Effects of Monoculture with Susceptible and Resistant Peanuts on the Virulence of *Cylindrocladium crotalariae*

M. C. Black, M. K. Beute, and K. J. Leonard

Former research assistant, professor, USDA, ARS, and plant pathologist, respectively, Department of Plant Pathology, North Carolina State University, Raleigh 27650. Current address of the first author: Texas A&M University Agricultural Research and Extension Center, Uvalde 78801.

Journal Series Paper 8866 of the North Carolina Agricultural Research Service, Raleigh.

Use of trade names does not imply endorsement by the North Carolina Agricultural Research Service of the products named or criticism of similar ones not mentioned.

Mention of a trademark or proprietary product does not constitute a guarantee or warranty of the product by the U.S. Department of Agriculture or criticism of similar ones not mentioned.

The technical assistance of Joyce Hollowell, Andy Martin, and Kevin Jones is gratefully acknowledged.

Accepted for publication 8 March 1984.

ABSTRACT

Black, M. C., Beute, M. K., and Leonard, K. J. 1984. Effects of monoculture with susceptible and resistant peanuts on the virulence of *Cylindrocladium crotalariae*. Phytopathology 74: 945-950.

Fifteen replicated populations of *Cylindrocladium crotalariae* were established in field microplots and exposed to a 3-yr monoculture of susceptible cultivar Florigiant or resistant cultivar NC 3033 peanuts (*Arachis hypogaea*). Twelve of the populations were composed of single isolates and three were composites of either six or 12 individual isolates. Six populations were studied for a fourth year. Mean inoculum density was lower following monoculture with NC 3033 than with Florigiant. Some populations were consistently high or low in virulence on both cultivars over years. In three populations, root rot relative to the cultivar × year mean decreased over years on NC 3033. In one of these three populations, root rot severity also decreased over years on Florigiant. Root rot for one individual isolate (population 617) on NC 3033 was near the cultivar × year mean over years 1–2, but root rot severity over years 3–4 was greater than the mean, indicating that resistance had been overcome. However, monoculture of resistant NC 3033 in microplots infested with three composite populations showed that quantitative resistance in peanut was stable for heterogeneous populations of *C. crotalariae*.

The Cylindrocladium black rot disease caused in peanuts (*Arachis hypogaea* L.) by *Cylindrocladium crotalariae* (Loos) Bell and Sobers was first diagnosed in Georgia in 1965 (3). The fungus has spread into all peanut production areas in the southeastern United States but is most serious in North Carolina and Virginia. Genotypes of *A. hypogaea* with high levels of resistance were identified soon after the pathogen became established (20), but this level of resistance is linked with undesirable agronomic traits (8). Breeding efforts have successfully combined a moderate level of resistance with acceptable seed size and yield, resulting in the release of NC 8C in 1982 (19).

No single method is highly effective for control of Cylindrocladium black rot on peanuts. Management practices that reduce disease include control of nematodes that enhance disease development and rotations with nonhosts (7,12). Peanuts in North Carolina and Virginia are routinely grown in rotations with corn, cotton, and/or tobacco (peanuts once every 2–3 yr or more) for control of *C. crotalariae*, other pathogens, and insects. Combining moderate resistance with soil fumigation reduced Cylindrocladium black rot in one season (1), but it is not known if fumigation will always provide financial return due to market factors and weather-

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

related yield fluctuations from year to year.

Resistance to *C. crotalariae* depends on inoculum density (7), and all peanut lines tested have developed some root rot (20). Resistance is quantitatively inherited (8,10) and is of a type thought to be stable (18). No races of *C. crotalariae* have been found (4,9,16), but some isolates obtained from lesions on roots of resistant plants were more adapted to resistant NC 3033 than others (4,9).

Hadley et al (9) designated virulence of *C. crotalariae* expressed against all peanut genotypes as "general virulence," and virulence expressed only against one genotype or only against genotypes with the same genes for resistance as "specific virulence." Isolates from roots of resistant NC 3033 seedlings and from microsclerotia in field soil following harvest of NC 3033 had high general virulence (4,6). Therefore, if loss of effectiveness of polygenic resistance in NC 3033 peanuts to *C. crotalariae* should occur, Vanderplank's concept of "weak" versus "strong" monogenic resistance genes (18) may apply also for polygenic resistance genes. However, proper management might indefinitely prolong cultivar effectiveness even if resistance in peanuts can be overcome by this fungus.

The purpose of the monoculture experiment reported here was to severely "abuse" peanut resistance and to attempt selection of pathogen populations with increased virulence. Our approach was to infest field microplots with several individual and composited populations of *C. crotalariae* and to determine whether monoculture of resistant and susceptible cultivars resulted in selection pressure for altered levels of virulence. Realistic rotations of hosts and nonhosts of *C. crotalariae* in infested soil were studied concurrently (6).

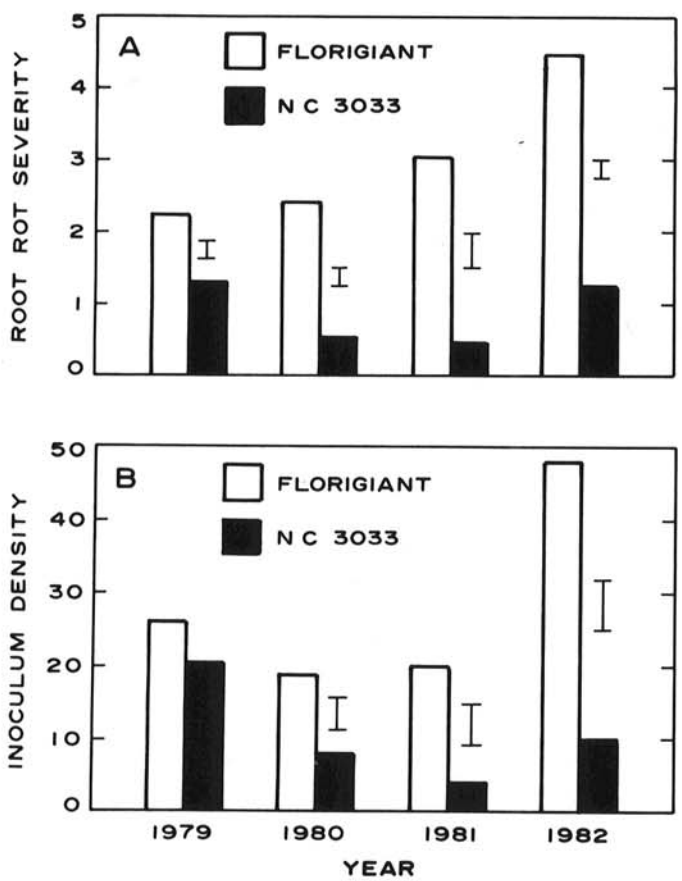

Fig. 1. General response of susceptible peanut cultivar Florigiant and resistant cultivar NC 3033 to *Cylindrocladium crotalariae*: **A,** root rot severity (0–5 scale) and **B,** postseason inoculum density determined in January following the indicated crop year. The initial inoculum density in 1979 was 35 microsclerotia per gram of soil. Bar represents Fisher's least significant difference (LSD) at $P = 0.05$. Probability of difference between cultivars was $P = 0.08$ for 1979 postseason inoculum density.

MATERIALS AND METHODS

Twelve isolates of *C. crotalariae* were chosen for this study from among 145 isolates evaluated in the greenhouse (4). All but one of those 145 isolates induced more root rot on cultivar Florigiant than on cultivar NC 3033, but six isolates included in this study had greater-than-average virulence on resistant NC 3033 and six had greater-than-average virulence on susceptible Florigiant (4). Microsclerotia for inoculum were increased in potato-dextrose agar (PDA) in petri plates in darkness for 5 wk. The number of microsclerotia produced by each isolate was estimated from a random sample of plates (14). Inoculum for field microplots was prepared by blending PDA cultures in water for 1 min at high speed in an Oster blender (Oster Corp., 5055 N. Lydell Ave., Milwaukee, WI 53217) without sieving. The microslcerotia-PDA-mycelium suspension was dispensed in 76-cm-diameter field microplots (2) and mixed to a depth of 20 cm with shovels to obtain an inoculum density of ~35 microsclerotia per gram of soil.

Microplots were established in Norfolk loamy sand soil at North Carolina State University Central Crops Research Station, Clayton, NC, in April 1979. Inoculum was placed in the soil 2–3 June 1979. Each microplot afforded above- and below-ground barriers to movement of microsclerotia (2). No attempt was made to keep inoculum density constant after the initial infestation. Planting dates were 4 June 1979, 20 May 1980, 15 May 1981, and 16 May 1982.

Successive plantings (monoculture) of either resistant NC 3033 or susceptible Florigiant were grown in the microplots from 1979 to 1982. The experimental design was a split plot of four replications with pathogen populations as main plots and cultivars as subplots in adjacent microplots. Main plots consisted of 12 populations of individual isolates, Composite-12 was composed of all isolates, Composite-3033 was composed of six isolates (205, 511, 508, 515, 617, and 621) selected for specific virulence on NC 3033 in the greenhouse, and Composite-Flor was composed of six isolates (110, 207, 402, 519, 603, and 612) selected for specific virulence on Florigiant.

Recommended management practices were followed (17), including recommended fertilizer and lime, alachlor as herbicide, carbofuran as insecticide/nematicide, methomyl and carbaryl as insecticides, and chlorothalonil as leafspot fungicide. In 1979–1981, seedlings were thinned to three plants per microplot after emergence. Six plants were grown per microplot in 1982. Landplaster (1,120 kg $CaSO_4$/ha) was applied to the soil surface in July each year. Pathogen populations in each microplot were maintained separately for the first three seasons.

In 1982, only six of the original populations were studied. Populations Composite-12 and 612 were well adapted on cultivar Florigiant, and 515 and 621 were well adapted on NC 3033 based upon symptoms in 1979–1981. Populations 617 and 205 were among populations well adapted on both hosts by 1981. Before planting in 1982, soil from replicate plots for each of the 12 treatments of interest (six populations, monoculture of two hosts) was removed to a depth of 50 cm and soil for replications was mixed for each treatment and placed back in the microplots.

Root rot was visually assesssed in late October on a 0–5 scale with 0 = no lesions and 5 = completely rotted roots (16). Soil was collected from each microplot with a soil sampling tube (vertical cores, 2 × 15 cm). Inoculum densities were determined and a greenhouse assay was conducted on two sets of samples. Samples in January 1980 and 1981 were ~2.5 kg per plot. Samples in May 1981 and January 1982 and 1983 were ~0.5 kg (only for inoculum density). Postseason (January each year) and preseason (May 1981) inoculum densities were estimated by elutriation and dilution plating (13).

A greenhouse assay of soil in each microplot was conducted with susceptible Florigiant and resistant NC 3033 as host differentials following the 1979 and 1980 seasons. Soil from each microplot (unadjusted for inoculum density) was packed into eight cylinders (inside diameter 3.5 cm, length 15.5 cm) and subirrigated (4). Single seedlings of Florigiant or NC 3033 were transplanted into four cylinders each for every soil sample. Root rot severity was evaluated at 5 wk.

RESULTS

Overall, root rot was less severe and inoculum density was lower following monoculture with resistant NC 3033 than with susceptible Florigiant (Fig. 1). Difference in overall mean root rot and inoculum density between cultivars increased each year. Generally, root rot severity and inoculum density before or after the season were not highly correlated (Table 1 and Fig. 2). Overall, root rot on Florigiant was more severe in each successive year. Root rot and inoculum density (Fig. 2) are expressed as deviations from the cultivar × year means found in Fig. 1. Populations that caused low root rot severity on Florigiant compared to other populations in 1979 and 1981 also caused low root rot severity on NC 3033 (nonsignificant population × cultivar interaction) (Fig. 2G). The relative difference in root rot in 1980 and 1982 between susceptible Florigiant and resistant NC 3033 was not consistent (significant interaction) and for some populations, root rot on the two cultivars was similar. However, root rot on NC 3033 for any population was never greater than on Florigiant (Fig. 2).

Root rot caused by pathogen populations was either less than, greater than, or inconsistent when compared to the cultivar × year means (Fig. 2). Populations 110, 519, and 515 consistently caused the least root rot on both cultivars. Population 207 caused a low level of root rot on Florigiant but was near average for NC 3033. Populations 508 and Composite-Flor consistently caused severe root rot on Florigiant. Over 3 yr, root rot caused by 508 and Composite-Flor on NC 3033 was near the means or above. Root rot caused by 621 was low on Florigiant, but inconsistent on NC 3033 (Fig. 2).

Populations of *C. crotalariae* Composite-12 and 612 induced low root rot severity over years on NC 3033. Root rot induced by Composite-3033 tended to decrease in severity over years on both cultivars. Populations 511 and 205 varied about the means in severity of root rot on both cultivars (Fig. 2).

Root rot severity on NC 3033 for population 617 was near the means for 1979 and 1980, then dramatically increased in 1981 and 1982. Root rot severity for 617 on Florigiant was near or above the means for all years (Fig. 2).

Soil was assayed in the greenhouse by growing seedlings of Florigiant and NC 3033 (as differential cultivars) in soil sampled in January of 1980 and 1981 from each microplot. Average seedling root rot on both differentials was more severe following harvest of NC 3033 than of Florigiant at similar inoculum densities, especially after 2 yr of monoculture. A third greenhouse assay of soil from selected microplots in April 1983 indicated that microsclerotia for population 617 mostly had high general virulence on both differentials.

If inoculum density estimated in January 1981 was low (<20 microsclerotia per gram), some of these same plots had inoculum density higher in June 1981 than in January (Fig. 3). If inoculum density in January was high (>20 microsclerotia per gram), inoculum density in June for the same plots was always lower. Although the host × sampling date interaction was not significant, more microplots in which NC 3033 was grown had low inoculum densities at both dates, and more microplots in which Florigiant was grown had high inoculum densities in January.

DISCUSSION

The lower overall root rot severity and postseason inoculum density of *C. crotalariae* for NC 3033 than for Florigiant peanuts (Fig. 1) corroborates previous observations (7,12,20). The difference between the cultivars in root rot severity over years probably would have been even greater if a lower initial inoculum density, nearer typical levels in peanut fields (13), had been used.

Other work showed that an increase in inoculum density in a high-inoculum-density range (such as occurred in most microplots of this experiment) did not result in a large increase in root rot severity (5). Also, postharvest inoculum densities were probably influenced by factors other than root rot severity at harvest, eg, time of lesion initiation and location of initial lesions on the root system. Therefore, this discussion concerns mostly the observed variation in root rot severity.

Distinction was not made between specific and general virulence in these monocultures because specific virulence can be detected only when isolates are evaluated at identical inoculum densities on differential cultivars (4,9). Monoculture did affect inoculum densities (Figs. 1 and 2) and perhaps also microsclerotial size (5) and gene frequencies for virulence (4,9). Greenhouse assays indicated that monoculture affected general virulence to a greater extent than specific virulence. Variation in root rot due to specific virulence in the greenhouse assays of isolates prior to this field experiment was not closely related to variation in root rot severity in the field microplots. Therefore, unless specified otherwise, reference to virulence in this discussion will refer to general virulence, ie, effective on all cultivars.

Population Composite-Flor had high virulence on both cultivars, whereas population 110, 519, and 515 had low virulence on both cultivars throughout this study (Fig. 2). Populations 207 and 205 also had low to near-average virulence on both cultivars. On the other hand, virulence in other populations increased on only one cultivar. Population 617 had low virulence for NC 3033 when this experiment was initiated. In the fourth year of monoculture with resistant NC 3033, population 617 induced much greater than average root rot severity on NC 3033, but not Florigiant. However, populations Composite-12 and Composite-3033 (each of which contained isolate 617) initially induced severe root rot on NC 3033, but this virulence decreased over years. Virulence of population 612 to NC 3033 also started high and declined with monoculture of NC 3033. Mass isolates from root lesions on 4-wk-old seedlings, instead of single-spore or single-microsclerotium isolates, were increased for initial inoculum in this study and population 612 may initially have been heterokaryotic for virulence.

Although one would expect selection for virulence to occur in composite pathogen populations because they are more variable, the reverse occurred in this experiment. Isolate 617 overcame

TABLE 1. Correlation coefficients[a] for root rot severity[b] (RR) at harvest in October with inoculum density[c] (ID) estimated before or after the growing season and with RR among years from monoculture of susceptible peanut cultivar Florigiant or resistant cultivar NC 3033 in microplots infested with populations of *Cylindrocladium crotalariae*

Variable, mo, year	RR 1979		RR 1980		RR 1981		RR 1982	
	Florigiant	NC 3033	Florigiant	NC 3033	Florigiant	NC 3033	Florigiant	NC 3033
ID January 1980	0.31*	0.24	0.26*	−0.08				
RR October 1980	0.24	−0.06						
ID January 1981			0.29*	0.17	0.13	0.15		
ID June 1981			0.32*	0.36**	0.22	−0.12		
RR October 1981			0.35**	0.05			0.19#	0.27#
ID January 1982					0.51**	0.63**	0.08#	0.07#
ID June 1982					0.06#	−0.11#	0.18#	0.40#
ID January 1983							0.24#	0.88**#

[a] One (*) or two asterisks (**) indicate significance at $P<0.05$ or $P<0.01$, respectively. $N = 60$ except those marked with "#," for which $N = 24$ (the number of microplots).

[b] Root rot severity visually rated on a 0 to 5 scale with 0 = no lesions and 5 = completely rotted roots.

[c] Inoculum density as microsclerotia per gram of soil.

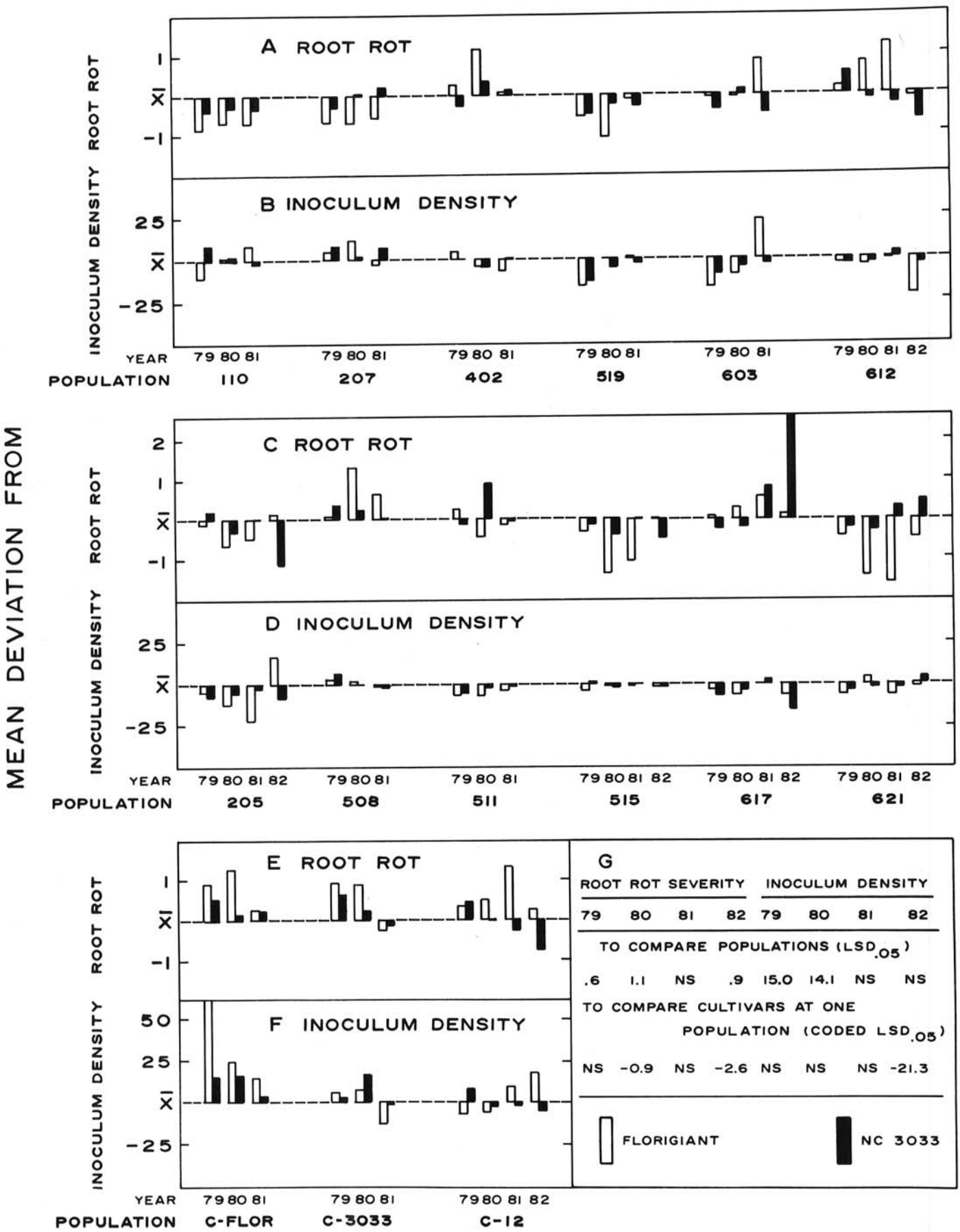
A ROOT ROT
B INOCULUM DENSITY
C ROOT ROT
D INOCULUM DENSITY
E ROOT ROT
F INOCULUM DENSITY
ROOT ROT
INOCULUM DENSITY
MEAN DEVIATION FROM
YEAR
POPULATION
79 80 81
79 80 81 82
110
207
402
519
603
612
205
508
511
515
617
621
C-FLOR
C-3033
C-12
G
ROOT ROT SEVERITY
INOCULUM DENSITY
79 80 81 82 79 80 81 82
TO COMPARE POPULATIONS (LSD.05)
.6 1.1 NS .9 15.0 14.1 NS NS
TO COMPARE CULTIVARS AT ONE POPULATION (CODED LSD.05)
NS -0.9 NS -2.6 NS NS NS -21.3
FLORIGIANT
NC 3033

Fig. 2. Root rot severity (0–5 scale) on susceptible peanut cultivar Florigiant and resistant cultivar NC 3033 in monocultures and postseason inoculum densities (in January following the indicated year) for *Cylindrocladium crotalariae*. Initial inoculum density in 1979 was 35 microsclerotia per gram of soil. Data are deviations from cultivar × year means: **A and B,** six populations of individual isolates originally selected for specific virulence on Florigiant; **C and D,** six populations of individual isolates originally selected for specific virulence on NC 3033; **E and F,** three composite populations composed of six isolates originally selected for specific virulence on Florigiant (C-Flor), six isolates originally selected for specific virulence on NC 3033 (C-3033), or all 12 isolates (C-12); and **G,** least significant differences (Fisher's LSD, $P = 0.05$). NS = not significant. Example: overall root rot severity for 612 in 1979 was less than for 110 because (+0.3 minus −0.6) = 0.9, which exceeds the LSD of 0.6. To compare cultivar means within a population in 1 yr, compare (column height for Florigiant minus column height for NC 3033) with the coded LSD ([uncoded LSD] − [Florigiant mean] + [NC 3033 mean]). Example: for 110 in 1980, bar height was −0.7 for Florigiant and −0.3 for NC 3033 and −0.7 minus −0.3 = −0.4, which is greater than the coded LSD of −0.9.

resistance, but when microsclerotia of 617 were composited with microsclerotia of five or 11 other isolates (Composite-3033 or Composite-12, respectively), virulence decreased over years with monoculture of NC 3033 (Fig. 2). Two of the three populations for which virulence on NC 3033 decreased were composites. In the third composite, Composite-Flor, virulence on NC 3033 was above, but near, the mean in 1980 and 1981 (Fig. 2).

Many microsclerotia of *C. crotalariae* are embedded in root and pod fragments following harvest (12,13). Sampling error, especially at low inoculum density, would account for some estimates being higher in late spring (Fig. 3). Another factor might be the technique for estimating inoculum density that involves blending of debris to disperse microsclerotia (13). Perhaps this was not completely effective in January and at low inoculum density where root rot and pod rot were not extensive. Increased time (January to June) in the soil could result in higher inoculum density estimates because degradation of host tissue would allow dispersal of microsclerotia.

Differences between inoculum density in January and June (Fig. 3) could also reflect lower survival of microsclerotia at high versus low inoculum densities. Density dependent survival has been observed for nematodes (H. Ferris, *personal communication*). When many microsclerotia form in extensively rotted roots, competition for metabolites may result in smaller microsclerotia and/or individual microsclerotia with fewer reserves than those formed in plants with many functioning roots.

If the cost of high virulence and fitness in *C. crotalariae* on resistant peanuts includes a lower survival rate, then this may account for stability of diverse populations. Robinson (15) postulated a limited spectrum of mean general virulence for obligate parasites with a maximum level for the species. *C. crotalariae* has some attributes of an obligate parasite, eg, it does not compete or increase saprophytically in soil. An increase in general virulence after growth in resistant NC 3033 was detected in this and other studies (4,6). We did not determine whether this increase in virulence was due to selection, or selection with parasexual recombination.

Genetic variation for specific virulence to resistant genotypes exists in *C. crotalariae* (4,9), but the existence of variability does not necessarily lead to instability in the resistance of NC 3033. With currently used rotations, the danger of increases in frequencies of genes for general or specific virulence to resistant cultivars seems low. If anastomosis between strains occurs frequently in or on roots, it could tend to maintain virulence genes in heterokaryons while limiting their expression. Frequencies of genes for high virulence on NC 3033 in heterogenous pathogen populations may decrease due to survival disadvantages such as competition with other less specialized genotypes during pathogenesis or lower fitness (limited ability to form new microsclerotia) compared with strains having only general virulence. Both phenomena could be involved in stabilizing selection sensu Vanderplank (18).

Results of this research indicate that the dynamics of virulence in populations of *C. crotalariae* is much more complex than previously realized. Specific virulence detected in greenhouse tests (4) was not expressed to the same extent in this field environment and the expression of specific virulence in the field may have been affected by unique environments from year to year. Effects of monoculture on virulence might vary among locations due to environmental interactions. Genotypic diversity of *C. crotalariae* among infested fields may vary from relatively homogeneous to heterogeneous.

Vanderplank's prediction (18) that quantitative (polygenic) resistance is stable seems to apply in most cases to this host-pathogen interaction. However, under unusual circumstances, the quantitative resistance of NC 3033 to *C. crotalariae* could be unstable, ie, effectiveness of resistance may be lost.

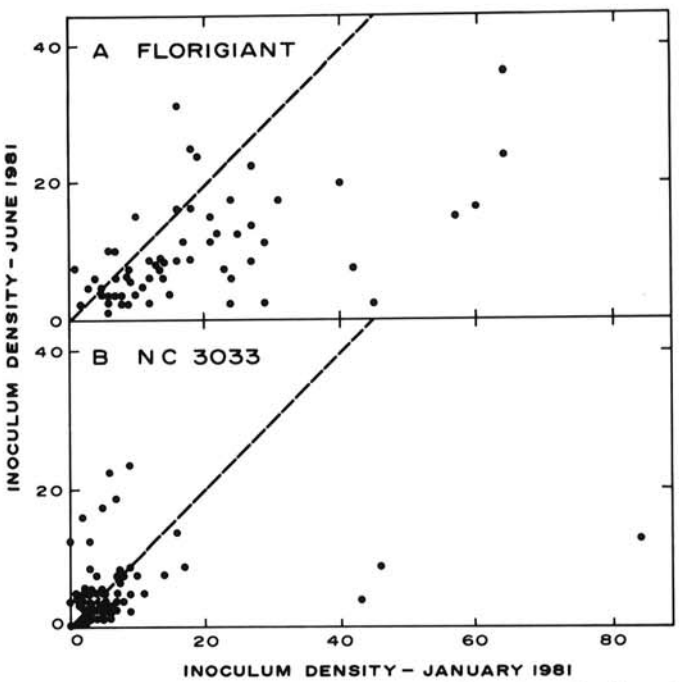

Fig. 3. Relationship between estimates of inoculum density of *Cylindrocladium crotalariae* in soil sampled in January 1981 and June 1981 following two consecutive growing seasons of: **A,** susceptible peanut cultivar Florigiant and **B,** resistant cultivar NC 3033 in microplots. Slashed line represents identical estimates in January and June.

This study shows that relying on a high level of resistance as the only control tactic in peanut monoculture could be detrimental. If the genetic linkage between resistance and poor agronomic quality can be broken, we recommend that growers avoid monoculture even though continuous use of high levels of resistance could provide short-term financial gain.

LITERATURE CITED

1. Bailey, J. E. 1983. Use of fumigants for black root rot (CBR) control. Virginia-Carolina Peanut News 29(1):14.
2. Barker, K. R., Daughtry, B. I., and Corbett, D. W. 1979. Equipment and techniques for establishing field microplots for the study of soilborne pathogens. J. Nematol. 11:106-108.
3. Bell, D. K., and Sobers, E. K. 1966. A peg, pod, and root necrosis of peanuts caused by a species of *Calonectria*. Phytopathology 56:1361-1364.
4. Black, M. C., and Beute, M. K. 1984. Different ratios of general:specific virulence variance among isolates of *Cylindrocladium crotalariae* from different peanut genotypes. Phytopathology 74:941-945.
5. Black, M. C., and Beute, M. K. 1984. Relationships among inoculum density, microsclerotium size, and inoculum efficiency of *Cylindrocladium crotalariae* on peanuts. Phytopathology 74:(In press).
6. Black, M. C., and Beute, M. K. 1984. Effects of rotations with susceptible and resistant peanuts, soybeans, and corn on inoculum efficiency of *Cylindrocladium crotalariae* on peanuts. Plant Dis.

68:401-405.
7. Diomande, M., and Beute, M. K. 1981. Relations of *Meloidogyne hapla* and *Macroposthonia ornata* populations to Cylindrocladium black rot in peanuts. Plant Dis. 65:339-342.
8. Green, C. C., Wynne, J. C., Beute, M. K., and Campbell, W. V. 1983. Relationships of CBR and insect resistance and yield among progenies of a CBR-resistant × insect-resistant cross. Peanut Sci. 10:84-88.
9. Hadley, B. A., Beute, M. K., and Leonard, K. J. 1979. Variability of *Cylindrocladium crotalariae* response to resistant host plant selection pressure in peanut. Phytopathology 69:1112-1114.
10. Hadley, B. A., Beute, M. K., and Wynne, J. C. 1979. Heritability of Cylindrocladium black rot resistance in peanut. Peanut Sci. 6:51-54.
11. Harris, N. E., and Beute, M. K. 1982. Histological responses of peanut germplasm resistant and susceptible to *Cylindrocladium crotalariae* in relationship to inoculum density. Phytopathology 72:1250-1256.
12. Phipps, P. M., and Beute, M. K. 1979. Population dynamics of *Cylindrocladium crotalariae* microsclerotia in naturally-infested soil. Phytopathology 69:240-243.
13. Phipps, P. M., Beute, M. K., and Barker, K. R. 1976. An elutriation method for quantitative isolation of *Cylindrocladium crotalariae* microsclerotia from peanut field soil. Phytopathology 66:1255-1259.
14. Phipps, P. M., Beute, M. K., and Hadley, B. A. 1977. A microsclerotia-infested soil technique for evaluating pathogenicity of *Cylindrocladium crotalariae* isolates and black rot resistance in peanut. (Abstr.) Proc. Am. Phytopathol. Soc. 4:146.
15. Robinson, R. A. 1976. Plant Pathosystems. Springer-Verlag, New York. 184 pp.
16. Rowe, R. C., and Beute, M. K. 1975. Variability in virulence of *Cylindrocladium crotalariae* isolates on peanut. Phytopathology 65:422-425.
17. Sullivan, G. A. 1983. 1983 profit-producing peanut practices. Virginia-Carolina Peanut News 29(1):10-11.
18. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 206 pp.
19. Wynne, J. C., and Beute, M. K. 1983. Registration of NC 8C peanut. Crop Sci. 23:183-184.
20. Wynne, J. C., Rowe, R. C., and Beute, M. K. 1975. Resistance of peanut genotypes to *Cylindrocladium crotalariae*. Peanut Sci. 2:54-56.