Chiteka *et al.* (1988a), 12 to 35.0 DAI, and Aquino *et al.* (1995), 21.8 to 30.8 DAI. Ricker *et al.* (1985) reported that percent sporulating lesions was a component of resistance for *Ca*, but that it did not appear to be a resistance component for *Cp*. We found the opposite trend with the genotypes in our study, but it is important to point out that *Ca* sporulation data were only recorded for the second trial, and therefore was not repeated.

Overall, the reproduction potential of *Cp* was greatest for Georganic of the genotypes tested. Although *Cp* spore production per unit leaf area was not statistically different among genotypes ($P = 0.07$), they were more similar for Georganic and Georgia Green than for DP-1 (Table 6). Typically, as lesion size decreases in response to host genetics, spore production decreases and latent period increases (Chiteka *et al.,* 1988a; Jogloy *et al.,* 1987). This trend was observed for all genotypes for *Ca*, and for Georgia Green and DP-1 for *Cp*, but Georganic presented relatively small *Cp* lesions, more *Cp* sporulation and short *Cp* latent periods. It

is possible that the temperature regime chosen in this study was more conducive for sporulation of *Cp* on Georganic than the other genotypes. Interactions between genotype and temperature have been observed with early leaf spot for incubation period, infection frequency, lesion diameter and latent period (Shew *et al.,* 1988; Waliyar *et al.,* 1994), although no genotype and temperature interactions were observed in the cited studies for spore production. These results suggest that the relative field resistance of Georganic to late leaf spot, compared to Georgia Green or DP-1, may interact with time of disease onset (time available for secondary infection cycles). Information on timing and quantification of secondary inoculum production under field conditions could help assess the precision of the results observed in the growth chamber study, and their effect in the field.

In conclusion, results from the growth chamber study support the field results that DP-1 and Georganic have greater resistance to *Ca* than Georgia Green. The components of resistance to *Ca* that were assessed in this study did not distinguish the resistance of DP-1 from Georganic, despite resistance differences observed in the field for these genotypes. Although not directly comparable, the resistance components measured in this study were within the ranges reported for the most resistant peanut genotypes previously reported. This suggests that the partial resistance to *Ca* and *Cp* in DP-1 and Georganic is at least in part a function of resistance components that have been previously reported.

Acknowledgments

The authors wish to thank Y.J. Lu for assistance with culture preparation, B. Wilson for assistance with the growth chamber, M. Heath, S. Gremillion, K. Parrish, S. Carter and A. McKeown for general assistance, and B. Mullinix, Jr. for statistical help.

Literature Cited

Abdou, Y.A.-M., W.C. Gregory, and W.E. Cooper. 1974. Sources and nature of resistance to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Beck and Curtis) Deighton in *Arachis* species. Peanut Sci. 1:6-11.

Anderson, W.F., C.C. Holbrook, and T.B. Brenneman. 1993. Resistance to *Cercosporidium personatum* within peanut germplasm. Peanut Sci. 20:53-57.

Aquino, V.M., F.M. Shokes, D.W. Gorbet, and F.W. Nutter Jr. 1995. Late leaf spot progression on peanut as affected by components of partial resistance. Plant Dis. 79:74-78.

Branch, W.D. 1996. Registration of 'Georgia Green' peanut. Crop Sci. 36:806.

Branch, W.D. and A.K. Culbreath. 1995. Combination of early maturity and leaf spot tolerance within an advanced Georgia peanut breeding line. Peanut Sci. 22:106-108.

Cantonwine, E.G., A.K. Culbreath, C.C. Holbrook, W.D. Branch, and D.W. Gorbet. 2003. Field response of runner-type genotypes to multiple diseases under conventional and strip-tillage, 2002. Biol. and Cult. Tests for Control of Plant Dis. (online.) Report 18:P009. DOI: 10.1094/BC18. The Amer. Phytopath. Soc., St. Paul, MN.

Cantonwine, E.G. 2005. The use of genetics and cultural practices to suppress foliar diseases of peanut and reduce fungicide requirements. Ph.D. thesis, University of Georgia, Athens.

Cantonwine, E.G., A.K. Culbreath, K.L. Stevenson, R.C. Kemerait, Jr., T.B. Brenneman, N.B. Smith, and B.G. Mullinix, Jr. 2006. Integrated disease management of leaf spot and spotted wilt of peanut. Plant Dis. 90:493-500.

Cantonwine, E.G., A.K. Culbreath, and K.L. Stevenson. 2007. Characterization of early leaf spot suppression by strip-tillage in peanut. Phytopathology 97:187-194.

Chiteka, Z.A., D.W. Gorbet, F.M. Shokes, T.A. Kucharek, and D.A. Knauft. 1988a. Components of resistance to late leaf spot in peanut I. Levels of variability-implications for selection. Peanut Sci. 15: 25-30.

Chiteka, Z.A., D.W. Gorbet, D.A. Knauft, F.M. Shokes, and T.A. Kucharek. 1988b. Components of resistance to late leafspot in peanut II. Correlations among components and their significance in breeding for resistance. Peanut Sci. 15:76-81.

Chiteka, Z.A., D.W. Gorbet, F.M. Shokes, and T.A. Kucharek. 1997. Components of resistance to early leaf spot in peanut-genetic variability and heritability. Soil Crop Sci. Soc. Florida Proc. 56: 63-68.

Chiyembekeza, A.J., D.A. Knauft, and D.W. Gorbet. 1993. Comparison of components of resistance in peanut to late leafspot in different environments. Crop Sci. 33:994-997.

Cook, M. 1981. Susceptibility of peanut leaves to *Cercosporidium personatum*. Phytopathology 71:787-791.

Foster, D.J., M.K. Beute, and J.C. Wynne. 1980. Spore production and latent period as mechanisms of resistance to *Cercospora arachidicola* in four peanut genotypes. Peanut Sci. 7:88-89.

Gorbet, D.W. 2003. New University of Florida peanut varieties for 2003. 03-2, Marianna NFREC Research Report, Marianna.

Green, C.C. and J.C. Wynne. 1986. Field and greenhouse evaluation of the components of partial resistance to early leafspot in peanut. Euphytica 35:561-573.

Jogloy, S., J.C. Wynne, and M.K. Beute. 1987. Inheritance of late leafspot resistance and agronomic traits in peanut. Peanut Sci. 14:86-90.

Johnson, C.S., M.K. Beute, and M.D. Ricker. 1986. Relationship between components of resistance and disease progress of early leaf spot on virginia-type peanut. Phytopathology 76:495-499.

Lu, Y.J., B.B. Shew, and H.L. Ypema. 2003. In vitro sporulation of *Cercospora arachidicola* from peanut and sensitivity of spore germination to pyraclostrobin. Published online at http://www.apsnet.org/meetings/2003/abstracts/a03ma394.htm.

Melouk, H.A. and D.J. Banks. 1984. Assessment of resistance to *Cercospora arachidicola* in peanut genotypes in field plots. Plant Dis. 68:395-397.

Norden, A.J., R.W. Lipscomb, and W.A. Carver. 1969. Registration of 'Florunner' peanut. Crop Sci. 9:850.

Parlevliet, J.E. 1979. Components of resistance that reduce the rate of epidemic development. Annu. Rev. Phytopathol. 17:203-222.

Ricker, M.D., M.K. Beute, and C.L. Campbell. 1985. Components of resistance in peanut to *Cercospora arachidicola*. Plant Dis. 69: 1059-1064.

Shaner, G. and R.E. Finney. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. Phytopathology 67:1051-1056.

Shew, B.B., M.K. Beute, and J.C. Wynne. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shew, B.B., T. Sommartya, and M.K. Beute. 1989. Components of partial resistance in peanut genotypes to isolates of *Cercosporidium personatum* from the United States and Thailand. Phytopathology 79:136-142.

Shokes, F.M. and A.K. Culbreath. 1997. Early and late leaf spots, pp. 17-20. *In* N. Kokalis-Burelle, D.M. Porter, R. Rodriguez-Kabana, D.H. Smith, and P. Subrahmanyam (eds.). *Compendium of Peanut Diseases, 2nd Ed.* APS Press, St. Paul, MN.

Smith, N.B. 2003. Peanut production survey state results, 2003. Univ. Georgia. Coop. Ext. Publ., Econ. College of Agricultural and Environmental Sciences.

Steel, R.G.B. and J.H. Torrie. 1980. Principles and Procedures of Statistics., McGraw-Hill, New York.

Subrahmanyam, P., D. McDonald, R.W. Gibbons, S.N. Nigam, and D.J. Nevill. 1982. Resistance to rust and late leafspot diseases in some genotypes of *Arachis hypogaea*. Peanut Sci. 9:6-10.

Waliyar, F., D. McDonald, P.V. Subba Rao, and P.M. Reddy. 1993. Components of resistance to an Indian source of *Cercospora arachidicola* in selected peanut lines. Peanut Sci. 20:93-96.

Waliyar, F., B.B. Shew, H.T. Stalker, T.G. Isleib, R. Sidahmed, and M.K. Beute. 1994. Effect of temperature on stability of components of resistance to *Cercospora arachidicola* in Peanut. Phytopathology 84:1037-1043.

Waliyar, F., B.B. Shew, R. Sidahmed, and M.K. Beute. 1995. Effects of host resistance on germination of *Cercospora arachidicola* on peanut leaf surfaces. Peanut Sci. 22:154-157.

Walls, S.B., J.C. Wynne, and M.K. Beute. 1985. Resistance to late leafspot of peanut of progenies selected for resistance to early leafspot. Peanut Sci. 12:22-27.

Watson, G.R., T.A. Kucharek, and F.M. Shokes. 1998. Components of resistance in peanut to late peanut leaf spot. Soil and Crop Sci. Soc. Florida Proc. 57:87-91.

Wynne, J.C., M.K. Beute, and S.N. Nigam. 1991. Breeding for disease resistance in peanut (*Arachis hypogaea* L.). Annu. Rev. Phytopathol. 29:279-303.