

Toward Sustainable Peanut Production: Progress in Breeding for Resistance to Foliar and Soilborne Pathogens of Peanut

B. B. Shew, Research Associate, Department of Crop Science; M. K. Beute, Professor, Department of Plant Pathology; and H. T. Stalker, Professor, Department of Crop Science, North Carolina State University, Raleigh, NC 27695-7616

Peanut, *Arachis hypogaea* L., is a legume grown in warm climates throughout the world. In many developing countries, peanut is an important source of calories, protein, and oil. In the United States, this high-value crop is used primarily in peanut butter and snacks. North Carolina and Virginia produce virginia-type peanut, which is large-seeded and used for roasted shelled or in-the-shell products. Yields average 2,800 to 3,400 kg/ha, but yields of 4,500 kg/ha or more are common for top producers. Gross returns of at least \$2,500/ha are typical. These large yields and high returns are a product of intensive management, which includes extensive land preparation, frequent application of pesticides, and use of high quality cultivars. Without these inputs, yields usually range from 800 to 1,700 kg/ha. Thus, there is a great need in developing countries to increase yield and quality in low-input production systems. In the future, increasing economic and environmental pressures in the United States also will require that high yields be produced with fewer inputs.

The fruiting habit of peanut is unique. After self-fertilization, flowers produce a specialized structure called a gynophore or peg, which grows towards the soil surface and eventually produces an underground pod. Stems, pegs, and pods therefore are in close contact with the soil. Fruit production extends over a period of 3 months in virginia types, which require a growing season of about 150 days. The long peri-

ods needed for pod filling and maturity, together with the proximity of plant parts to the soil, exacerbate disease problems in peanut.

Breeding for resistance to peanut diseases is a high priority at North Carolina State University because several major pathogens cause extensive direct or indirect losses in the North Carolina-Virginia area. The best means to manage resistance also must be determined to fully exploit new cultivars. Two examples will illustrate how pathologists and breeders have cooperated to develop disease resistance in virginia-type peanut.

Cylindrocladium black rot (CBR), caused by the soilborne fungus *Cylindrocladium parasiticum* Crous, Wingfield, & Alfenas (syn. *C. crotalariae*; 5), is a root, peg, and pod rot first described on peanut in 1966 (1). In the 1970s, the disease became established in the North Carolina-Virginia area, where it threatened to devastate the peanut industry. Disease incidence in excess of 80% and yield losses of 50% or more were common in infested fields. Efforts to control the disease began with biological studies conducted in the early 1970s; early attempts at chemical control were unsuccessful or uneconomical (reviewed by Beute [2]).

Initial screening of *A. hypogaea* revealed only partial resistance to *C. parasiticum* (34), although more recent evaluations have shown that high levels of partial resistance also may be available in wild species of *Arachis* (H. T. Stalker, unpublished). A CBR-resistant germ plasm, NC 3033, was released in 1976 (3). Unfortunately, the strong association between CBR resistance, small seed size, and low yield first seen in NC 3033 has continued to hamper efforts to produce cultivars of virginia-type peanut with high levels of CBR resistance.

Expression of partial resistance to *Cylindrocladium* black rot depends on the level of inoculum present (19), so a quantitative assay of resistance was needed. The assay developed allows greenhouse screening of a large number of entries, and

results generally are predictive of resistance in the field (18). Susceptible genotypes become severely diseased at inoculum densities as low as 0.5 microsclerotia per g of soil, whereas NC 3033 expresses moderate resistance at 50 microsclerotia per g (19).

Inoculum density also affects resistance evaluations in the field. Using an assay developed to quantify populations of *C. parasiticum* in field soil (20), Hau et al. (12) found that inoculum was highly clustered. Pataky et al. (18) showed that significant errors in classifying genotype response were possible because of this heterogeneous pattern of inoculum. Analysis of covariance can correct for differences in inoculum density (26). In an alternate approach, Culbreath et al. (6) assayed soil before planting and assigned genotypes to plots based on preplant inoculum densities. This method allowed very precise separation of genotypes based on numbers of dead and wilted plants.

The intensive program for selection and incorporation of CBR resistance in virginia-type peanut resulted in the release of the cultivar NC 8C in 1982 (31). Although NC 8C allowed growers with infested fields to continue to produce peanut, susceptible cultivars yielded better in the absence of disease. NC 8C was not popular with peanut shellers because it required different handling procedures during processing. Backcrossing of NC 8C to the susceptible parent resulted in release of the more commercially acceptable NC 10C in 1988 (32).

NC 10C is less resistant to CBR than NC 8C, and optimum performance requires the grower to use specific management practices such as proper rotations (26), delayed planting (25), and nematode control (7,8). When integrated with good cultural practices, fumigation with metam sodium lowers soil inoculum density enough to nearly eliminate CBR on NC 10C (4), but higher levels of resistance are needed to reduce the monetary and environmental costs of disease management. Advanced generation breeding lines cur-

This research was partially funded by USAID Peanut CRSP grant DAN-40480G-SS-2065-00. Recommendations do not represent an official position or policy of USAID. Mention of a trade name or proprietary product does not constitute a guaranty or warranty of the product named and does not imply approval to the exclusion of other products that may also be available.

Corresponding author: B. B. Shew
E-mail: d-shew@ncsu.edu

Accepted for publication 10 September 1995.

rently being evaluated will give rise to these new cultivars.

Early leaf spot, caused by *Cercospora arachidicola* S. Hori, and late leaf spot, caused by *Cercosporidium personatum* (Berk. & M.A. Curtis) Deighton, are the major foliar diseases of peanut worldwide. Epidemics on susceptible genotypes cause nearly complete defoliation, which dramatically reduces yield. Foliar fungicides are highly effective for control of leaf spots, but they represent 16% of the cost of peanut production in North Carolina, excluding application expenses and environmental impacts. Fungicides are not used extensively in developing countries.

A. hypogaea lacks single-gene immunity to leaf spots, and resistance to the two diseases is inherited independently (33). Breeding for resistance in the Southeast has focused on late leaf spot, and a runner-type cultivar (Southern runner) with partial resistance was released in 1987 (10).

Resistance breeding in North Carolina has focused on *Cercospora arachidicola*, the predominant pathogen in the region. virginia-type germ plasm initially identified as having moderate levels of resistance included NC-GP 343, NC 3033, the cultivar NC 5 (14), and the insect-resistant cultivar NC 6 (15), which had NC-GP 343 as a parent. Advanced-generation selections resulting from crosses of these lines exhibited moderate resistance to early leaf spot (13); but yield, quality, and resistance levels were not high enough to justify release of new cultivars under current market conditions.

A detached leaf technique (16) showed that some, but not all, components of resistance are intercorrelated (21) and that greenhouse results were predictive of performance in the field. Separate AUDPCs for disease incidence and defoliation are the most reliable indicators of resistance in field isolation plots (13,15).

Although inheritance of resistance generally is thought to be under additive control, occasionally dominance or epistatic effects have been significant (33). High levels of partial resistance to leaf spots have been found in some exotic genotypes of *A. hypogaea*, but genetic studies have shown low combining abilities and strong correlation between leaf spot resistance, small seed size, and low yield in some of this germ plasm (11).

Several diploid wild species of *Arachis* have much higher levels of partial resistance to leaf spots than *A. hypogaea* (a tetraploid), and some are immune to one or both diseases (28). Tetraploid lines that were recovered from a cross of *A. hypogaea* × *A. cardenasii* Krap. & W.C. Gregory were selected for resistance to leaf spots and released (27). These lines also were crossed to NC 5 or NC 6; several advanced generation selections had AUDPCs for early leaf spot only 28% of those for GP-NC 343 and AUDPCs for

late leaf spot equal to those for Southern runner (24). Resistance appears to be negatively correlated with large seed size and high yield, but variation among lines should allow selection for these traits (H. T. Stalker, unpublished). These highly resistant lines may be of great use in developing countries, where seed size is of much less concern than in the North Carolina-Virginia growing area.

Many growers in North Carolina and Virginia use a weather-based advisory to reduce numbers of fungicide sprays for control of early leaf spot. NC 6 requires a less stringent advisory and fewer sprays than the standard cultivar NC 7 (15). Unfortunately, incidence of late leaf spot on both NC 6 and NC 7 generally is greater than on previous cultivars (22), possibly because more healthy leaves remain available for infection late in the growing season. Widespread planting of cultivars with resistance to only one leaf spot may result in exchange of one disease for the other (23). Resistance to early leaf spot is somewhat unstable across locations (29), perhaps because temperature influences resistance expression differentially in some genotypes (30). Deployment of peanut cultivars with leaf spot resistance will require excellent management and careful monitoring of the crop.

Through the 1960s, little interest or effort was devoted to breeding for disease resistance because of a perceived lack of variability in peanut. Remarkable progress has been made since then in developing resistant cultivars and breeding lines, and in identifying resistant germ plasm in *A. hypogaea*. In addition, very high levels of resistance or immunity to major peanut pathogens, including *Cercospora arachidicola*, *Cercosporidium personatum*, *Cylindrocladium parasiticum*, and *Meloidogyne arenaria*, have been found in wild species of *Arachis*, particularly in sections of the genus more distantly related to cultivated peanut (28). Recent progress in lowering barriers to interspecific crossing (28), molecular mapping of the peanut genome (9), and developing efficient transformation systems for peanut (17) should allow exploitation of this resource. The continued improvement of peanut through conventional and novel breeding methods will lead to more sustainable production in the future.

LITERATURE CITED

1. 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.
2. Beute, M. K. 1980. *Cylindrocladium* black rot (CBR) disease of peanut (*Arachis hypogaea*). Pages 171-176 in: Proc. Int. Workshop Groundnuts, ICRISAT, India.
3. Beute, M. K., Wynne, J. C., and Emery, D. A. 1976. Registration of NC 3033 peanut germplasm. *Crop Sci.* 16:887.
4. Cline, W. O., and Beute, M. K. 1986. Effect of metam sodium, peanut genotype, and inocu-

- lum density on incidence of *Cylindrocladium* black rot. *Peanut Sci.* 13:41-45.
5. Crous, P. W., Wingfield, M. J., and Alfenas, A. C. 1993. *Cylindrocladium parasiticum* sp. nov., a new name for *C. crotalariae*. *Mycol. Res.* 97:889-896.
6. Culbreath, A. K., Beute, M. K., and Campbell, C. L. 1991. Spatial and temporal aspects of epidemics of *Cylindrocladium* black rot in resistant and susceptible peanut genotypes. *Phytopathology* 81:144-150.
7. Culbreath, A. K., Beute, M. K., Shew, B. B., and Barker, K. R. 1992. Effects of *Meloidogyne hapla* and *M. arenaria* on black rot severity in new *Cylindrocladium*-resistant peanut genotypes. *Plant Dis.* 76:352-357.
8. Diomande, M., and Beute, M. K. 1981. Relation of *Meloidogyne hapla* and *Macroposthonia ornata* populations to *Cylindrocladium* black rot in peanuts. *Plant Dis.* 65:339-342.
9. Garcia, G. M., Stalker, H. T., and Kochert, G. 1995. Introgression analysis of an interspecific hybrid population in peanut (*Arachis hypogaea* L.) using RFLP and RAPD markers. *Genome* 38:166-176.
10. Gorbet, D. W., Norden, A. J., Shokes, F. M., and Knauff, D. A. 1987. Registration of "Southern Runner" peanut. *Crop Sci.* 27:817.
11. Hamid, M. A., Isleib, T. G., Wynne, J. C., and Green, C. C. 1981. Combining ability analysis of *Cercospora* leafspot resistance and agronomic traits in *Arachis hypogaea* L. *Oleagineux* 36:605-609.
12. Hau, F. C., Campbell, C. L., and Beute, M. K. 1982. Inoculum distribution and sampling methods for *Cylindrocladium crotalariae* in a peanut field. *Plant Dis.* 66:568-571.
13. Johnson, C. S., Beute, M. K., and Ricker, M. D. 1986. Relationship between components of resistance and disease progress of early leaf spot on Virginia-type peanut. *Phytopathology* 76:495-499.
14. Kornegay, J. L., Beute, M. K., and Wynne, J. C. 1980. Inheritance of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in six virginia-type peanut lines. *Peanut Sci.* 7:4-9.
15. Matyac, C. A., and Bailey, J. E. 1988. Modification of the peanut leaf spot advisory for use on genotypes with partial resistance. *Phytopathology* 78:640-644.
16. Melouk, H. A., and Banks, D. J. 1978. A method of screening peanut germplasm for resistance to *Cercospora* leafspot. *Peanut Sci.* 5:112-114.
17. Ozias-Akins, P., Schnall, J. A., Anderson, W. F., Singait, C., Clemete, T. E., Adang, M. J., and Weissinger, A. K. 1993. Regeneration of transgenic peanut plants from stably transformed embryogenic callus. *Plant Sci.* 93:185-194.
18. Pataky, J. K., Black, M. C., Beute, M. K., and Wynne, J. C. 1983. Comparative analysis of *Cylindrocladium* black rot resistance in peanut: Greenhouse, microplot, and field testing procedures. *Phytopathology* 73:1615-1620.
19. Phipps, P. M., and Beute, M. K. 1977. Sensitivity of susceptible and resistant peanut cultivars to inoculum densities of *Cylindrocladium crotalariae* microsclerotia in soil. *Plant Dis. Rep.* 61:300-303.
20. 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.
21. Ricker, M. D., Beute, M. K., and Campbell, C. L. 1985. Components of resistance in peanut to *Cercospora arachidicola*. *Plant Dis.* 69:1059-1064.
22. Shew, B. B., Bailey, J. E., and Beute, M. K. 1993. Response of virginia-type cultivars of *Arachis hypogaea* L. to peanut leaf spots.

- Phytopathology 83:1375.
23. Shew, B. B., and Beute, M. K. 1990. Peanut genotype effects on occurrence of *Cercospora arachidicola* and *Cercosporidium personatum* in North Carolina. Proc. Am. Peanut Res. Educ. Soc. 22:42.
 24. Shew, B. B., Beute, M. K., and Stalker, H. T. 1992. Resistance to *Cercospora* leaf spots in peanut genotypes derived from crosses with wild *Arachis* spp. Phytopathology 82:1140.
 25. Sidebottom, J. R., and Beute, M. K. 1989. Control of *Cylindrocladium* black rot of peanut with cultural practices that modify soil temperature. Plant Dis. 73:672-676.
 26. Sidebottom, J. R., and Beute, M. K. 1989. Inducing soil suppression to *Cylindrocladium* black rot of peanut through crop rotations with soybean. Plant Dis. 73:679-685.
 27. Stalker, H. T., and Beute, M. K. 1993. Registration of four leafspot-resistant peanut germplasm lines. Crop Sci. 33:1117.
 28. Stalker, H. T., and Moss, J. P. 1987. Speciation, cytogenetics, and utilization of *Arachis* species. Adv. Agron. 41:1-40.
 29. Waliyar, F., Bosc, J. P., and Bonkoungou, S. 1993. Sources of resistance to foliar diseases of groundnut and their stability in West Africa. Oleagineux 48:283-287.
 30. Waliyar, F., Shew, B. B., Stalker, H. T., Isleib, T. G., Sidahmed, R., and Beute, M. K. 1994. Effect of temperature on stability of components of resistance to *Cercospora arachidicola* in peanut. Phytopathology 84:1037-1043.
 31. Wynne, J. C., and Beute, M. K. 1983. Registration of NC 8C peanut. Crop Sci 23:184.
 32. Wynne, J. C., Beute, M. K., Bailey, J. A., and Mozingo, R. W. 1991. Registration of 'NC 10C' peanut. Crop Sci. 31:484.
 33. Wynne, J. C., Beute, M. K., and Nigam, S. N. 1991. Breeding for disease resistance in peanut (*Arachis hypogaea* L.). Annu. Rev. Phytopathol. 29:279-303.
 34. Wynne, J. C., Rowe, R. C., and Beute, M. K. 1975. Resistance of peanut genotypes to *Cylindrocladium crotalariae*. Peanut Sci. 2:54-56.